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2nd Edition

Textbook of Forensic Medicine & Toxicology

Nageshkumar G Rao

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Textbook of Forensic Medicine and Toxicology

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SECOND EDITION

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Textbook of Forensic Medicine and Toxicology

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Dedicated to my beloved parents

Late Sri S Gopal Rao and Late Smt Sharada Bai G Rao

"If I have the belief that I can do it, I shall surely acquire the capacity to do it, even if I do not have it at the beginning".

—Mahatma Gandhi

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FOREWORD TO THE SECOND EDITION

Ramdas M Pai
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Law and medicine are the world's oldest noble professions that are claimed to have been wedded long ago, transforming into a science of facts assisting in to resolve the social evils. No other professional endeavour has ever struggled in recent decades as forensic medicine to develop into what it is today! Indeed, with escalating violence, terrorism and such worldwide criminal activities, it is obvious that degree of application of forensic principles and its concepts accomplished a lot in solving the crime mysteries fascinatingly. As a doctor, a forensic expert applies his/her medical knowledge to the knowledge of law not only in solving the crime, but also imparting justice in the court of law to the distressed or dead.

Professor Nageshkumar G Rao, was our faculty, worked as Head of the Department of Forensic Medicine at Kasturba Medical College (KMC), Mangalore, has profound experience and knowledge to compile and revise this book. His research publications available in the world forensic literature have earned many honours conferred on to him by the various authorities in India. I understand that Dr Rao got the coveted honour of ICFMT Annual Congress Award, 2008. In 1987, Kasturba Medical College had conferred him with *Dr TMA Pai Gold Medal for Research publication*.

I have gone through this revamped second edition of ***Textbook of Forensic Medicine and Toxicology***. The book has very useful information for the medical and law students, and for the practising physicians and legal professionals. The publisher's efforts to reprint the first edition clearly spell out the popularity of the book. Innumerable references cited in each chapter construe the scientific base for the book. The book also includes worthy appendices at the end, comprising of question bank providing theory and viva-voce questionnaires, varsity examination methodology and suggested syllabus in both theory and practical examinations.

I am sure, the second edition of the book too will get very good reception. My best wishes to Prof Nageshkumar G Rao.

Ramdas M Pai

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FOREWORD TO THE FIRST EDITION

Forensic Medicine has claimed its full share in dramatic progress of medicine during the present century. No aspect of patient care is free from its growing impact and Medical Jurisprudence of yesteryears has moved from the fringes to the center stage of a doctor's training. Today, forensic issues confront not only the specialist but also the young doctor treating a victim of violence in a village, a consultant facing a consumer claim and an investigator seeking informed consent for a new procedure. Forensic medicine has cast its mantle on every facet of the interaction between medicine and society.

Unlike the predecessors, today's physicians are called upon to deal with a new variety of problems, which impinge on science, ethics and law. *In vitro* fertilization, surrogate motherhood, brain death, organ donation, consumer protection and the living will raise unprecedented questions, which demand the attention of the best minds in medicine, jurisprudence and law. Furthermore, the spread of violence and increase in diabolical crimes has aggravated the problems for the practicing physician. To tackle the practical side of these questions, forensic medicine has summoned the resources of science in full measure— DNA technology for identity tests, neutron-activation analysis and scanning electron microscopy for crime detection and many other examples illustrate the scientific advances in its methodology. It is imperative that the medical student gains a clear understanding of the practical, legal and philosophical issues in forensic medicine during the course of his or her training. This is as important for his or her trouble-free practice as for the safety of the society whom he serves.

Professor Nageshkumar G Rao has drawn upon his rich experience as a teacher in writing this book, which is primarily addressed to the medical students. They will find here a mine of up-to-date information on every aspect of forensic medicine, presented lucidly and expertly with collection of excellent color photographs and simple line drawings. The coverage includes ethics, legal procedures, consumer protection, thanatology, autopsy examination, trauma, toxicology, and other important topics. Professor Rao deserves our compliments for preparing this easily readable text which I hope will become popular among medical students and the practitioners of medicine.

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FOREWORD TO THE FIRST EDITION

Textbook of Forensic Medicine and Toxicology is a lucid elucidation of the essence of medicolegal aspects of medical practice, which is essential knowledge, nay, compulsory knowledge, for all medical students, and practicing doctors irrespective of their specialty. Professor Nageshkumar G Rao, presently our Head of the Department of Forensic Medicine and Director of Postgraduate studies, has been my student whom I know intimately, right from his undergraduate student days.

After paying due obeisance to his postgraduate training and teaching expertise in the specialty of Forensic Medicine, I must confess that he has matured into a good teacher and an excellent communicator at the same time.

The book is concise, but covers all vital aspects of Forensic Medicine. I feel it will be an asset to every practicing doctor to be kept with him for ready reference. With the consumer problems coming into medicine in a big way, this assumes greater significance. I wish we had taught our students the Indian code of ethics, which, in my opinion, is far superior to the Hippocratic ethics that we all swear by.

Consumerism is a bane to the practising doctors; but we must remember that it is the medical profession, which has taken medicine to the market place and converted it into a business, like any other profit making business, to attract consumerism. "Never make money in the sick room" was Hippocratic dictum. With corporate business getting into hospitals based on the five-star western culture (while large hospitals in the West are closing down), it was inevitable that some checks and balances had to be introduced into the practice of medicine.

The format and the printing of the book have been of very high order. Photographs, line drawings, flow charts, and tables in the book are relevant and extremely useful. Colour photographs presented in 60 plates with nearly 300 pictures are excellent. The language is simple and easily understandable. The book should be in the clinic of every practising doctor. This would be a blessing as a ready reference in the courtroom also. I see a bright future for this book. I feel it may help the exam going student as well. I wish the book all success.

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FOREWORD TO THE FIRST EDITION

Forensic Medicine is a vast subject, which needs to be explored by an overburdened student, in very little time available to him. There are umpteen number of text books available in the market, but many of them give archaic information, which is no more relevant in the current scenario. Forensic medicine is a rapidly changing subject, and there was a dire need to look at it from a fresh angle. By their very nature, the existing books, were not able to do justice with this situation. They were existent in the market for a long time, and the authors were sometimes reluctant to jettison old and archaic information, which had earlier found favor with the students.

I first met Nageshkumar G Rao, the author of this eminently readable book, more than a decade ago, during an academic conference in Berhampur, and was immediately struck by his academic brilliance. I couldn't help being drawn towards him, and gradually found myself drifting closer and closer to him. During the next few years, we all saw him edit the *Journal of the Indian Academy of Forensic Medicine*, which undoubtedly had its best period under his stewardship. Not only did he edit the journal, but from time to time, published brilliant academic papers in it too. Many times I found that the papers were brilliant enough to have found a place in some more widely circulated journals originating from some Western countries, and I often spoke my heart out to him. I was struck by the answer he gave me. He told me, "Anil, we have to enrich our own journals, not the foreign ones." Such was the commitment of this author towards Forensic Medicine, and Indian Forensic Medicine in particular.

For a long time we all wanted to have a book, which could look at Indian forensic medicine from a fresh angle, and judging by my experiences with him, I had no doubt that it was Nagesh who could do it. When I was in the Edinburgh Medical School, Scotland, during the late eighties and early nineties, working with the doyen of Forensic Medicine, Professor Anthony Busuttil, I maintained close contact with him, and advised him in this direction. During the mid-nineties, I found myself in Japan working with Professor Katsuji Nishi, and later in Armed Forces Institute of Pathology, Washington DC with Dr Charles Stahl, and during those periods too I would often write to him, stimulating him to do this long pending job. I suspect, it was that stimulation which finally spurred him on to write this book.

From then onwards, whenever I would actually meet him during an academic conference, I would always ask him what he had done in that direction. Last year, during the Annual IAFM conference at AIIMS, New Delhi he showed me some work he had done in this direction, and I was happy he had started. But when I first had a look at the complete manuscript, I couldn't believe myself. Undoubtedly, I had expected a sterling book from him, but this was a different stuff altogether. It was concise and to-the-point, and yet contained all the latest stuff that should have rightly been there. For instance there is a detailed and scholarly treatment of latest Acts like The Consumer Protection Act, The Human Organs Transplantation Act and even the latest ethical and legal position on diseases like AIDS. No existing book on forensic medicine deals with these subjects as comprehensively as this one. Toxicology has been dealt with from a fresh angle too. The book has a number of diagrams, which make the text easier to comprehend.

I suspect, whenever a new textbook comes to the market, the author owes an explanation not only to students, but to all his academic brethren. The question often asked is, "What was the need of a new book, when a plethora of textbooks were already available?". But fortunately Nageshkumar G Rao will not have to go through this often embarrassing exercise. This book is in many ways different from the existing textbooks, and undoubtedly would come as a succor to all the students of this country and abroad.

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PREFACE TO THE SECOND EDITION

The book is a complete revitalization of its predecessor published around a decade ago. The publisher's effort to reprint the previous edition earlier explains the popularity of the book. However, as an author I felt the need to revise the text on account of rapid advances and developments in forensic research globally. This edition was made possible by the combined efforts of nearly sixty brilliant academicians who served as the *honorary review panel members and contributors* of this book.

The contributors helped me in improving the contents by providing new information, chapters, photographs and such other materials, while the review panel did the critical evaluation, appended recent advances, checked the language and made essential changes in the text. *Dr Antony Busuttil*, Regius Professor of Forensic Medicine of the University of Edinburgh; *Dr S Subramaniam*, Forensic Pathologist, Dept of Pathology, Kuwait University, Kuwait; *Dr L Thirunnavakarasu,* Retired Professor and Head, Dept of Forensic Medicine, Victoria Hospital, Bangalore Medical College and St. John's Medical College, Bengaluru, Karnataka, India *Dr Alexander F Khakha*, Professor and Head, Dept of Forensic Medicine, Vardhaman Medical College, Safdar Jung Hospital, New Delhi, India and *Dr B Santha Kumar,* Professor and Head, Dept of Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu, India are five elite gentlemen and the best of my friends, whose selfless helps and remarkable efforts were crucial in revising this book. Besides, one of my brilliant postgraduate students–*Dr Ritesh G Menezes*, currently the Assoc. Professor of Forensic Medicine, at Kasturba Medical College, Mangalore, Karnataka, India has provided enduring and outstanding assistance in every phase of this venture.

The first edition with *two parts* and *26 chapters* has now been transformed into *five parts* and *40 concise and quality chapters* packed with recent scientific advances. This edition also includes Appendices with four important addenda on *Question Bank, Syllabus and Varsity Examination Aid*, Laws of Relevance to Medical Profession in India and, Starvation and Neglect and Law. I have avoided too many case stories, historical anecdotes and quotes in this book, which I felt were superfluous and digressed from an academic point of view. The primary focus of this over 600 pages edition has been to incorporate relevant subject matter covering the *syllabus* recommended by the *Medical Council of India* using simple, comprehensible language. Over 1500 references, 700 photographs, 500 drawings, charts and tables found in this book will make the intricate theory of the subject extremely easy to understand for every reader. Especially students will not only excel in examinations but also achieve a thorough, usable knowledge of the subject for future professional life. M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India has published this book with great care using international standard printing technologies. They have designed an attractive cover, and utilized the power of coloured printing to create clear and beautiful pages so that reading of this book can be made pleasurable and interesting.

Suggestions, constructive criticisms and thoughts for improving this book are more than welcome. Kindly email them to *prof.nkgr@gmail.com* I am sure that the forensic panorama provided in this edition will draw the attention of every reader and generate more response than the previous edition from students, professionals, clinicians, general practitioners, police and law officials alike.

Nageshkumar G Rao

PREFACE TO THE FIRST EDITION

Forensic Medicine and its methods have been in practice down the ages in history, but its scientific status and the development as a single discipline are of only recent origin. It appropriately now constitutes an integral part of undergraduate and postgraduate medical curricula. Modern Forensic Medicine, with an objective of making the subject specialty more wider, practical and to evolve into a tertiary specialty, has been re-introduced under five major subdivisions or newer taxonomy, viz, *History and Developments in Forensic Medicine, Medical Jurisprudence, Forensic Pathology, Clinical Forensic Medicine and Forensic Toxicology* as appreciated globally. In this book an attempt is made to introduce these newer concepts in India, as approved and proposed by the Medical Council of India *(Gazette of India, May, 17, 1997).*

The 26 chapters presented in this edition, amalgamating all these five major subdivisions of the subject specialty, not only harvest of my personal experiences gained from the living and dead cases, I have been involved as *medicolegal expert*, but also blend with the cream of knowledge gathered from innumerable references in forensic literature.

As a teacher in Forensic Medicine over the last two decades I have been feeling the need for a well-illustrated textbook, with precise and specific information. With short and lucid text and nearly 300 *colored photographs, 200 B/W photographs, line drawings and simple flow charts*, and *100 tables* rendering easy understanding, recollection and reproduction in the examinations by medical students, I feel this textbook is the first of its kind in India. Perhaps this book could also cater to the needs of every *medical practitioner, investigating police officer, lawyer, law enforcing authority*, *court* and *forensic scientist*. My earnest hope is that this new book will find favorable response from all medical students and other concerned group of readers and find a suitable place in Forensic literature.

Nageshkumar G Rao

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Part I: Introduction and Evolution

Chapter 1

Introduction

Medicine and Law were wedded from the earliest times, perhaps from the perceived necessity of protecting the community from the irresponsible acts of unqualified medical practitioners and quacks. Religion and superstition were intimately entangled with the medical art for time immemorial and this has also rubbed on to the dealings between those practicing medicine and those practicing law. From this interaction between these two professions of medicine and of law, emerged the specialist discipline and later the academic subject, *forensic medicine*.

DEFINITION

Forensic medicine is defined as that branch of medicine, which deals with the application of medical and paramedical scientific knowledge to the knowledge of both civil and criminal law in order to aid administration of justice.[1-10]

The word *forensic* is derived from the *Latin* word *forensis*, which implies something pertaining to the *forum*. In ancient Rome, the 'forum' was the communal meeting or market place where those with public responsibility discussed civic and legal matters, and where justice was dispensed and indeed seen by the public to be dispensed.[2,4,7-10]

In ancient India also, settlement of disputes was done by *Panchayat* where a group of *panchas* or *five* village elders were authorized to settle the dispute.[6,8] Thus, the word *forensic* essentially conveys any issue related to the debate in relation to medical matters that can occur in a court of law.

FORMER TERMINOLOGIES

Forensic medicine was earlier known as ***Medical Jurisprudence*** (*Juris* meaning *Law* and *prudentia* meaning *knowledge*). Indeed the first university chairs in this subject also bear the additional title of *Medical Police*.[3] The specialist in this discipline was supposed to be knowledgeable in matters of public health and hygiene, industrial health, epidemics of disease and other matters, which nowadays pertain together to the specialty of Public Health Medicine. For this reason also ***Social Medicine*** (*Medicina Socialis*) was thought to fall within the remit of the same specialist and this is still the case in the continent of Europe, e.g. France, Portugal and Italy.[1-3]

Social medicine pertains to medical matters related to employment and includes such other, matters as disease and injuries acquired at work, compensation for such, through insurance companies, and so on. In the Anglo-Saxon scheme of things and in those other systems derived from it, the specialist in forensic medicine does not have this additional clinical and community-related remit.

This discipline has also been termed as ***State Medicine***, which was the code of medical ethics and practice developed to regulate the code of conduct for registered medical practitioners, to guide and regulate the professional activities of the doctors and to standardize and supervise the medical profession. In the continent of Europe, the term ***Legal Medicine*** is often preferred and accepted to explain the interaction of professions of medicine and law.[3-5]

This range in terminology should direct the reader to the nuances of practice that exist worldwide in this specialty. It is, perhaps more important that the specialty is not taken to include forensic science as an integral and essential part of it. Although in a number of countries scientists and medical practitioners rub shoulders with each other and often work in the same department, there should not be a conscious or subconscious trend to mix the two specialists: Doctors may be scientists, but are not necessarily so. Doctors offer opinions based on their observations. In the same vein scientists are rarely medical men, but they measure accurately the characteristics of physics, chemistry, biology, etc. in which they are involved. To ensure that the best advice is given to lawyers, it is essential that our legal colleagues and the courts are made well aware of such a distinction and the reasons and situations in which it should be made.

The two main aspects of legal medicine are pathology and clinical work. **Forensic Pathology** is practised by those who are able to carry out autopsies and who have the appropriate level of knowledge and expertise to distinguish the various pathological processes which may occur in the human body as a consequence of aging, natural processes, disease and injuries of various types.[7-10] The **Clinical Forensic Medicine** deals with those who are still alive and on whom a medicolegal opinion is required. This includes those who have been traumatised physically and/or sexually, but who have not succumbed to their injuries, those who are under the influence of alcohol and/or drugs in relation to such matters as driving, human rights abuses, etc.[7-10] The latter medical practitioners have been referred to as police surgeons, casualty surgeons, forensic medical examiners, and this branch of legal medicine is often specifically referrd to as forensic medicine.[1-10]

Thus, although medical practitioners have given evidence in the courts and professional opinions of their findings over the years since the dawn of history, the academic and specialised status of this specialty and its development as a single specialised discipline with its own teaching programmes, diplomas and certificates, and curriculum of postgraduate specialisation is of only recent origin. In most countries this subject appropriately now constitutes an integral part of undergraduate and postgraduate medical curricula, and it is furthermore fully integrated into the training of police officers, lawyers, the judiciary and others.

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Chapter

2

Historical Perspectives

ORIGIN OF THE TERMINOLOGY FORENSIC MEDICINE

The study of primitive cultures reveals a close relationship between magic (*witchcraft*) of the *sorcerer, shaman, witch doctor, etc.* and development of science and medicine. With the evolution of civilisation and its general progress, Legal medicine was born as a separate branch of medical discipline on its own merit, and has now reached its present professional and academically respected status.[1-5]

The history of a subject is always considered as the *key to the past, explanation of the present and/or signpost for the future.*

An effort has been made to provide brief details of the early developments in this specialty through the different centuries as an aide memoir to those researching its historical background.

4000-3000 BC

Existing records confirm an interaction between legal and medical matters and this is to be found in the histories of the *Sumerian*, *Babylonian, Indian, Egyptian, Assyrian* civilisations apart from *Chinese* and *Indian* data *on Materia Medica* which include in them information on many *poisons.* It has been accepted widely that the *Indus Valley Civilisation (3250-200 BC)* is much more ancient than written chronicles on Indian history. Forensic medicine as it is practised today in India has attained its present state of development and high probity by passing through several phases of evolution. Amidst the anciently entrenched bonds of the medical arts with superstition, religion, magic, mysticism, folklore and custom, etc. through various centuries and generations, the modern growth and evolution of Indian civilisation had developed an Indian system of medicine, based on the accepted Western system of medicine mainly due to many years of British domination.[4-14]

3000-1000 BC

Imhotep *(2980 - 2900 BC),* the Grand Vizier is considered as the *first medicolegal expert.* He was both *Chief Justice* and the *Chief/Personal Physician* to *Pharaoh Zoster,* the ruler of Egypt. He was claimed to be the *God of Medicine.* Furthermore, in ancient Egypt, the actual practice of medicine was subject to legal provisions. The doctor was punished for the wrong treatment of patient. Stab injuries were recognised for their lethal potential. Egyptian doctors were aware *(like Indian doctors of the same period)* that fractures of skull could occur without any overlying soft tissue injury. The Egyptians were very proficient in the art of preserving the dead body by *mummification.* *Criminal abortion* was punishable during this period.[1,9-11] From the clay cuneiform tablets and the *Papyri* as recovered from *Samaria, Babylon,* etc. a general idea about the then-current system of law, crime and punishment can be gleaned. Punishment, including corporal punishment and mutilation: Cutting of ears, hands, nose and feet, hard labor in prison for varying periods, the throwing of convicts to the crocodile or lion, etc. were well established as modes of punishment for the guilty.[1,9]

Evidence of medicolegal knowledge which was embodied can be found in early legal systems[1,9] such as in the:

- **Code of Hammurabi of Babylon** *(2000-1000 BC)*, which is the oldest written code of law written by *Hammurabi*, King of Babylon at about 2200 BC. It is well known for its provision of punishment of physicians found wanting and guilty of improper treatment with the potential for civil and criminal responsibility.
- **Code of the Hittites** *(1400 BC)*, which constituted a lengthy table of legal compensation for personal injuries.

1000-50 BC

The developments during this period are highlighted below.[1,5,9,10]

- In Greece, around *460-355 BC,* **Hippocrates**, the physician of antiquity and the father of medicine, dealt in his teachings with medical ethics, lethality of wounds, causes of sudden death, etc.
- **Aristotle (384-322 BC)** is regarded as *Father of Modern Family Planning*, as he advocated population control by inducing abortion before animation of fetus. He postulated upper age limit of procreation in males to be around 70 years, and age of menopause in females around 50 years.
- The most important of the Pre-Christian legal codes was *Roman Law,* on which, to this very day, many principles of law throughout the world are based, and whose *rationale* and *phraseology* still pervades many legal systems, e.g. *novus actus interveniens, res Ipsa Loquitor*. The *Lex Aquillia* dealt with the lethality of wounds *(572 BC)*. The ***Tabulae Duodecem*** contained a code of laws enacted in *451-450 BC*. These tables contained a number of provisions of medico legal significance concerning matters such as *competency of the mentally ill, gestation period* for development of the human fetus, euthanasia, eugenics, etc.
- The *amicus curiae* of Roman law*:* Literally translated this phrase meaning *friend of the court who* was appointed as advisor to the judge on matters requiring specialised knowledge. They are honorary advisors and paid no fees for their expert opinions or advices given. Thus, the effective utilisation of expert evidence in the judicial system had its origin in the Roman practice.

- In Rome, the operation of *Caesarean section surgery* was advised to *save life of the child* and also solution of medical problem of *inheritance*.
- Antistius, the physician, opined by externally examining the body of Julius Caesar (*100-44 BC)* that out of 23 injuries on the body, the *one that entered the chest* between first and second rib *was the mortal one*.

During the Time of Christ

In the time of Christ in the Middle East there was both the local Jewish influence and as elsewhere the Roman influence. Suicide in Jewish law was regarded as abhorrent as there was the belief that the individual who took his own life was possessed by evil spirits and that by taking his life he would pass the evil spirits on to other members of the community.[9]

In Greece suicide was considered an act of self-destruction and rebellion against the Gods. In these cases the medical expert had to reach a decision as to whether the deceased person committed suicide and should therefore be punished. Punishment would usually consist of denial of the right to a funeral.[9-11]

In Rome, those soldiers who committed suicide were considered deserters and those criminals who committed suicide to escape punishment were also condemned.[5,9,13]

The priests in the temple decided on disease management and those who were cured particularly of leprosy had to be seen by the priests before they were declared as *clean* again and were able to join the community again.[9,13]

1st and 5th Century AD

Literature of forensic interest was observed as early as 2nd-3rd century AD.[5,14-16] *Pliny the Elder* in his treatise mentioned about suspended animation, sudden and natural deaths, suicide, etc.

The *Justinian Code (529-564 AD)* prescribed regulation of medical practice and imposition of penalties for malpractice. It also recognised expert testimony. It has been clearly enunciated in the *Digest* that *Physicians are not ordinary witnesses but they give judgement rather than testimony.* The help of medical experts was sought especially in respect of proof of pregnancy and its duration, time of delivery, sterility, impotence, inheritance, rape, abortion, marriage, divorce, survivorship, mental illness, poisoning, etc. The Barbarians, who overthrew the *Roman Empire*, laid down in a statute that in the court of law, the help of *medical experts* should be taken to evaluate *injuries*, before meting out *punishment*.

5th-10th Century AD

During this period there was a close relationship between the development of medicine and medical ethics and the teaching of the Catholic Church, which held both a political and religiously powerful position, which pervaded every aspect of life.

Legal medicine extends all through the history in one form or another. In the fifth century, Germanic and Slavic people overthrew the Roman Empire in Western Europe. These tribes, Salian Franks, the Alemanni, the Goths, the Vandals and the Lombards were considered Barbarians and destroyers of culture and civilisation, and yet these people were the first to lie down by statute that medical experts should be used to determine the cause of death. They had moved beyond the practice of personal vendettas and called for individual and community responsibility.[2] The Wergeld, or blood-price, was paid to the victim by the suspect criminal, or in the case of murder, to the victim's family and relatives. A necessary result of this system was an evaluation of the wounds by the courts, and the courts had to rely on the expertise of a competent medical person.[2,4] There is clear reference to the use of these medical experts in the writings of the courts. The *Lex Alemannorum* gives precise anatomical details of wounds and the reparation given with the situation and gravity of these wounds and orders that medicolegal examinations were to be made for that purpose.[2] *Charlemagne (782-814 AD) in his Capitularies* enjoined that the judges should seek medicolegal opinion from competent experts in *cases of wounding, suicide, infanticide, rape, divorce, impotence, bestiality, etc*.[5]

11th-12th Century AD

Frederick II ordained that, *would-be-physicians* fulfill the following requirements if they wanted to practice the art of medicine as physicians; the candidate had to be:

- Aged 21 years
- Born legitimately
- Studied logic (philosophy) for 3 years
- Studied medicine for 5 years according to teaching of Hippocrates, Galen and Avicenna
- Served a year's apprenticeship
- Had to pass an examination conducted by his teachers
- He would have to take *oath* to treat the poor free and visit his patients as required.

Hence, the *medical teaching*, *training and practice* in those days were *restricted under law*- thus, ***State Medicine*** *was born*.[1-3,5]

12th-15th Century AD

In China, a handbook called *Hsi Yuan Lu* was published in 1250, which contained descriptions of postmortem examinations of bodies and pointed to differences between those injuries caused by sharp and blunt instruments. It also commented whether an individual found in water had died of drowning or had been dead beforehand, or if a burned individual had been dead before an onset of a fire.[4,10]

Other achievements during this period can be summarised as below:

- A public inquest was made obligatory in cases of sudden death in England with *crowners (now* coroners) being appointed for this purpose by the monarchy and entrusted with keeping the King's please.
- In 1209, Pope the Innocent III, ordained the appointment of doctors in Law Courts for examination and opinion in case of injuries.
- In 1249, Hugo De Lucca, a famous surgeon, was appointed as medicolegal expert in Bologna, Italy.
- In 1374, the Pope, through a Bill, allowed autopsy examination with the penalty of excommunication being withdrawn from those involved in such examinations.

16th-17th Century AD

Records of forensic pathology in Europe began in 1507 when a volume known as the Bamberg Code appeared. Twenty-three years later ***Emperor Charles V*** issued a more extensive penal code called the ***Constitutio Criminalis Carolina***. These two documents underlined the importance of *forensic pathology* by requiring that *medical testimony* be made available to the courts, and especially in trials that questioned the manner of death as infanticide, homicide, abortion, or poisoning.[4,10] Records show that wounds were opened to show the depth and direction, although complete autopsies may not have been performed in every case.[5,10,11]

As the centuries progressed, forensic pathology became more and more utilised by the investigators to help solve crimes, to distinguish between homicidal and accidental drowning, descriptions of bullet and stab wounds, findings in asphyxia cases, infanticide and natural death.[1,9]

Michaelis and ***Bohn*** held the first formal lectures in forensic pathology at the University of Leipzig, Germany, where students were instructed in natural and violent deaths. The achievement of the century is that the ***Bishop of Bamberg*** codified medical evidence in all cases of violent deaths in penal code officially.[5,9]

- ***Ambroise Pare*** wrote a *treatise* on different *medicolegal problems* including death from various causes, how to differentiate *antemortem injuries* from *postmortem ones, etc.* Model *Case reports* were also incorporated.[9]
- *Methodus Testificandi* dealing with wounds, poisoning and sexual matters were compiled by ***Codronchius,*** an Italian physician of Imola.[5]
- ***Fortunatus Fidelis*** of Palermo, Sicily, published a systematic treatise on *legal medicine* in 4 volumes entitled *De Relationibus Medicorum*.[5]

17th-18th Century

Following achievements during the period are considered worth remembering:[5]

- ***Paulo Zacchia****, a papal physician,* published *Quaestiones medicolegales* covering not only different aspects of Forensic Medicine but also that of *public health and pastoral medicine* in 17th century.
- ***Valentini***: in 1701 published *Pandectae medico-legalese*—a work to challenge that of Zacchia.
- The first medicolegal journal was published in Berlin under the Editorship of ***Uden*** *and* ***Pyl*** in the latter part of 17th century.
- *Antoine Louis, Chaussier, Mahon, Fodere, Orfila (Father of modern toxicology)* from *France,* and *Henke, Mende and Johan Ludwig Casper* from *Germany* were considered as the famous medicolegal experts. The latter's monumental work (1856), entitled *Praktisches Handbuch der Gerichtlicvhen Medizin* was published through 9 editions with an English translation in 1861-65.
- *Chairs of Professorship* in *Medical Jurisprudence* was established in *German, Italian and French Universities* in the early part of 18th century.
- ***Andrew Duncan*** became the first Professor in the subject at Edinburgh and gave his first set of lecturers in 1807. ***Sir Robert Christison*** became *Professor of Medical Jurisprudence* at the age of 24 years *in 1882.* He initially became famous as a medicolegal expert at this time in ***Case of Burke and Hare*** and later produced the first complete British pharmacopoeia.
- This century was marked for substantial developments in medicolegal relations and by severe struggles to encourage the more effective use of scientific methods. In courts of the past, the proof of innocence in the criminals was essentially based on *superstitions* and *physical evidences. The criminal defending himself was expected to display his innocence under various methods applied, such as:*
  - *Trial by ordeal:* It was common in all countries and some of the examples in practice were:
    - *The test of fire:* The defendant was required to carry hot coal or iron bar and if his body got burnt, he was declared guilty.
    - *The ordeal by water:* Here the accused held submerged in water for sometime, was disproved to be guilty if did not turn unconscious.
    - *The poison test:* The deadly poisons were forced upon the accused and slightest discomfort shown by him declared his guilt.
    - *Death by compurgator:* Parties in both criminal and civil cases could satisfy the demands of the court by swearing to the facts under Christian oath.

In 19th Century AD

One of the greatest boon implemented to the mentally unsound in English law during this century was *McNaughten Rule.*

Alfred Swaine Taylor is the most famous name in *English Legal Medicine in 19th Century.* He became a Professor of Medical Jurisprudence at the Guy's Hospital Medical School in 1834. This century happens to have been associated with several contributions to the subject and most popular among them are:

Alfred Swaine Taylor's first edition of *Principle and Practice of Medical Jurisprudence* published in the year 1865. The book has been revised through several editions by reputed medicolegalists of the period and still accepted as a classic book on the subject.

FORENSIC MEDICINE IN INDIA

Indian Forensic medicine and its evolution can be perceived through 3250-200 BC in the *Indus Valley Civilization*, and other treatises on Indian history such as *Manusmriti (3102 BC), Vedic Literatures of Vedic age (2000-1000 BC), Agnivesa Charaka Samhita (700 BC). Sushruta Samhita (200-300 AD)* and *Kautilya's (Chanakya's) Arthashastra (300-500 AD),* though adds to this, the most recent well recorded evolutions occurred during 1000-1600 AD. During this period because of *Turks, Pathans, Mughals,* etc. invaders who came to plunder, spread religion or to rule over for short intervening periods, law and order in India was not proper and adequate. The Hindu rulers, more or less followed the laws prescribed by *Manusmriti* and *Kautilya's Arthashasthra*, but the *Muslim rulers* who ruled with laws based on *Koran, Hadis* and *Sara.*[11,13,14] During Mughal period, though the crimes and punishments remained same as in the past, unnatural sexual offences flourished more and execution by trampling under elephants feet or throwing to wild animals, were implemented in addition.[3,5] The Portuguese, Dutch and French, though ruled India in 16th, 17th and 18th AD century respectively, it was the English East India Company not only settled but ultimately conquered the whole of India and introduced modern medicine and legal procedures mostly similar to the system prevalent in the British isles.[13]

Inquest by Coroner's system was introduced in Calcutta (Kolkata) and Bombay (Mumbai) by Coroner's Act, 1871 with Police system all over rest of the country. The first medical college of the country, *Medical College of Calcutta* commenced in the year 1835 and the first chair in Medical Jurisprudence was established in 1845 with *Dr CTO Woodford* as the first Professor of Medical Jurisprudence. The first postmortem in India was held by *Dr Buckley* in Madras (Chennai) in 1663 in a case of suspected *Arsenic poisoning.*[10,12,13] *The Indian Penal Code* was promulgated by *Act XIV of 1860* and thus codified various crimes and punishments. *The Criminal Procedure Code* was enacted by the *Act XXV of 1861 and Act VIII of 1869* streamlined the criminal procedures. *Indian Evidence Act of 1872* also codified laws in respect of evidence in case of trials in the courts. After the independence in the year 1947, new amendments and statutes were added to these Acts. The terminology *Medical Jurisprudence* changed to *Forensic Medicine* after the independence. *Dr Jaising P Modi* was *first Indian Physician* who handled cases of *medico-legal nature* provided norms to suit the Indian atmosphere and conditions in *crime investigation* marking the definite role for doctors, which coined him, the title of *Father of Indian Forensic Medicine*. He also wrote the first Indian textbook *Medical Jurisprudence and Toxicology.*

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Part II: Medical Jurisprudence

Chapter 3

Doctor and the Law

INTRODUCTION

As per existing practice, the law relating to criminal procedure applicable to all criminal proceedings in India (except Jammu and Kashmir) is contained in *Criminal Procedure Code (CrPC) 1973*, which came into force with effect from April 1st, 1974.[1-4] The entire law is covered in *484 sections* in *two schedules*. These two provide machinery for the punishment of offenders against the substantive criminal law of the land.[5-7]

It is a fact that the *CrPC* compliments *Indian Penal Code (IPC)* which defines the various offenses and provides the punishments. Further, though *CrPC* is procedural law, it covers many other matters. It deals with the *constitution and structure of criminal courts, their classification and powers and prescribes the procedure* for criminal proceedings.[4,8-11]

According to Mehta HS,[12] law as laid down by the State and the Parliament is known as Codified Law or Statute Law. Besides this there are certain laws made by judges known as Common Law or Law of Torts, which includes certain wrongs or injuries caused by one man to another, which are usually not covered by statute law.[4]

COURTS IN INDIA

Courts of Law in India are of two types:[4] *Civil Courts* and *Criminal Courts*. Civil courts try only *civil cases*, whereas criminal courts try only *criminal cases.* The criminal courts further belong to different categories namely, *Supreme Court, High Court, Sessions Court,* and *Magistrates Court.* Recently, Government of India has set up certain *Fast Track Courts* for the speedy disposal of cases. These courts have the status of additional session courts.[1,4,13] Supreme Court can try both civil and criminal cases. A medical man may be deposed in both *civil* and *criminal courts,* but mostly in the latter. The *criminal courts* and their powers are discussed in this chapter.[1,3,5]

Supreme Court

Supreme Court is the *highest judicial tribunal of the country* and is located at New Delhi, the capital of India.

Powers of Supreme Court:

- It is a court of appeal.
- It supervises and interprets law in the country.
- The law declared by Supreme Court is binding on all other courts of the country.
- It usually takes the cases referred from State High Courts. However, cases can also be filed directly in Supreme Court.
- It can pass any sentence stated in law.

High Court

High Court is the highest judicial tribunal in the State and is located usually in the State Capital. *However, some of the High Courts not located in the State Capital and they are: Kerala—Cochin, MP—Jabalpur, Assam—Guwhati, Orissa—Cuttack, and UP—Allahabad.*

Powers of High Court:

- It is also a court of appeal.
- It can take up all cases of criminal offenses.
- It can pass all sentences authorised by the law.

Session's Court (District Session's Court)

This is the highest judicial tribunal for district and is located in the district head quarters. The court is presided over by the *Sessions Judge*, appointed by the High Court. High Court may also appoint *Additional Sessions Judges* and *Assistant Sessions Judges* to exercise jurisdiction in the court of sessions.

Powers of Session Courts:

- It takes up only the cases of criminal offenses referred by Magistrate's courts.
- It can pass all sentences authorised by the law; however, the death sentence passed by it has to be confirmed by the High Court.

Magistrate's Court

Magistrate's courts are criminal courts presided over by the *Judicial/ Metropolitan Magistrates*. In every district the State Government after consultation with High Court may establish as many of *Courts of Judicial Magistrate* as it may consider necessary. Depending on the revised set up of courts and allocation of magisterial functions, magistrates belong to two categories, namely judicial magistrates and executive magistrates (Fig. 3.1). The former are appointed by and are under the control of High Court, while the latter, as per CrPC, Section 20, are appointed by and are under the control of the State Government. Thus broadly speaking functions, which are essentially judicial in nature, are the concern of judicial magistrates, while the functions which are 'police' and administrative in nature are the concern of executive magistrates.

Executive Magistrates

As per section 20 CrPC, executive magistrate in a district could be – district magistrate, additional district magistrate (wherever necessary), subdivisional magistrate, or subordinate executive magistrate (Fig. 3.2).

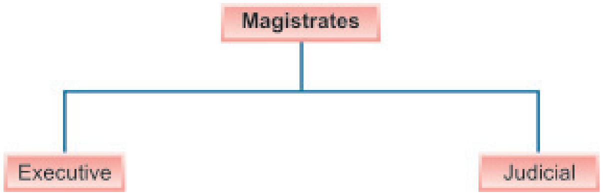

Magistrates
Executive
Judicial

Fig. 3.1: Different types of magistrates in general

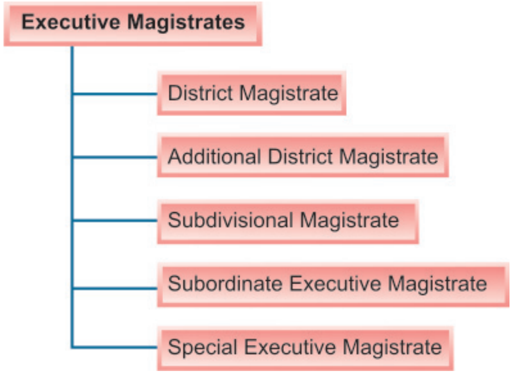

Executive Magistrates
District Magistrate
Additional District Magistrate
Subdivisional Magistrate
Subordinate Executive Magistrate
Special Executive Magistrate

Fig. 3.2: Different types of executive magistrates

The State Government if it thinks necessary may appoint special executive magistrate (section 21, CrPC). Executive magistrates are usually officers of *revenue department*, like *District Collector, Subcollector* or *Tehsildar*, and placed *in charge* of a district, subdivision or *taluk* and have all the powers of a district or *subdivisional magistrate*.

Judicial Magistrates

On the judicial side magistracy differs according to the population figures. Cities with a population of less than one million are considered nonmetropolitan cities, while those with more than one million are considered as metropolitan areas. According to section 11, 12 CrPC, judicial magistrates in an order of hierarchy in a non-metropolitan area are (Fig. 3.3) Chief Judicial Magistrate, Additional Chief Judicial Magistrate, Subdivisional Judicial Magistrate, and Judicial Magistrate First Class. As per Section 13 CrPC the High Court in consultation with Central/State Government may appoint *Special Judicial Magistrates.* According to section 16, 17, CrPC, judicial magistrates in a metropolitan area in an order of hierarchy (Fig. 3.3) are—Chief Metropolitan Magistrate, Additional Chief Metropolitan Magistrate, and Special Metropolitan Magistrate. As per Section 18 CrPC the High Court in consultation with Central/State Government may appoint *Special Metropolitan Magistrates* also. Figure 3.3 highlights different types of magistrates empirical in India.

Functions of Chief Judicial Magistrate (CJM)[4]

- CJM will be the *chief of all other Judicial Magistrates* in the district.
- CJM will *allocate* work to different courts and *supervise* their functions in the district.
- He can pass *any sentence* authorised by the law, *except* death sentence, sentence of life imprisonment or imprisonment for more than 7 years.

The court of Chief Metropolitan Magistrate shall have the same powers as that of the Chief Judicial Magistrate.

In addition he will exercise judicial powers *within metropolitan area, over the port area of the town, over the limits of navigable rivers or waterways* leading there to the town.[2-4,12]

Powers of Judicial Magistrates

Powers of different judicial magistrates in order of their ranks are tabulated in Table 3.1.

All magistrates are authorised to award twice the amount of imprisonment; he or she is permitted to order against two or more counts of offenses in one trial. But in no case one can be imprisoned for more than *14 years* by any magistrate (*Section 81, CrPC*).[4]

Special Magistrate

He or she could be a *metropolitan, judicial* or *executive magistrate*, appointed for special purposes, as for example to try cases of *rioting* when a number of people are arrested. They

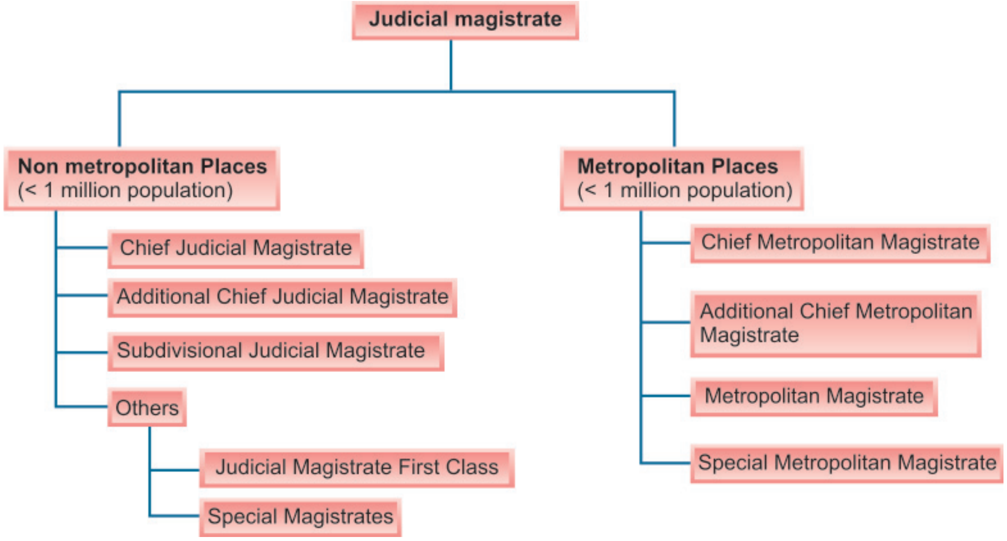

Judicial magistrate
Non metropolitan Places
(< 1 million population)
Chief Judicial Magistrate
Additional Chief Judicial Magistrate
Subdivisional Judicial Magistrate
Others
Judicial Magistrate First Class
Special Magistrates
Metropolitan Places
(< 1 million population)
Chief Metropolitan Magistrate
Additional Chief Metropolitan Magistrate
Metropolitan Magistrate
Special Metropolitan Magistrate

Fig. 3.3: Types of judicial magistrates in general

Table 3.1: Enumerating the powers of different types of magistrates

Type of Magistrate	*Death sentence*	*Imprisonment*	*Solitary confinement*	*Fine*
Chief Judicial Magistrate (CJM)	No	Up to 7 yr	Yes	Any amount
First Class Magistrate (JMFC)	No	Up to 3 yr	Yes	Rs. 5000

are also appointed whenever a regular magistrate cannot cope up with *extra load of work* or the *enquiry* has to be completed within a certain time limit. These magistrates could be of *any class*.[4,13]

Railway Magistrate: He or she will be of the rank of *First Class Judicial Magistrate* and is appointed to try cases of offenses under *The Railway Act*.[1,4,13,14]

Magistrate in Juvenile Court (Juvenile Magistrate): This is a principal magistrate/ a chief judicial magistrate and usually a *woman* and she presides over a *Juvenile Court* and tries *juvenile offenders,*[15] who are children less than eighteen years of age (Juvenile Justice Act, 2000) and are accused of having committed a crime. These offenders are tried in juvenile courts under the *Children Act, 1960*, and if found guilty are usually not imprisoned or punished as an adult offender but, sent to a *Child Reformation Centre* called *Borstal*.[1,4,14]

Cases Tried by Judicial Magistrates

All judicial magistrates can try cases such as:[1,4,13,14]

- *Warrant case:* It means a case relating to commission of a *cognizable offense*. Thus, a case which makes one liable for arrest without warrant, e.g. homicide, rape, etc. Such cases are usually instituted upon police report, but that can also be done on private complaint.
- *Summons case:* It means that on cases of *noncognizable offenses* and being not a warrant case, a police officer has no authority to arrest without warrant. Usually, these cases are instituted on complaint and being simple case, punishment will usually be imprisonment of one year or less.

Public Prosecutor

He or she being a *public servant* under *section 24 CrPC*, is a legal expert, appointed by Central or State Government for conducting court prosecutions or other proceedings like *appeal,* etc. on behalf of the government. *Doctors when summoned to give evidence can approach him/her for the case file or other such help*. Public prosecutors could be of two ranks: *Additional Public Prosecutor,* and *Assistant Public Prosecutor*.

Coroner's Court

Coroner's Court is a court of inquiry and not court for trial presided by the officer appointed by the government, called Coroner.[8,9,10,12] *He is helped and assisted by some persons known as members of Jury. The jury consists of 5, 7, 9, 11, 13 or 15 persons (usually 7), who are men of education and of social position. This court existed in India at Mumbai and has been abolished in India since 1999.*[1,13,20]

Duties and Powers of a Coroner

Coroner is of the rank of First Class Judicial Magistrate, but he cannot pass a sentence. Various duties and powers of a coroner are enumerated below:

- *To hold inquiry in all cases of all unnatural or suspicious deaths, death of a prisoner, etc. dying within his jurisdiction.*
- *To view the dead body and decide whether to hold an inquiry or not.*
- *He can order any medical man usually a Police Surgeon, to hold postmortem examination and to summon him to give evidence in his court.*
- *He can also summon other persons as expert witness.*
- *He can order for exhumation examination of a dead body for identification and for medicolegal postmortem examination.*
- *When a verdict of foul play is established in his court, he can issue warrant for arrest of the accused person for trial in the Magistrate's Court. If the accused cannot be identified, he usually returns an* ***open verdict*** *against this unknown person.*

Open verdict: It means that the inquest is adjourned indefinitely due to want of information and could be reopened at any later date if further information becomes available.

Indications

- When cause of death is not found after autopsy due to putrefaction.
- In cases of poisoning where evidence is not available to differentiate between accident and suicide.

After the inquest, he forwards a copy to the Commissioner of Police.

- He can grant remuneration to the medical man for attending the court for giving evidence. The fee offered is usually the traveling expense.
- He can appoint a Deputy Coroner during his illness or unavoidable absence (it is a special power, which cannot be ordered, by a magistrate). Table 3.2 enumerates the differences between the coroner's and magistrate's courts.

Legal Sentences that can be Passed under Law

As per *Section 53, IPC,* on conviction, criminals are punished by:

- Death sentence, which is to be passed by court of sessions, subject to confirmation by High Court.
- Life imprisonment is to be passed by court of sessions, the time usually comprises 20 years, which can be *reduced* to 14 years for *good behaviour* of the prisoner.
- Imprisonment types:
  - Rigorous imprisonment with hard labour—all courts and First Class Magistrate can pass this order.
  - Simple imprisonment—all courts and magistrates can pass this order.
  - Solitary imprisonment—all courts and First Class Magistrates can pass this order.
- *Monetary fine:* High Court and Sessions Court can impose any amount of fine. But First Class Magistrate and Second Class Magistrate cannot impose more than Rs. 5000/- and Rs. 1000/- respectively.
- *Attachment of movable property:* This can be done by High Court, Sessions Court, and Chief Judicial/Metropolitan Magistrate as per court's direction and power.
- *Detention in reformatories:* This can be ordered by Chief Judicial Magistrate or Judicial Magistrate of Juvenile Court, where the offender is below 18 years and sent to Reformatory Centres/ Borstal Schools.[14]
- Usually court can order *detention till rise of court* for *contempt of court*.

Table 3.2: Differences between coroner's and magistrate's courts

Coroner's court	*Magistrate's court*
• It is only a court of inquiry.	• It is a court of trial.
• The accused need not be present during the trial.	• The accused must be present during the trial.
• Coroner has no power to award the punishment to the accused but can punish those guilty of contempt of court, e.g. nonattendance.	• The magistrate can impose fine and punishment as prescribed by the law to the accused as well to those guilty of contempt of court.

EXHUMATION

Definition

Exhumation is defined as a *lawful disinterment* or digging out of a buried dead body from the grave (Figs 3.4A to K) for medicolegal examination.[1,3,7,8,11,18,20]

Objectives

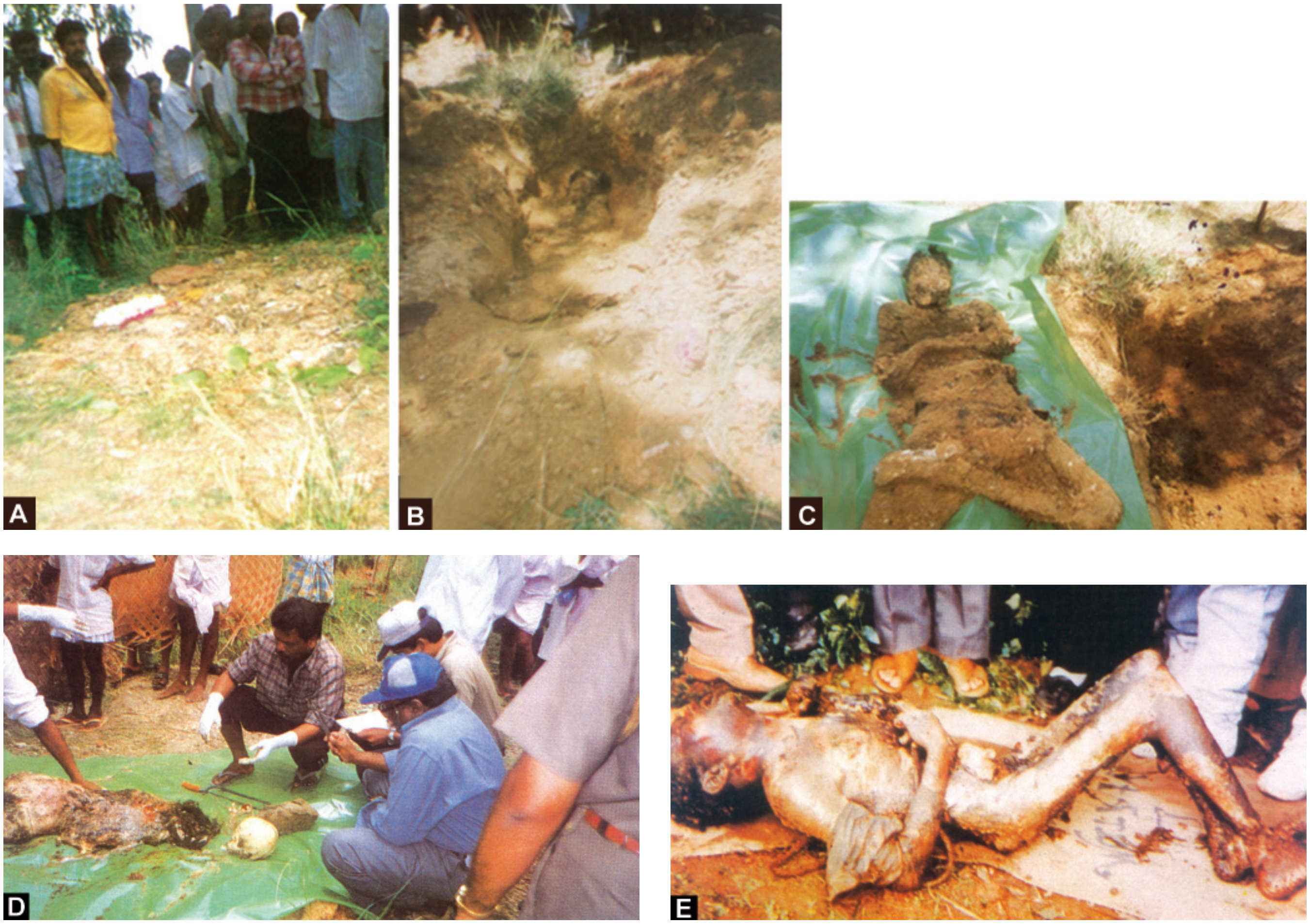
Exhumation is done with definite objectives and basically they are:

- Identification, i.e. confirming the individuality for civil or criminal purposes.
- Autopsy with or without chemical examination of viscera in case of deaths, in which foul play is suspected.
- Second autopsy, when the first autopsy report is ambiguous.

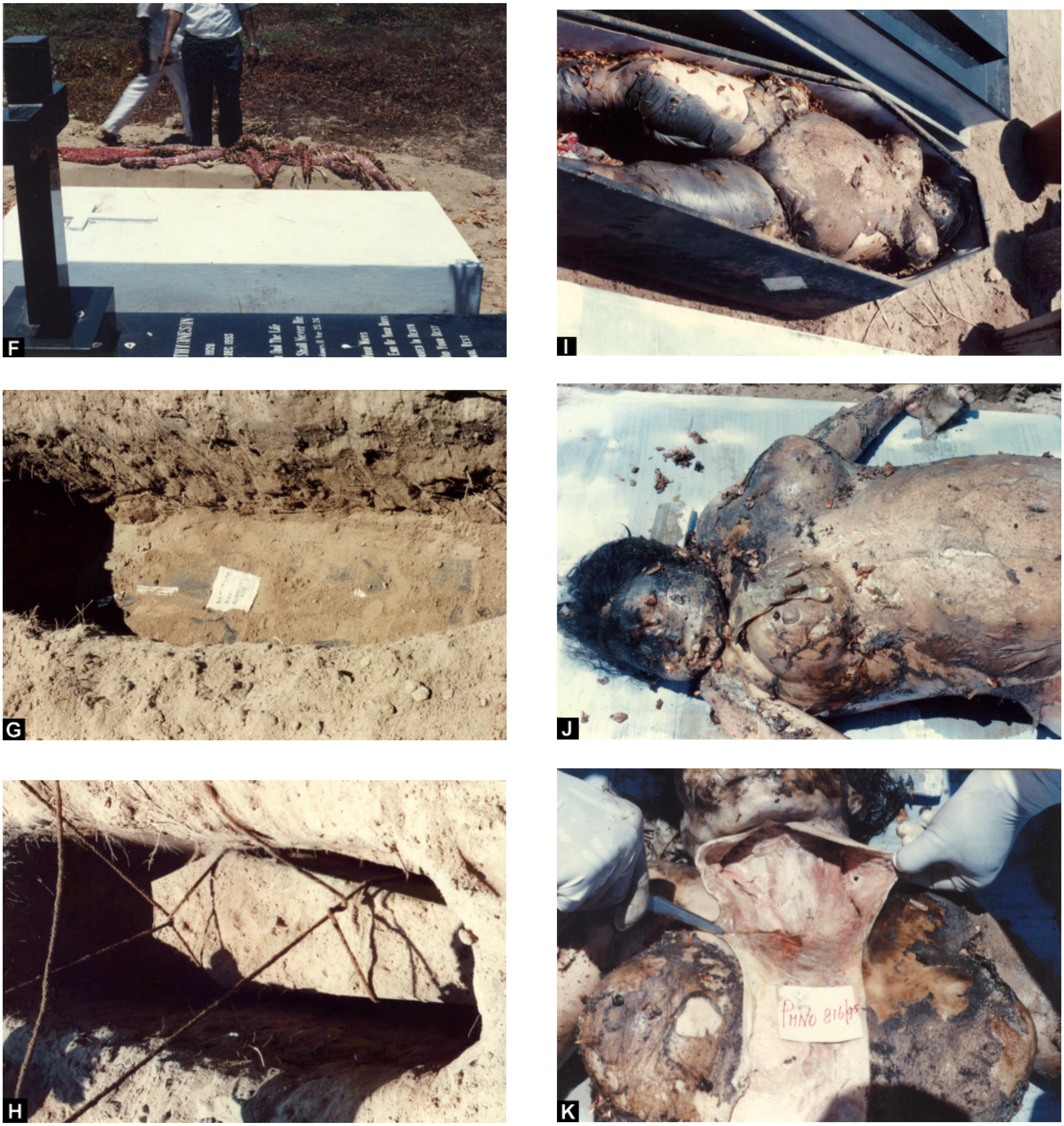
Procedures

Exhumation is done under the *order of appropriate officers*, in the presence of a police officer, during daytime, preferably in the forenoon.[3,5,12]

1. District or Subdivisional Magistrates or *Tehsildar* are specially empowered to order for exhumation. *Note:* Police officer cannot order for exhumation.
2. Medical officer's presence is required for doing the medicolegal postmortem examination, which may have to be done at the spot covered away from the public or in a close-by morgue.
3. The medical officer should preferably stand in the wind side of the body to avoid inhalation of gases.



Figs 3.4A to E: *Exhumation procedures:* (A) Identifying the grave, (B) Dug open grave exposing the body burried, (C) Dead body lifted up for postmortem examination, (D) Conducting the postmortem examination on exhumed cadaver, (*Courtesy*: Dr KWD Ravi Chandar, Professor and Head, Department of Forensic Medicine, Mysore Medical College, Mysore) (E) Killed by throttling and then attempted to conceal the crime by burning, failing which the body was burried. Exhumation ordered later, followed by autopsy, confirmed actual cause of death and crime



Figs 3.4F to K: *Exhumation procedure:* (F) Identifying the grave, (G) Dug open grave exposing the coffin, (H) Coffin being lifted up, (I-J) Coffin opened with exposure of the dead body-putrefied, (K) Autopsy performed (*Courtesy:* Capt. Dr B Santha Kumar, Professor and Head, Department of Forensic Medicine, Govt. Stanley Medical College, Chennai)

Precautions at Exhumation

- The grave is first identified and after the grave is dug, the undertaker identifies the coffin (Fig. 3.4A), if any.
- The body has to be identified by as many persons as possible before sending for postmortem examination.
- Following viscera and materials are sent for chemical analysis:
  - About 500 gm of soil from above, below and in actual contact with the body.
  - Hairs from head and pubic region.
  - Nails, teeth and bones.
  - Viscera such as liver, stomach and intestines.

Time Limit for Exhumation

In India and England, there is no time limit but in countries like France, Scotland and Germany, etc. *10, 20, 30 years* respectively are the time limits for exhumation.

INQUEST

Definition

Inquest is defined as the preliminary inquiry into the cause of sudden, suspicious and unnatural death, which is apparently not due to natural causes.

Explanation

In case of unnatural deaths (Fig. 3.5), an urgent investigation into the cause of death is a must to apprehend and punish the criminal. Thus, inquest is a judicial inquiry and the term "inquiry" means an action that extends to beyond what can be observed with person's own eyes.

Types of Inquest

Globally, there are four types of inquests in practice and these are *magistrate's inquest, police inquest, coroner's inquest* and *medical examiner's system of inquest*. Currently, in India only magistrate and police inquests are in practice. Medical examiner's system is practiced in certain States of USA.

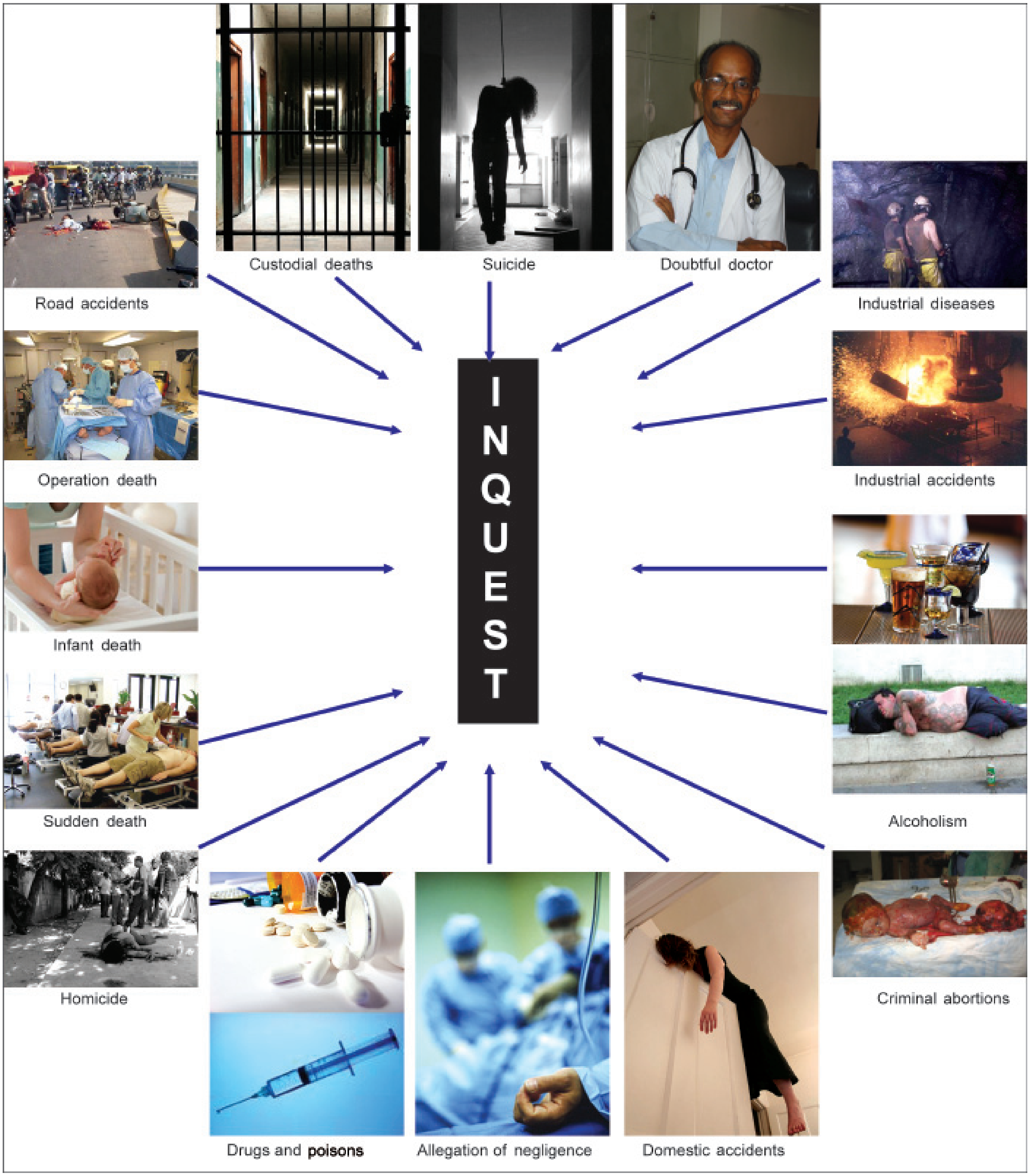

INQUEST
Road accidents
Custodial deaths
Suicide
Doubtful doctor
Industrial diseases
Operation death
Industrial accidents
Infant death
Sudden death
Alcoholism
Homicide
Criminal abortions
Drugs and poisons
Allegation of negligence
Domestic accidents

Fig. 3.5: Indications for inquest

Each of these, except Coroner's system is discussed individually. Figure 3.5 illustrates the various indications for inquest in Indian circumstances.

Procurator Fiscal

The *procurator fiscal* is a type of inquest, charged statutorily with the duty of making public enquiry into the causes of fatal accidents, and in special circumstances of sudden death in Scotland.[17]

Magistrate's Inquest

Magistrate's inquest[1,3,4,13,20] is an inquest conducted by a District Magistrate, Subdivisional Magistrate, and magistrate of the first class rank or any other magistrate as empowered by the State Government, such as Collector, Deputy Collector or *Tehsildar* (Executive Magistrates).

Special Magistrate's inquest[1,3,4,13,20] are held in cases of:

- Lock-up deaths
- Deaths while under police interrogation
- Deaths in prison
- Deaths in police custody
- Deaths due to police firing
- Exhumation
- Alleged dowry death
- In all cases where the police normally conducts inquests, magistrate can hold an additional inquest, or in place of the police inquest.

Places of Practice

It is practiced throughout in India. However, it is not held routinely, but held only on special indications mentioned above. *Note* that in any case of unnatural death, magistrate may hold an inquest instead of or in addition to the police inquest.

Police Inquest

The inquest is held by police officer *(called Investigating Officer)* not below the rank of *Senior Head Constable.* Figure 3.5 illustrates indications for police/coroner's inquest in India.

Procedure

- The police officer on receipt of information of death proceeds to the place of occurrence and holds an enquiry into the matter in presence of the inhabitants of the locality.
- He then investigates the case and writes a report describing the appearance of the body wounds, stating how they were caused and by which weapon.
- The witnesses are called *panchas; panch (five)* witnesses or *panchayatdars* will sign the same and the inquest report prepared so is known as a *Panchnama.*
- If no foul play is suspected, the dead body is released to the legal heirs of the deceased for the purpose of cremation and disposal.
- In suspicious cases, bodies are sent for postmortem examination to the Government Medical Officer or an authorised forensic/ medicolegal expert, employed in a private medical college.

Postmortem Reports

These are documentary evidence, always written by the doctor who has done the autopsy, in prescribed forms, in *triplicates*, to be served as mentioned below:

- The first copy is sent to the investigating officer, through an authorised constable who collects the same.
- The second copy is sent to the Police Superintendent or Magistrate of the area in a sealed cover.
- The third copy is filed in the office files for further reference.

Note: In private medical colleges where only the qualified forensic experts are allowed to do the medicolegal autopsies, the report must be forwarded to investigating officer through the Head of the Department of Forensic Medicine.

Place of Practice

Police inquest is practiced throughout India.

Medical Examiner's System

Medical examiner's inquest is a type of inquest held by medical practitioner.[16,18-20]

Procedure

- A medical practitioner known as *Medical Examiner* is appointed to perform the functions of coroner.
- He has *no authority* to order arrest of any person.
- He has to *visit the* scene of crime and conduct the inquest and also the postmortem examination. Hence, this is considered *superior* to any other system of inquest.
- Medical examiners system of inquest is not practiced in India. It is held in most of the parts of United States of America.

COURT PROCEDURES IN CRIMINAL COURTS

There are several court procedures, which a doctor may have to know in attending the criminal court and tender his evidence[1,7,8,13,14,18,20] and they are:

- Attendance in court
- Subpoena (summons)
- Warrant
- Conduct money
- Oath taking
- Recording of evidence.

Attendance in Court

Most of the medical reports/medical certificates are not acceptable in the court of law, unless testified in the presence of the accused. Thus, the medical officer as an expert witness will have to attend the court on a particular day, during the trial, for deposition and cross-examination of the contents in the report issued by him to the court.

Subpoena (Summons)

Definition

Subpoena is defined as a document compelling the attendance of a witness on a particular day and time in the court of law under penalty.

Explanation

Subpoena is a written document issued in duplicate by the presiding officer of the court with proper seal and signature, to be served to the witness demanding his/her presence in court punctually on the specified date and time for giving evidence in connection with a particular case and with warning to not to be absent without prior permission of the court. It can be served also to produce any official document or any paper before the court of law (Box 3.1A). However, summons is a milder form of process.

Procedure of Serving the Summons

Usually, it is issued by presiding officer of the court, delivered by a court official or a police constable. Person receiving should sign on the original and keep the duplicate with him.

Box 3.1A: Copy of summons issued by the court of law in Karnataka State, India

Govt. of Karnataka

FORM No. 33

SUMMONS TO WITNESS
(See Sections 61 & 244)

To

WHEREAS complaint has been made before me that **(name of the accused)** . . . . of **(address)** . . . . or is suspected to have committed the offense of **(State the offense concisely with time and place)**. . . . . and it appears to me that you are likely to give material evidence or to produce any document or other thing for the prosecution.

You are hereby summoned to appear before this court on the day.....next at Ten O'clock in this forenoon, to produce such document or thing or to testify what you know concerning the matter of the said complaint and not do depart hence without leave of the court; and you are hereby warned that, if you shall without just excuse neglect or refuse to appear on the said date, a warrant will be issued to compel your attandance.

Dated, this . . . . day of . . . . 201 . . . . .

Seal of the Court Signature

If person summoned to is not available, it may be served to:

- The other major member of the family/ relatives, but not to a servant.
- If the person is a government servant, it may be served through the head of the office in which he or she is employed.
- It may be even affixed on some conspicuous part of the house in which the person summoned ordinarily resides.
- Summons by post: It can even be sent by registered post. However, on these occasions, court may not consider this as being served. If the postal authority returns the cover stating he or she is refusing to receive the same, court considers that it has been served in spite of not receiving it.

Rules of Summons

If a medical officer is summoned to attend two courts on a particular time and day, following *rules* may be opted:

- *Criminal cases* should be given preference over civil cases.
- If both are criminal cases, *higher courts* should be given first preference. However, the medical officer should *inform* the other court which he or she is not attending.
- If both cases are of the same ranking courts, summons received *earlier* should be attended first.
- *Noncompliance to summons* in a civil case, may render one liable to action for *damages,* but in a criminal case, fine or even imprisonment (unless some satisfactory excuse is given) may be ordered.
- He or she cannot leave the court without the permission of the magistrate or the judge.
- If he or she fails to attend summons in time, a warrant can be issued to compel his or her attendance.[4,13,14]
- An attendance certificate (Box 3.1B) will be issued by the court on demand.

Warrant (Witness Warrant)

Definition

Warrant is an authority under the seal and signature of the presiding officer of a court to a person to be arrested and produced before the court to be dealt with according to law. It is a written order from a court, commanding police to perform specified acts/ arrests to produce the witness in court of law.

Types

Warrant could be *bailable warrant (BW)* or *non-bailable warrant (NBW)*, issued through police by a court to compel attendance of the witness in court on a fixed date.[3,4,12,13] A court may issue this in lieu of or in addition to summons for appearance in the court for following reasons:

- If there is reason to believe that he or she will abscond or will not obey the summons.
- If witness has failed to appear before without prior reasonable excuse, though clearly served with summons.
- In a case of *breach of bond of security* for appearing (*vide section 90 CrPC)*.

Conduct Money

Definition

Conduct money is the *fee* offered to a *witness* in *civil case*, at the time of serving of summons to cover the *travelling expenses* for attending the court.

In the civil cases: A Government Medical Officer gets the *conduct money* when he or she serves a summons.

In the criminal cases: Where state is the prosecuting party, Government Medical Officer will *not* get conduct money, but *as per law (Section 312 CrPC)*, he or she will be paid the *travel allowances (TA) by the court*. This is also called *Witness*

Box 3.1B: Copy of certificate of attendance issued on attending summons issued by the court of law in Karnataka State, India

Crl. R.P. 68 Govt. of Karnataka

CERTIFICATE OF ATTENDANCE

Form No. 15 (Criminal)
Certificate of
Attendance

Certified that . . . . appeared before me as a witness on behalf of . . . . in Session's Criminal Case No. . . . . on the file of this Court for . . . . days from. . . . . to. . . . . in this official capacity to dispose the facts within his knowledge and that he has been paid the undermentioned allowances

	Rs.	P
Travelling Allowance	..	
Subsistance Allowance	..	
Total	..	

Court of the . . . . 201 . . . . .

Judge/Magistrate

Bata.[1,4,10,13] In a private criminal case, Government Medical Officers will get fee, private practitioners will get fee from the *state* or the *private party* concerned.

Oath Taking

Before deposition of evidence begins, witness must take an *oath* or *affirmation*. *Unoathed* evidence is not admissible to the court of law, except when a person is *below 7 years of age*.[18,20]

Oath

The witness is to take oath by reading or quoting the following with the help of bench clerk:

- *I swear before Almighty God that the evidence I shall give to the court, touching the matter in question, shall be truth, the whole truth and nothing but truth.*
- *The evidence which I shall give to the court shall be truth, the whole truth and nothing but truth. So help me God (Indian Court of Law).*[2,4]
- *I swear to tell the truth, the whole truth, and nothing but truth, so help me God.*

If witness desires to give his evidence on solemn affirmation he or she will take oath by saying:

- *I solemnly affirm that the evidence I shall give to the court, touching the matter in question, shall be truth, the whole truth and nothing but truth.*
- *I solemnly affirm that the evidence which I shall give to the court shall be truth, the whole truth and nothing but truth.*[2,4]
- *I solemnly affirm, to the truth, the whole truth, and nothing but truth (USA Court of Law Oath).*[16]

As per *section 191, IPC,* a witness who willfully makes a false statement after taking oath is considered as guilty of crime of *perjury* (giving false evidence under oath) and may be prosecuted. Punishment for perjury is dealt with as per section 193, IPC.

Recording of Evidence

After taking the oath, the recording of evidence will be done by following (Fig. 3.6) four steps:[1-8,10,13,14,18,20]

1. Examination-in-chief
2. Crossexamination
3. Re-examination
4. Court questions.

Figure 3.7, provides a bird's eye view of court scene in the form a sketch for familiarising the court, providing the place/ locations of various court officials, public prosecutor, defense counsel (lawyer), witness (doctor), accused (criminal), judge/ magistrate/court presiding officer, court audiance, etc. This figure will be of great help to every doctor who is a beginer visiting the court as witness for first time.

Examination-in-chief

- This is done by prosecution side, i.e. by the *public prosecutor* in State cases or by the lawyer engaged by the prosecution side in private cases. In other words here the party who calls to give evidence in the court examines the witness. In criminal cases the public prosecutor does it, while in civil cases, the pleader of the party who cited him as a witness does it. The objective of this examination is to bring out the facts of the case known to the witness concerning the case.
- At this stage, the questions which are put to the witness and answers elicited, are both recorded by the court. Here, witness is to relate the *facts fully within his or her knowledge* regarding the case.
- But if the Judge is *convinced* that the witness is *hostile* (refer page 28), *leading questions* may be allowed by him.
- Usually, no leading questions are allowed during examination-in-chief. However, according to Prof JB Mukharji, they are allowed with the permission of the court under following circumstances:[18]

Examination-in-chief (By public prosecutor)

Q. “Doctor, what precautions are necessary before injecting penicillin?”

A. “A sensitivity test should be done”

Cross-examination (By defense counsel/lawyer)

Q. “Doctor, is it possible that an anaphylactic reaction may still occur even though the sensitivity test is negative?”

A. “Yes”

Re-examination (By public prosecutor)

Q. “Doctor, what would be the difference in the anaphylactic reaction in a case where the test was positive and another case in which it was negative?:”

A. “In the former case, it would be far more severe; in the latter case, it would be mild.”

Court questions (By presiding officer/magistrate/judge)

Q. “Doctor, is there no way by which one can be absolutely certain if the patient is sensitive to penicillin or not?”

A. No.

Fig. 3.6: Recording of evidence in court of law. (*Note*—Doctor as witness-in the witness box)
(*Courtesy:* All performers are students of Meenakshi Medical College and Research Institute, Kanchipuram, Tamil Nadu at the under graduate Convention and Mock Trial Contest of CME in Basic Law and Ethics organised by the Department of Forensic Medicine 4-6 April 2007, on behalf of National Foundation of Clinical Forensic Medicine)

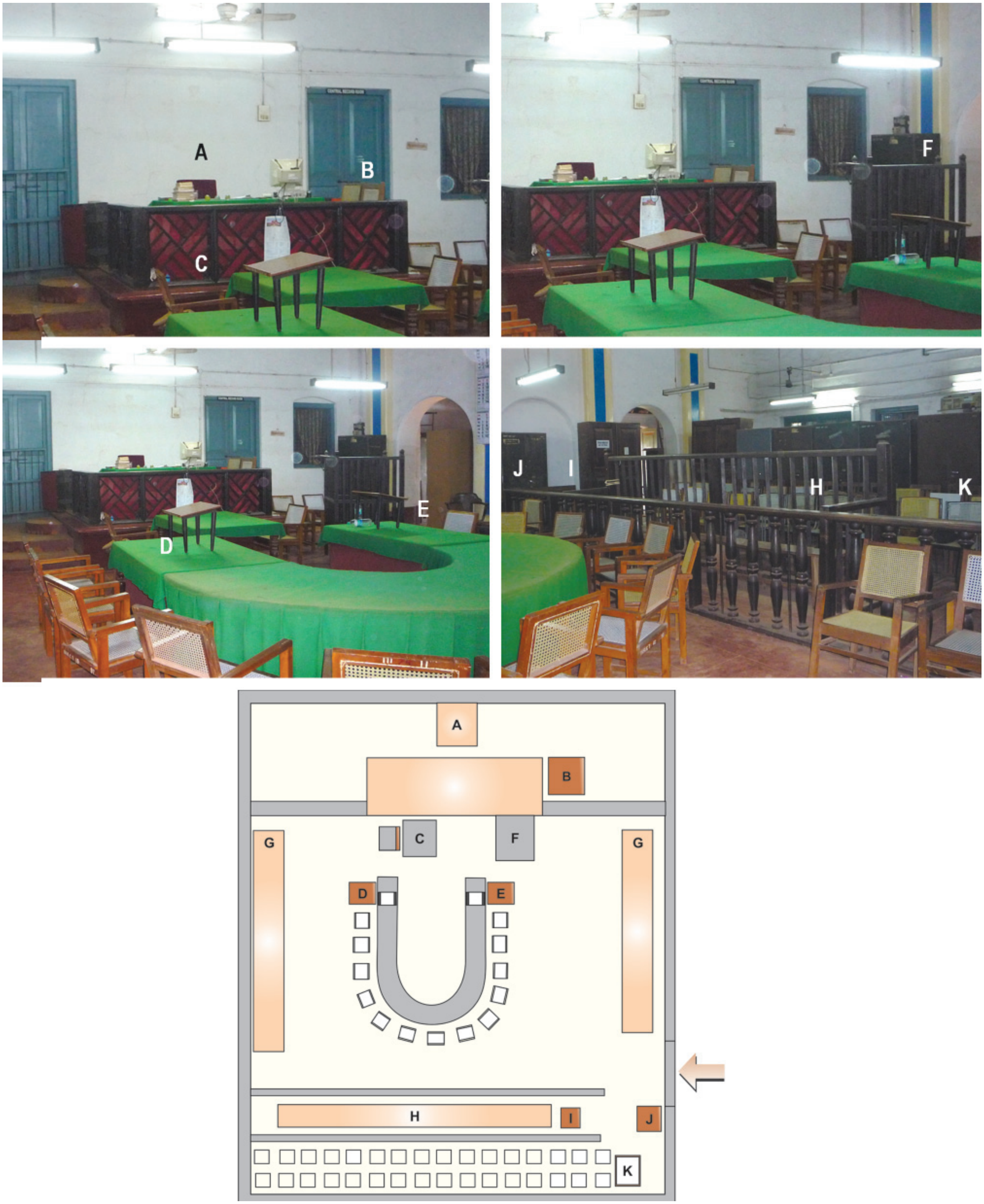


Fig. 3.7: Photographs and sketch presenting bird's eye view of sessions courts in India: A—Presiding officer/Judge/Magistrate; B—Court stenographer; C—Bench clerk, D—Defense lawyer; E—Public prosecutor; F—Doctor (witness box, usually on the left side of the presiding officer); G—Waiting place for other expert witnesses arrived on summons; H—Criminal in custody; I—Police constable; J—Court announcer; K—Court audience (Photographs by Dr Ritesh G, Manager, Assoc. Professor of Forensic Medicine; KMC, Mangalore, Karnataka)

- While making introductory remarks/ presenting undisputed matters (example: when the name, occupation, etc. of the witness are asked).
- While presenting identification facts – when attention of witness is drawn for identifying person/thing.
- To assert memory.
- While giving contradictions.
- When a witness is declared as hostile witness.

Leading Questions

Definition: Leading questions are those questions, which suggest their own answers conclusively, may be by a simple 'yes' or 'no', or the answer wished for, e.g.:

Routinely to a witness who had seen Mr A (accused) hitting Mr B (victim) by an iron bar on Sunday evening at 6 pm, the question asked will be:

- *Where had you been at that time?*
- *What did you see?*
- *With what object did Mr A hit? Etc.*

However, when the witness becomes hostile, questions become leading questions and will be asked as follows:

On Sunday, 6 pm evening did you see Mr A hit Mr B by iron bar?

Cross-examination

The lawyer of the opposite party, i.e. defense lawyer, conducts the cross-examination.

He or she will always try to weaken the evidence given by the witness during examination-in-chief and try to prove before the court that the evidence given is untrustworthy and unbelievable.

In this stage, leading questions are allowed. During this stage of cross-examination, witness must be very careful and should not answer the question, if he or she does not understand it properly.

There is no time limit for a cross-examination. It may last for hours or even days.

The judge or the magistrate may disallow *irrelevant questions (section 152 IEA)* and cut short the cross-examination.

Note: French law takes a person to be always accused, where as in English and Indian law a person is to be proved an accused (benefit of doubt is given to the person accused; the person is innocent, till otherwise proved).

Re-examination

The prosecution side (Public prosecutor) does this, in order to clear up certain ambiguities or discrepancies that might have been made by the witness in the cross-examination.

However, no question relating to new matters, are to be asked (without the permission of the court). If new matters are asked, the defense will take another chance of cross-examination.

Court Questions

The judge/magistrate/jury can ask any question to the witness during any stage of trial in order to clarify certain facts/findings of case.

The evidence thus recorded by presiding officer - judge/magistrate should be next given to the witness for his signature, should be read fully by the witness, and signed.

If there are any corrections in the evidence recorded, all typographic errors must be corrected and got signed by the presiding officer of the court. The witness is liable to be called again, if necessary, if further evidence is needed.

MEDICAL EVIDENCE

Medical evidence could be in two[1,11,13,14,18,20,23] forms, namely—*Documentary evidence* and *Oral evidence.*

Documentary Evidence

These are again of several types and following constitute few examples:

- Medical certificates for ill health, insanity, death, etc.
- Medicolegal reports such as injury report, postmortem report, age report, reports on sexual offenses like rape.
- Dying declaration and dying deposition.

Medical Certificates

Medical certificates are documents issued by a doctor only after confirming for what reasons it is being issued. It usually consists of three parts:

1. Date, time and place of examination.
2. Name, age, sex of the person to be examined with his/her signature and two identification marks.
3. Final opinion: Include number of days of rest/such other advices given with reasoning. Name, Signature, Designation, Medical Council Registration Number, and address of the doctor with rubber stamp/seal should follow it.

Death Certificate

Death certificate must be issued in a specified manner, as suggested by the World Health Organisation (WHO format) mentioning facts like name, age, sex, address, cause of death, etc. Death certificate should be withheld if there is any suspicion of foul play.[11,20]

Medicolegal Reports

Medicolegal reports are documents prepared by the Medical Officers after being asked or ordered by the Police Officer or Magistrate. They are often related to criminal cases and should include four parts:[11,20]

- *Preamble:* Date, place and time of examination including name, age, sex, signature/left thumb fingerprint, two identification marks, consent, etc. of the person to be examined.
- *Reasoning part:* Facts observed on examination.
- *Opinion:* The inference drawn from facts.
- *Concluding part:* Name and signature, designation, registration number, address of the doctor certifying, with his/her rubber stamp/seal.

A medicolegal report may not be admitted as evidence unless the doctor attends the court and testifies the facts under oath. Defense Council can also cross-examine the doctor over the report.

Dying Declaration

Definition

Dying declaration is a statement, verbal or written (or even by gestures) made by a deceased person before his/her death, relating to the circumstances leading to death.

Explanation

Dying declaration should fulfill following ingredients:

- Person making the statement is likely to die.
- Statement must be made as to the cause of his/her death or the circumstances resulting in his/her death.
- The cause and manner of his/her death must be in question.
- Statement must be complete.
- Declarant must be of competent mental state to make the statement.

Procedure

Following steps must be observed and taken care of:

- As soon as the severely injured person is brought to a hospital casualty, it is the duty of the medical officer to inform the magistrate to have a dying declaration recorded.
- If the doctor thinks that the patient will not survive till the arrival of the magistrate, he/she can record the same himself or herself.
- The treatment should not be delayed or avoided while recording dying declaration.
- The major role the doctor has to play and help the magistrate in recording the dying declaration is that he/she should determine the condition of the patient and assess whether the dying person is mentally sound *(compos mentis)*.
- If the declarant dies or becomes unconscious while recording, the doctor should record the information obtained up to the point.
- It is usually recorded in forms of questions and answers without prompting (best in patient's own words).
- It should be recorded in the identical words of the patient in his/her own vernacular.
- No suggestions or leading questions are allowed.
- No outsider is allowed to be present.
- After completion of writing, it is read over to the patient who has to approve the same by his/her signature on the same or if illiterate by taking his/her left hand thumb impression, inserted as proof of approval. Signature of two witnesses is mandatory. Even if doctor records it, witnesses must also sign the same. It is then dispatched to the court for official use.

Medicolegal Importance

- Admissibility of dying declaration is limited to criminal cases where the cause of death is being inquired into *(manslaughter)*
- In English Law, dying declaration is only admissible if the person is in full possession of his/her senses and believes that his/her recovery is impossible and death is imminent—the legal assumption being an individual would speak nothing but truth during the last moments of his/her life.
- All dying declarations are not exclusively taken into consideration to judge the guilty.
- All courts base their judgment on the circumstances of the individual case.
- If the victim does not die, then the dying declaration is void and the victim has to testify in the court.

Dying Deposition

Dying deposition is almost a dying declaration. The main difference being that it is always recorded by a magistrate in presence of the accused or his/her lawyer. Legally, the dying deposition is more valuable than dying declaration as the accused has got the opportunity to challenge and cross-examine. The Medical Officer's presence is not indispensable, but he/she may have to certify the mental fitness of the patient. Table 3.3 gives differences between them. However, currently this is not in practice in India.

Oral Evidence

Oral evidence is always superior to documentary evidence in trial for the reasons that, the person has to prove on oath that the evidence is true and is cross-examined. However, a person giving documentary evidence is also supposed to do the same, exceptions being:

- Dying declaration.
- Printed opinion of experts in the form of textbooks, when author is either dead or stays at a very distant place, and to bring him/her would mean unnecessary loss of time and money.
- Evidence previously given in a judicial procedure.
- Deposition of a medical witness in a lower court, attested by the magistrate.
- Chemical examiner's report is sufficient and his/her personal attendance is usually not necessary.

All these documentary evidences are accepted by court as unsworn documents, without any oral testimony.

Types of Oral Evidence

Types of oral evidence could be of two types:

1. *Direct:* Refers to facts, which are seen, heard or perceived by any other sense.
2. *Circumstantial:* It proves one or more of the subsidiary circumstances or associated events.

WITNESS

Definition

Witness is defined as a person who provides evidence about a fact in the court of law under oath and being summoned to court to attend without failure and under penalty.

Classification

Witnesses are classified into following types:

Common Witness

Common witness is that person, who narrates what he/she has heard or perceived or states the facts observed by him/her.

Expert Witness

Expert witness on account of his/her special professional training and skill, is capable of giving opinion or deducing inferences from the facts observed by himself or by others, i.e. Medical Men, Chemical Examiner, Fingerprint Expert, Handwriting Expert, Ballistic Expert, etc.

Medical Man (Doctor)

A medical witness is generally considered as both common and expert witness.

- Common because he/she can say the size, positions and number of wounds, etc.

Table 3.3: The differences between dying declaration and dying deposition

Criteria	*Dying declaration*	*Dying deposition*
Made to	Medical officer/ Magistrate	Magistrate only
Oath	Not necessary	Necessary
Presence of accused/his lawyer	Not needed	Allowed
Cross-examination	Is not possible	Permitted
Legal value	Less	More

- Expert, because he/she can say with certainty whether the wound is antemortem or postmortem; accidental, homicidal or suicidal; age of wounds; and can give opinion about the cause of death.

Tips to the Expert Witness

Forensic practitioner is often called to court to testify as an expert witness. The following are some guidelines for effective expert witness testimony:

- Before going to court
  - Keep thorough notes, records, photos, diagrams, etc.
  - Carefully prepare your reports and consider appropriate language, completeness and opinions.
  - Prepare your prosecutor, public prosecutor (PP), assistant public prosecutor (APP) with a pretrial conference.
  - Review the case before you enter the courtroom.
  - Never allow a prosecutor or defense lawyer to push you beyond your area of expertise on opinion. Ask yourself, "can my testimony withstand the evaluation of a competent opposing expert".
- Giving expert testimony
  - Wear conservative clothing. Avoid wearing lapel pins or jewelry, which designate membership in a club, group, or religion.
  - When answering questions, respond to the judges/magistrate, not the lawyer. Talk to the judge/magistrate and make eye contact.
  - Pause before giving your answer. Give the opposing counsel a chance to object. Also, consider what you are going to say.
  - Try to avoid 'ums..' and 'ahs..' when speaking.
  - Present a professional demeanor. It is not only important what you say, but how you say it and how you look saying it.
  - Be aware of your physical presence—how you stand and your body language. Don't fumble with exhibits or props; watch out for change in pockets.
  - Don't fidget or sway when standing.
  - Watch your demeanor. Don't appear too confident (it may be construed as arrogance).
  - Avoid animosity. Appear sincere, objective, polite and fair. Concede, if you don't know the answer.
  - Express your opinions with emphasis. Educate using no technical language. Make your testimony interesting.
  - *Concentrate:* listen to every word of counsel. Don't 'jump the gun' with your replies.
  - Pause to clarify concepts.
  - Remember: a court clerk is recording everything you say. Be specific when giving your testimony.
  - Respond directly to the question being asked.
- Cross-examination
  - You do not have to answer any question 'yes' or 'no'. Explain your rationale.
  - Be careful with questions such as. 'Is it possible that…?' or 'Is it fair to say…?'
  - It is acceptable to say, 'I don't believe, I am qualified to answer that question'
  - Listen to all aspects of the hypothetical question. If you are unsure what the lawyer means, ask for a clarification.
  - If you are not given a chance to explain your answer, ask if you can respond to clarify your statement.
  - Remember your demeanor and attitude. You are not supposed to take sides.
  - The louder and more belligerent the lawyer becomes, the more composed and polite you should become. Don't lose your temper, be cool.
  - If you are asked a particularly lengthy, confusing question simply say, 'I don't understand your question, could you rephrase it?'
- Other points
  - Speak in a manner that lay people can understand. Avoid jargon, especially 'Scientifics'.
  - Go over prior testimony, reports, and exhibits before the trial. Try to have a pretrial conference with the prosecutor.
  - Be objective. You are not an advocate. It should not matter to you whether the case results in a guilty or not guilty verdict. Your job it to be an advocate for your opinion.
  - Use visual aids, analogies, illustrations from everyday life, and the chalkboard.
  - Pay attention to the judge/magistrate. 'Objection sustained' means you cannot answer the previous question.
  - Do not box yourself into a corner. Beware of questions like, 'Have you ever made a mistake?' and 'Is not it possible…'
  - Don't try to 'weasel out' of a difficult question.
  - Be aware of the cross-examination trick of taking an excerpt from a publication out of context or an excerpt from a non-existent article. You can always ask the lawyer to show you the article to refresh your memory or even to read it out.
  - Be able to say: 'I don't know' or 'I was wrong'.
  - Take the lead to meet with and talk with counsel.
  - Mentor new, inexperienced prosecutors. If he/she is new or inexperienced in a given area, be patient and offer your assistance as an expert. Try to remember how you felt the first few times you appeared in court.

Skilled or Scientific Witness

Skilled or scientific witness is one, who has specialised knowledge of technical subjects. He/she may be expert but usually he/she has no first hand knowledge of the particular case.

Two Other Types of Witness

Two other types of witness *hostile witness* and *unfavourable witness* are often encountered in legal practice. These are independent witness who make contradictory statements and the prosecution itself can cross-examine them with permission of the court during trial. However, question of value of their evidence usually arises.[13,21,22]

Hostile Witness

When a witness makes statements against the interest of the party who has called him/her, he/she is declared as a hostile witness.[13] A hostile witness is thus described as one who is not desirous of telling the truth at the instance of the party calling him/her.

Unfavourable Witness

The unfavourable witness is one called by a party to prove a particular fact, but fails to prove such fact or proves an opposite fact.[22]

This makes it necessary that he/she should be cross-examined by the very party who has called him so as to demolish his/her stand. Under Section 142 (IEA) the court can grant permission for asking leading questions and under Section 154

(IEA) for cross-examining the party's own witness which is wholly left to the discretion of court.

The Behaviour of a Doctor in Court (Rules/Conduct and Duties of a Doctor in a Witness Box)

Medical witness must maintain certain principles listed below:

- Study and master the facts of the case before attending the court. Study recent aspects on it.
- Attend the court promptly and punctually, dressed decently, consistent for the dignity as a doctor.
- Doctor, while entering as well as leaving the court room should show his respect to the presiding officer by bowing down or joining hands and also use the word *Your honour* while addressing him/her.
- Usually, as a rule, doctor's evidence is taken soon after his/her arrival as the court relishes his/her importance for time and duty. However, if there is any delay, he/she can politely inform the public prosecutor that he/she is waiting.
- Avoid unnecessary talking about the case with anyone else while in court premises.
- Speak slowly, but loudly and distinctly.
- Use plain language, avoiding superlatives or exageration and technical terms as far as practicable.
- Do not lose temper in a court, because lawyer will try to make you do such. If you do not know a thing, say it plainly. Sometimes, the defense lawyer quotes a passage from a book and you are asked to give your opinion. Do not give opinion unless you yourself read the whole passage, when it may give a totally different meaning.
- Avoid long discussions and theoretical arguments in a court. Answers if possible, should be brief and to the point.
- *Volunteering statement*: A witness is not supposed to volunteer his/her statement in a court, unless called for to do so. This may be true, when the witness is a layman, but not so in case of a medical witness. Though a medical witness is called upon by one side to give evidence in the court, he/she must not forget about *honesty and fair dealings*, his duties to the opposite party. He must remember that he/she must help the court with his/her special knowledge to elicit the truth. Hence, if he/she thinks the court should be appraised of some facts, which have not been asked from him, he should volunteer his statements.

DOCTOR AND SCENE OF CRIME[1-3,7-8,11,14,20,24]

The term *scene of crime* is the place of any suspicious or unnatural death. It is important that all these need an open minded approach along with a good observational capacity and patience. Here the satisfactory outcome basically depends upon the combined efforts made by the various team members and experts visiting the scene. Among these, role of a doctor (medicolegal expert/forensic physician/forensic pathologist) is essential. Ideally speaking, police must invite this doctor to the scene immediately prior to the disturbance of scene or before natural changes in the dead body take place, allowing an expert to comment on time since death and cause of death. However, it is not surprising that the police rarely involves a forensic pathologist in crime scene examination. There are also several occasions when pathologist cannot attend while the body is still at scene. It is still beneficial for the pathologist to go and visit the scene of crime which will help him to obtain assistance in interpretation of the case. Retrospective visit to the actual site and examination of scene might be helpful in deciding certain road traffic accident cases or such other cases. Usually, removal of the body from crime scene is done by the police. Body can be wrapped in a sheet of plastic material avoiding any contamination by foreign material during removal or shifting. It is suggested that every doctor who is involved in crime scene visit/examination must carry a *Crime Scene Examination Kit* of his own and it should include special equipments of his choice, such as:

- Hand lens for examining injuries
- A measuring tape and ruler
- Autopsy instruments
- Personal camera for the record of features of your own interest
- Clean containers (glass/plastic) with proper fitting stoppers/lids and paper/polythene envelops with labels, rubber gloves, swabs, glass slides, etc.
- Suitable thermometer for recording rectal temperature/atmospheric temperatures.

CONDUCT AT THE SCENE

On receiving call from investigating officer (IO), the doctor should report to the scene of crime punctually prior to any interference. At the outset he should make note of the posture, clothing and other features of the body and surroundings. Do not alter the position of the deceased. Always describe things in detail prior to their being picked up or even before touching and examining them. Never make hasty opinions or conclusions. Leave the place only after you have finished your examination completely. Enumerated below are certain points of medicolegal concern which a doctor cannot afford to miss and they are described as follows:

- Prepare notes—On the position of the deceased, highlighting the details about things around.
- Always draw a sketch of the scene mentioning relevant measurements – Sketch is usually helpful in refreshing the memory of the officer and help in establishing the bonafide nature. It would also help in arguing out and establishing the case in absence of photographs. Include particulars about the surroundings around the dead body, covering about the walls, floorings, furniture, doors and windows, fixtures, fittings, etc.
- Photographs may be taken along with. Always remember 'A picture is worth a thousand words'.
- Use handlens, U-V lamp, etc, as they are not only essential, but are also of great help in visualising certain things in detail.
- Collect all the trace evidence materials such as smears and stains of blood, mud, semen, saliva, or any other material, or any poison. These are to be collected for referring to FSL for further examination.
- Collect empty/partly full or partially empty containers—if any. If suspected of containing harmful materials they are noted and collected for further analysis.
- Clothing of the body – look for whether it is normal or disturbed, deranged, such as lifted up skirt with pulled down panties, or unhooked or torn buttons of a blouse or brassieres. They are common in a sexual offence case.
- Tear in the dress worn may also be important to note as it may correspond to the stab wound or firearm wound, etc.
- Blood at the scene—following should be noted:
  - *The amount of blood shed:* Dural sinus / major neck veins such as jugulars, usually shed excessive blood, if torn.
  - *The distribution of blood:* Here look, whether there is a large pool of blood near the body or just a trail of small splashes or drops. Assess whether it spurted from the victim or had flown out in slow stream or fallen drop by drop, etc.

- – *The shape of splashes, drops or smears:* Better record them as noticed.
- – *Blood and its location:* Blood stains may be seen on the wall, furniture, and ceiling or on any other objects in the room, suggestive of arterial spurting from small or medium sized artery, e.g. radial artery at the wrist. Venous bleeding is slow steady flow, which can produce a pool of blood if the victim is at rest and separate drops, more widely spaced suggest that victim was live for sometime and ambulant after the injury.
- – *Bloodstains and its shapes:* Spurts of blood on flat vertical surfaces, e.g. a wall, can produce a series of linear streaks looking like inverted exclamatory marks. Single drops from a vein, differ in appearance according to angle at which the drops fall on a flat surface.
- – *The skin ridge impressions in smears:* Fingerprint experts could be of help.
- – *The relation of the blood to the body:* Pooling of blood under the dead body suggests lack of mobility change in position of the body after death.
- – *Presence of drag marks of blood under the body:* Suggests exact scene of assault and death. Blood trail in the scene indicates that victim received wounds at a distance from place where body was found.
- – *Blood and resembling material stains:* Paint, dye, jam, sauce, tomato ketchup, pan spitting, etc. resemble fresh blood, and can be readily misleading. Rust and tar stains are not easily distinguished from dried bloodstains and need expert examination opinions.
- – *Photograph of bloodstain:* This is certainly useful, but photography should be in colour.

- Seminal stains—may be noticed on the clothing worn by the victim or may be seen at the scene on some other objects like bedsheets, undergarments, handkerchief, in the waste paper basket, used to wipe off the genitalia and then discarded, etc. Mention must be also made about the relationship of these stains to the body in order to correlate or explain the crime.
- Postmortem hypostasis—distribution, colour, and fixing of postmortem hypostasis needs careful study and reporting. Its distribution indicates the position of the dead body after death. For example, stain will be seen in lower extremities, external genitalia, and lower part of the forearms of the dead body found in hanging position. Likewise colour of postmortem lividity can draw attention in poisoning cases, e.g. CO poisoning renders cherry red colouration to the lividity. Thus, lividity can give clue accordingly to the cause of death as well.
- Hairs or fibres at the scene–hairs can be the only clue to the crime happened. Hairs can provide crucial information about the criminal when properly identified. Hence, if hairs are noticed at the scene they must be collected and preserved for further examination at FSL.
- Importance of good illumination–scene always must be examined in proper illumination, with accessories such as handlens, UV light, etc.

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Chapter

4

Ethics of Medical Practice

INTRODUCTION

Ethics is a science of moral values or principles. Medical ethics is thus described as moral principles (*code of conduct)*, which should guide the members of medical profession in their dealings with the patients, their relatives, community, and with other colleagues in profession. The principle objective of the medical profession is to render service to humanity with full respect for dignity of human beings.

Every doctor, whatever is his/her speciality, has to discharge medicolegal responsibilities and to solve medicolegal problems from the very first day of his/her medical practice. Almost everything a doctor does in practice of medicine is in some manner or the other is governed by the legal system. Quite often, a doctor victimizes himself/herself to the litigious tactics of the public, irrespective of whether they are actual causative factors or not.[1]

The major reasons for this are, firstly *ignorance about medical law and ethics* by the young medicos, leading to the consequences of negligent behaviours/failure to discharge compulsory duties towards the patient and the state, amounting to either *infamous conduct* or negligence charges. Secondly, members *of the general public* are becoming increasingly aware of their rights due from a doctor, questioning the legality of issues.[2]

Hence, it is absolutely essential that every member of medical profession clearly knows what exactly are their compulsory duties and proper behaviour towards their patients. They should also know who are the authorities proposing these rules and what penalties one is likely to be punished with in the event of breach of them.

Origin of Medical Ethics is as old as the *origin of Medicine*. To the earliest man, medicine was known in the form of *magic*, *witchcraft, worship and various objects of nature*. To protect themselves from their charlatan effect, ancient men framed set of regulations to control these medical men, this may be marked as the earliest code of ethics. About the medical ethics in India, details mentioned have been noticed in the *first treatise* on *Indian medicine,* i.e. *Agnivesa Charaka Samhita*: Charaka's Oath, supposed to be composed in about 7th century BC. It is said that *specific codes* regarding the training, duties, privileges and social status of the physicians were mentioned in it.[3,4]

CHARAKA'S OATH

According to this, the teacher instructed the disciples, in presence of sacred fire, Brahmans and Physicians thus:

Thou shalt be free from envy, not cause other's death, and pray for the welfare of all creatures. Day and night, thou shalt be engaged in the relief of patients, thou shalt not desert a patient, not commit adultery, be modest in the attire and appearance, not be drunkard or sinful, nor associate with abettors of crimes. A person known to the patient shalt while entering a patient's house, accompany you. The peculiar customs of the patient's household shall not be made public.

Arthashastra of Kautilya of *3rd-5th centuries BC* is considered to be further improvised code of ethics. Here, physicians were required to have taken a written permission from the ruler (king) to practice medicine, their practice being regularised from time to time and they were punished for negligence.[3]

Sushruta, the *Father of Indian Surgery* was another famous authority in Indian system of medicine, which composed *Sushruta Samhita (200-300 AD)* and defined clearly the qualities, responsibilities and duties of physicians. They were so carefully written that they are in no way inferior to the modern concepts of medical law and ethics.[4]

However, the *Medical Council of India (founded in 1933)* formulates the modern code of medical law and ethics in India. It is framed and worded on the same line as in the *Declaration of Geneva* by the *World Medical Association*, which again is a restated modern version of the oldest code of medical ethics, the *Hippocratic oath*: held in high esteem by every medical professional.[5]

CODES OF MODERN MEDICAL ETHICS

Modern code of medical ethics is basically developed from several ethical principles put forth by noble men and organisations in medical profession from the historical period of medical practice such as *Hippocratic oath, Declaration of Geneva* and *International Code of Medical* Ethics, etc.[5,8] Although these are apparently three separate codes, they are framed well dependent on each other, more so with *Hippocratic oath*. Added to this are *Declaration of Sydney, Declaration of Tokyo,* etc. which are recent in origin.[6,7,8]

Hippocratic Oath

Hippocratic oath is the oldest code of medical ethics, which is 25 centuries old now; its basic tenets remain as valid as ever. However, the historical attractiveness of archaic language and formulations, which became anachronistic, led to the restatement of the same in the *Declaration of Geneva*. New doctors at convocation ceremonies formerly pledged Hippocratic oath, though this is rare now. An English translation of this oath is given below:

- I swear by Apollo the physician, and Aesculapius and Health, and All-heal, and all the Gods and Goddesses, that according to my ability and judgement:

- I will keep this oath and this stipulation—to reckon him who taught me this art equally dear to me as my parents, to share my substance with him, and relieve his necessities if required; to look upon his offspring in the same footing as my own brothers, and to teach them this art, if they shall wish to learn it, without fee or stipulation; and that by precept, lecture and every other mode of instruction, I will impart a knowledge of the art to my own sons, and those of my teachers, and to disciples bound by a stipulation and oath according to the law of medicine, but to none others.
- I will follow the system of regimen, which, according to my ability and judgement, I consider for the benefit of my patients, and abstain, from whatever is deleterious and mischievous.
- I will give no deadly medicine to anyone if asked, or suggest any such counsel, and in like manner I will not give to a woman a pessary to produce abortion.
- With purity and holiness, I will pass my life and practice my art.
- I will not cut persons laboring under the stone, but will leave this to be done by men who are practitioners of this work.
- Into whatever houses I enter, I will go into them for the benefit of the sick, and will abstain from every voluntary act of mischief and corruption; and further, from the seduction of females or males, of freemen or slaves.
- Whatever, in connection with my professional practice, or not in connection with it, I see or hear, in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret.
- While I continue to keep this oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men, in all times. But should I trespass and violate this oath, may the reverse be my lot.

Declaration of Geneva

Following the gross transgression of medical ethics during Second World War, the *World Medical Association* restated the *Hippocratic oath* in a modern style, and called it as *Declaration of Geneva,* as given below:

- I solemnly pledge myself to concesrate my life to the service of humanity.
- I will give to my teachers the respect and gratitude that is their due.
- I will practice my profession with conscience and dignity.
- The health of my patient will be my first consideration.
- I will respect the secrets, which are confided in me.
- I will maintain by all means, in my power, the honor and the noble traditions of the medical profession.
- My colleagues will be my brothers.
- I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient.
- I will maintain the utmost respect for human life from the time of conception.
- Even under threat, I will not use my medical knowledge contrary to the laws of humanity.

Declaration of Sydney

This was the first published to guide to determine the *time of death* of comatose donor in *brain death*. The patient is not dead until the life support has been withdrawn. This was first adopted by the 22nd World Medical Assembly, Sydney, Australia, August 1968, and amended by the 35th World Medical Assembly, Venice, Italy, October 1983, and the WMA General Assembly, Pilanesberg, South Africa, October 2006. Following is the text of this declaration released on June 14, 2006.[6]

- Determination of death can be made on the basis of the irreversible cessation of all functions of the entire brain, including the brainstem, or the irreversible cessation of circulatory and respiratory functions. This determination will be based on clinical judgment according to accepted criteria supplemented, if necessary, by standard diagnostic procedures and made by a physician.
- Even without intervention, cell, organ and tissue activity in the body may continue temporarily after a determination of death. Cessation of all life at the cellular level is not a necessary criterion for determination of death.
- The use of deceased donor organs for transplantation has made it important for physicians to be able to determine when mechanically-supported patients have died.
- After death has occurred, it may be possible to maintain circulation to the organs and tissues of the body mechanically. This may be done to preserve organs and tissues for transplantation.
- Prior to postmortem transplantation, the determination that death has occurred shall be made by a physician who is in no way immediately involved in the transplantation procedure.
- Following determination of death, all treatment and resuscitation attempts may be ceased and donor organs may be recovered, provided that prevailing requirements of consent and other relevant ethical and legal requirements have been fulfilled.

Declaration of Tokyo

This was first published in 1975. Declaration of Tokyo gives the guidelines for medical doctors concerning torture and other cruel, inhuman or degrading treatment or punishment in relation to detention and imprisonment. This was adopted by the 29th World Medical Assembly, Tokyo, Japan, in October 1975. It is the privilege of the medical doctor to practice medicine in the service of humanity, to preserve and restore bodily and mental health without distinction as to persons, to comfort and to ease the suffering of his or her patients. The utmost respect for human life is to be maintained even under threat, and no use made of any medical knowledge contrary to the laws of humanity. For the purpose of this declaration, torture is defined as the deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority, to force another person to yield information, to make a confession, or for any other reason. Following is the text of this declaration released:[7]

- The doctor shall not countenance, condone or participate in the practice of torture or other forms of cruel, inhuman or degrading procedures, whatever the offence of which the victim of such procedure is suspected, accused or guilty, and whatever the victim's belief or motives, and in all situations, including armed conflict and civil strife.
- The doctor shall not provide any premises, instruments, substances or knowledge to facilitate the practice of torture or other forms of cruel, inhuman or degrading treatment or to diminish the ability of the victim to resist such treatment.
- The doctor shall not be present during any procedure during which torture or other forms of cruel, inhuman or degrading treatment are used or threatened.

- A doctor must have complete clinical independence in deciding upon the care of a person for whom he/she is medically responsible. The doctor's fundamental role is to alleviate the distress of his or her fellow men, and no motive whether personal, collective or political shall prevail against this higher purpose.
- Where a prisoner refuses nourishment and is considered by the doctor as capable of forming an unimpaired and rational judgement concerning the consequences of such voluntary refusal of nourishment, he/she shall not be fed artificially. The decision as to the capacity of the prisoner to form such a judgement should be confirmed by at least one other independent doctor. The consequences of the refusal of nourishment shall be explained by the doctor to the prisoner.
- The World Medical Association will support, and should encourage the international community, the national medical associations and fellow doctors to support the doctor and his/her family in the face of threats or reprisals resulting from a refusal to condone the use of torture or other forms of cruel, inhuman or degrading treatment.

INTERNATIONAL CODE OF MEDICAL ETHICS

This was first adopted by the *Third General Assembly of the World Medical Association* at London in October 1949.[8] International code of medical ethics is again solely based on *Declaration of Geneva* and framed as sets of *duties of doctor in general, to the sick and to each other.* The English text of this code is as follows:

Duties of Doctors in General

- A doctor must always maintain the highest standards of professional conduct.
- A doctor must practice his profession uninfluenced by motives of profit.
- Following practices are deemed unethical:
  - Any self-advertisement except such as is expressly authorised by the national code of medical ethics.
  - Collaboration in any form of medical service in which the doctor does not have professional independence.
  - Receiving any money in connection with services rendered to a patient other than a proper professional fee, even with the knowledge of the patient.
- Any act or advice, which could weaken physical or mental resistance of a human being, may be used only in his/her interest.
- A doctor is advised to use great caution in divulging discoveries or new techniques on treatment.
- A doctor should certify or testify only to that which he/she has personally verified.

Duties of Doctors to the Sick

- A doctor must always bear in mind the obligation of preserving human life.
- A doctor owes to his or her patient complete loyalty and all the resources of his/her science. Whenever an examination or treatment is beyond his/her capacity, he/she should summon another doctor who has the necessary ability.
- A doctor shall preserve absolute secrecy on all he/she knows about his/her patient because of the confidence entrusted in him.
- A doctor must give emergency care as a humanitarian duty unless he/she is assured that others are willing and would be able to give such care.

Duties of Doctors to Each Other (Medical Etiquette)

The terminology is used to indicate the conventional law of courtesy to be observed among the members of medical profession.[8] It is the rules of conduct growing the relationship in the medical profession and includes the following:

- A doctor ought to behave to his/her colleagues, as he/she would have them behave to him/her.
- A doctor must not entice patients from his/her colleagues.
- A doctor must observe the principles of "The Declaration of Geneva approved by the World Medical Association".

Declaration of Helsinki

This code of conduct for doctors emerged during 1964, introduced by the World Medical Association. It was revised in Tokyo in 1975, and governs medical research.[9] This was intended purely to embark upon any experimental scheme of treatment or whenever clinical trials for a new drug were proposed on human beings as an experiment. Such experimentations upon human beings were more common during the Second World War, involving most of the war prisoners, as experimental subjects, for a new drug or other novel methods of treatment in medicine.

CODES OF MEDICAL ETHICS IN INDIA

The Medical Law and Ethical Codes for the medical professionals in India are prescribed by the Indian Medical Council, under the section 20-A of Indian Medical Council Act of 1956 and Amendment Act No: 24 of 1964.[10,11] The text of the code is actually prescribed in two parts*: The Declaration* and *the Code Proper.*

The Declaration

This is based on *Declaration of Geneva*. At the time of registration as registered medical practitioner each applicant shall be given a copy of the declaration by the Registrar of concerned State Medical Council and shall be made to read and agree to abide by the same. The actual text of declaration of the code is provided by the Medical Council of India, is medical ethics in India, may be presented as follows:

Declaration of Code of Medical Ethics in India

- I solemnly pledge myself to consecrate my life to the service of humanity.
- Even under threat, I will not use my medical knowledge contrary, to the laws of humanity.
- I will maintain the utmost respect for human life from time of conception.
- I will not permit consideration of religion, nationality, race, party politics or social standing to intervene between my duty and my patient.
- I will practice my profession with conscience and dignities.
- The health of my patient will be my first consideration.
- I will respect the secrets, which are confided in me.
- I will give to my teachers the respect and gratitude that is their due.
- I will maintain by all means, in my power, the honour and noble traditions of the medical profession.
- I will treat my colleagues with all respects and dignity.
- I shall abide by the code of medical ethics as enunciated in the medical council regulations, 2002 (Professional Conduct Etiquette and Ethics)
- I make these promises, solemnly truly and upon my honour.

Place Signature
Date Name
Address

The Code Proper

Code of Medical Ethics

Code proper and other laws prescribed by the Indian Medical Council is dealt with in detail under separate side headings below, namely: *Indian Medical Council and State Medical Council, Registered Medical Practitioner, Professional Misconduct, Professional Secrecy, Consent, Medical negligence.*

INDIAN MEDICAL COUNCIL (IMC)

The Indian Legislative Assembly, in the year 1933, passed an Act, The Indian Medical Council Act, 1933 (Act No: XXVII of 1933). With this the Indian Medical Council was created. This Act, however, now got amended by the Indian Medical Council Act, 1956, and the Indian Medical Council (Amendment) Act, 1964, which extend to the whole of India.[1,10-12]

The medical profession in India is thus brought under the control of Indian Medical Council, by maintaining a register, entering the names of all Medical Practitioners of the country in it, before being allowed to practice the art of medicine. It lays down actual code of conduct (ethical codes) for the medical profession in India based on *Geneva Declaration*, which each qualified Medical Practitioner has to agree to and abide by, at the time of getting his/her name registered in the register of the council (through State Medical Council). The formation of the office actual, the functions proper and medical law and ethical codes of the Indian Medical Council may be discussed in detail now.

Office of Indian Medical Council

Indian Medical Council's office is situated in the capital of the country – New Delhi and comprises of a set of members of the profession from *each state* and *universities* by election, as well as by *nominations directly* by the *Government of India* in consultation with State[1,4,10-14] Governments (Fig. 4.1). Thus, it will have:

- One member from each state (appointed by government).
- One member from each university (elected).
- One member from each State Medical Register, representing non-Indian degrees (Part-II, III schedule) (elected).
- Seven members from each State Medical Register representing Indian degrees (Part-I, III schedule) (elected).
- Eight members nominated directly by Central Government.

The above body through *election* from among themselves forms an *executive committee* comprising of two officers: ***President*** and ***vice-president*** and ***20 members***. The ***Secretary*** who is a *paid executive* of the council is an *ex-officio* member. However, any number of *subcommittees* comprising of *7 to 10 members* may be formed to carry out the functioning of the council from above group.

Duration of the Office

The *tenure of the council* is for a term of not more than *5 years* or *till new office is formed*.

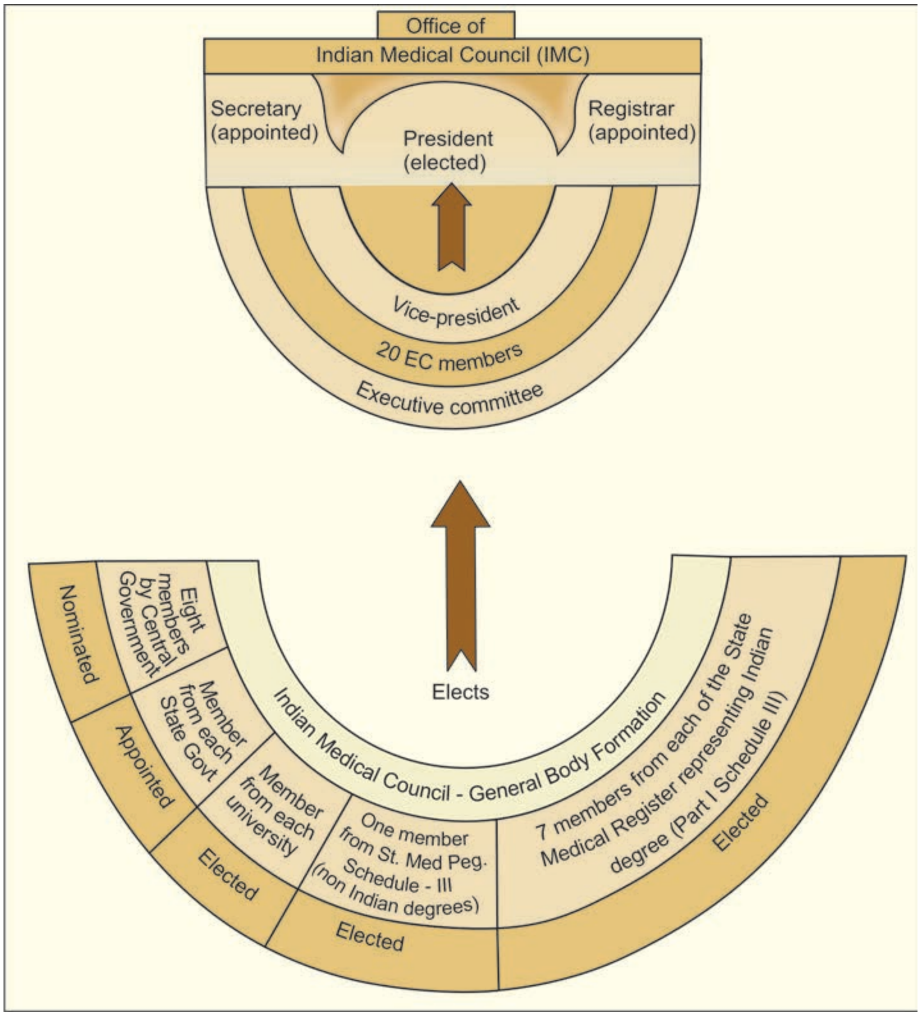

Office of
Indian Medical Council (IMC)
Secretary (appointed)
President (elected)
Registrar (appointed)
Vice-president
20 EC members
Executive committee
Elects
Eight members by Central Government
Nominated
Member from each State Govt
Appointed
Member from each university
Elected
One member from St. Med Peg. Schedule - III (non Indian degrees)
Elected
Indian Medical Council - General Body Formation
7 members from each of the State Medical Register representing Indian degree (Part I Schedule III)
Elected

Fig. 4.1: Formation of Medical Council of India

Codes of Medical Ethics Prescribed

Though the *Indian Medical Council* prescribes the ethical codes, their enforcement rests with the *State Medical Councils* and various aspects of the same will be discussed under *State Medical Council* separately.

Functions of Indian Medical Council[10-18]

Maintenance of Medical Register

An officer by name *Registrar is* appointed by the *Council*, who will maintain a register called *Indian Medical Register*. This contains the names of all medical persons who are enrolled on any of the *State Medical Register* and who possess a recognised medical qualification. A name once entered is erased normally on *death of the doctor* or *as a disciplinary action.* Names may also be *erased temporarily* and then *reentered*, depending on the time and condition of disciplinary actions taken.

Maintenance of Medical Education

Postgraduate education: It prescribes standards of postgraduate medical education for the universities and would also offer advice to universities so as to maintain uniformity all over the country.

Undergraduate education: It maintains standards at undergraduate medical education by appointing inspectors who inspect the facilities and examinations held by universities/medical institutions is in India, the purpose being to recommend recognition of the qualification to Central Government.

Indications for Inspection

MCI may arrange for an inspection of a medical college under following circumstances:

- For *every medical qualification* when it is newly introduced.
- For every *five years* routinely, to determine the standards, equipments, training, staff pattern and other facilities. If the inspection result is unsatisfactory, the inspectors concerned can make recommendation to the Medical Council of India for withdrawing recognition accorded earlier.

Recognition of Foreign Medical Degrees

Two important *sections of Indian Medical Council Act 1956, govern this*[10] and they are:

Section 12, empowers the council to recognize medical qualifications granted by institutions *outside India* and to enter in a *scheme of reciprocity with Medical Councils of Foreign Countries* in the matter of mutual recognition of medical qualifications *between the two countries*.

Section 13(4) of the *same Act*, gives authorisation to the council to recognize foreign medical qualifications which are not included in the *schedule (II),* based on *reciprocal recognition*.

Procedure: A person with such qualification should first send an application through Central Government. The full literature regarding the course of study, syllabi, duration of the course, etc. may also be required to be furnished. The council may consult directory of *medical institutions published by WHO*.

Appeal against Disciplinary Action

The *Indian Medical Council* can give advice to the *Central Government (Health Ministry)* regarding *appeals* preferred by a medical *practitioner against the decisions taken by the State Medical Council on disciplinary matters*.

Procedure of Appeal

The *medical practitioner,* against whom the *State Medical Council* has taken disciplinary action, may *appeal* to the *Central Government* in writing stating the grounds of the appeal and be accompanied by all relevant documents including a *receipt for* paying the nominal fees *(Rs. 20/-)* to Central Government. The appeal should be filed *within 30 days* from the date of decision appealed against.

The Final Decision

The final decision of the Central Government, which is given after consulting Indian Medical Council, is *final* and *binding* on the State Government and the State Medical Council.

Warning Notices

Indian Medical Council may issue ***warning notice*** to every doctor ***at the time of getting enrolled as Registered Medical Practitioner***. This comprises of information about certain unethical practices known as *infamous conduct* in a professional respect.[10,15,16]

Explanation

Warning notice hence implies that whenever a Registered Medical Practitioner commits professional misconduct, they are self-warned about the offence and the consequences of disciplinary action by the State Medical Council. Thus, warning notice is not a written letter or notice or any document by State Medical Council or Indian Medical Council through post or a messenger to the offending Registered Medical Practitioner.

The council usually appoints a *secretary for* its day-to-day work. The secretary is the executive officer of the council. He/she shall maintain the *Indian Medical Registrar* and is to update it periodically by erasing the names of practitioners who have:

- Died
- Convicted by criminal court
- Guilty of professional misconduct

He shall also *restore* the names of those doctors whose period of temporary erasure expires.

STATE MEDICAL COUNCIL (SMC)

After the Indian Medical Council Act 1933, 1956 and *the Amendment Act of 1964*, several State Governments have created *State Medical Councils* bypassing the *State Medical Acts,* to keep a *control* over the medical profession at a *State Level*. The formation of the office actual and the functions proper of State Medical Council are discussed.[5,8,10,13,15,16]

Office of State Medical Council

Each state medical council comprises of a set of members of the profession from the state by election, as well as by direct nomination by State Government. Thus, it will have:

- Members from Registered Medical Practitioners at the State *(elected).*
- Members directly nominated by the State Government.

This body elects from among themselves ***president*** and ***vice-president***. **In addition a *registrar* is appointed to be in-charge of day-to-day administration.**

Duration of the Office

The duration of the office is for a maximum period of 5 years or till new office is formed.

Functions of State Medical Council

Maintenance of Medical Register

The *State Medical Council* also maintains a *State Medical Register*. This Register will contain the *name, address, qualifications* and *date of qualification of every person who is registered under this Act* on paying a prescribed fee. On registration a *number* is allotted to each person *with a certificate*, enabling him/her to be considered as *Registered Medical Practitioner.*[5,10,11]

Display of this certificate in the place of his/her practice helps the lay public to distinguish a *qualified doctor* from the unqualified. It is the duty of the *Registrar* to *inform* Indian Medical Council, from time-to-time, about the *additions and deletions* from the *State Medical Register* regularly.

Disciplinary Control

Though the other functions mentioned above form the bulk of the Council's workload, it is the *disciplinary function* (Fig. 4.2), which is most controversial and receives maximum attention from the medical profession, public and the press.

The *State Medical Council Acts empower* the respective council to *erase* the name of any doctor from the *Register,* when *convicted* of any felony, misdemeanor, crime or offence, or judged after due enquiry by the council to have been *guilty of infamous conduct in a professional respect (Professional Misconduct).* This power of the councils is intended to *protect the public* and not to be a punitive measure against attending practitioners, though the deterrent value is admitted. As *erasure or suspension* is the most serious *professional disaster*, which can overtake the doctor, the disciplinary machinery of the Medical Councils should be fully *understood* by every practitioner.

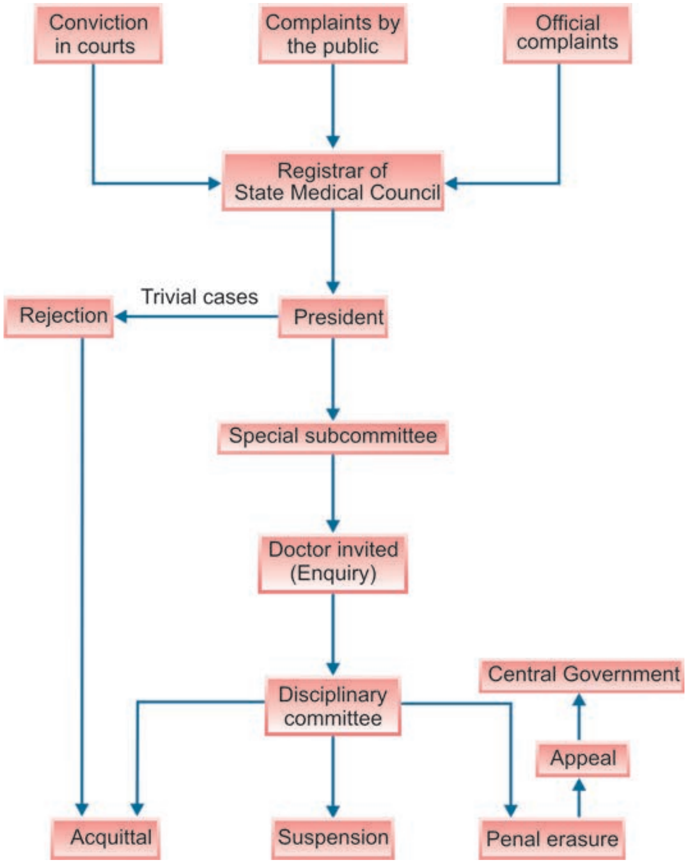


Fig. 4.2: Disciplinary functions of State Medical Council

Disciplinary Enquiry

Disciplinary enquiry may arise usually from two sources.

- Accusations of *professional misconduct* either from a member of the public or a professional colleague or a certain public officer.
- A statutory notification from the court officials where a doctor is *convicted* for any *criminal offence*.

Procedure

On *receipt* of such a notification or accusation of professional misconduct, the *Registrar* of *State Medical Council* submits it to the *President and on scrutinising* the same one of the following is opted.[6-10]

- *Rejection of the case:* Obviously false, malicious or otherwise unfounded trivialities are rejected at this stage *(acquittal if no prima facie case existing)*.
- *Submission to special committee:* The cases, which cannot be rejected, shall be referred to a *special committee* of the *President* and a *few other members*, one of whom is a *nonmedical man* of good repute. The committee will examine the facts of the case and on giving an opportunity to the accused doctor to defend, refers the case to the disciplinary committee, who decides the punishment under disciplinary action. The *disciplinary committee* is the definitive body of the *State Medical Council*, which hears cases of serious nature or offences, which have been repeated after previous warnings by subcommittees. The committee has to decide the disciplinary actions enumerated below:
  - *Acquittals:* The case may be dismissed if no *prima facie* evidence against the accused.
  - *Warning:* If it is a case of first offense, doctor may be sent back merely with warning not to repeat the same.
  - *Suspension:* It is the eraser of the name of the doctor from the register for a specific period considering the gravity of offense, to be reinstalled at the end of the period of suspension.

Penal Erasure (Professional Death Sentence)

This means erasure of the name of the doctor from the register permanently when the offense is confirmed beyond doubt allowing not to practise the profession. When a practitioner's name has been erased from the register, he/she may apply for restoration at various fixed intervals. He/she may also appeal to the Central Health Ministry against the decision of the State Medical Council.

REGISTERED MEDICAL PRACTITIONER

Definition

A Registered Medical Practitioner is a qualified doctor who has been registered in State/Indian Medical Councils.

Procedure of Getting Registered

After passing qualifying examinations (MBBS) one has to undergo a period of compulsory training of internship (House Surgeonship) for a period of minimum one year in an institution approved by the Indian Medical Council. On completion of this training he/she will have to fill in certain *application forms* and pay the *nominal fee to the State Medical Council*. The office which will then scrutinize the contents in the application and if approved, enters the name of the doctor in the register, and provides him a Registration Certificate and a code number by which he/she may be referred for all further communications.

Declaration

At the time of registration, the Registrar of State Medical Council will provide a declaration form, which contains all the medical law and ethical codes. The doctors has to sign this and get attested by Registrar/a Registered Medical Practitioner and return it to the Registrar, will have to be abide by the same, breach of which will lead him to the charges of professional misconduct, which leads to the punishment of either warning or penal erasure.

Merits of Registration as Registered Medical Practitioner

On getting registered a doctor has certain merits such as *achieving some special rights and privileges*. He/she also have to *perform certain duties towards the patients and the state*. Both of these are discussed below.[5,7,10-17]

Rights and Privileges of Registered Medical Practitioner

- He/she is legally recognised as medical man and is entitled to possess/dispense, to prescribe medicines listed in dangerous drug act.
- He/she can set up medical practice anywhere in India.
- He/she is enabled to hold official and semi-official (government) appointments at public hospitals.
- He/she is entitled to sign statutory medical certificates, such as for birth, death, mental illness, etc.
- He/she is entitled to sue for recovery of his or her fees in court of law. However, *gratuitous service* may be rendered to a deserving poor patient, as well as to all other physicians and his/her dependent family members.
- He/she is entitled to perform medicolegal autopsies.
- He or she is entitled to give evidence at any inquest or in any court of law as an expert.
- He/she is exempted from serving on a jury and at an inquest.
- He/she can choose his/her patient.
- He/she can add title, description, etc. to his or her name.

Duties of Registered Medical Practitioner

- To exercise a reasonable degree of skill and knowledge in treating a patient.
- To attend and examine a patient as long as there is a need after commencing the treatment. However, he/she can terminate his/her services, when another physician attends his patient or the patient starts using remedies other than those prescribed by him/her.
- To provide proper and suitable medicine to the patient either directly or by a prescription.
- To give proper instructions to the patient or relative of the patient regarding the use of medicine, dosage schedule, diet, etc. and warn the dangers if not used properly.
- To appoint a substitute doctor during temporary absence, with the consent of patient, especially in obstetrics and gynecology cases.
- To warn the possibilities of cross-infecting the others, in case the patient is suffering from communicable disease.
- To take proper care of children and adult patients, who are unable to take care of themselves.
- To inform all the risks involved in treatment plan. However, under the *Doctrine of therapeutic privileges* doctor need not disclose everything.[6-10]
- To handle poisons carefully and give proper treatment to a poisoning case and also cooperate with law enforcement authorities in deciding whether the case is a suicide, homicide or accident.
- To inform health authorities of a communicable disease reported to you under the *Doctrine of privileged communication* forgoing the professional secrecy principles of ethical code.[6-10]
- To exercise all duties with regard to surgery, i.e. taking consent, operating under proper anesthesia, aseptic measures, pre- and postoperative care, etc.
- To treat war prisoners, civilians of any country, etc. under the duties proposed in Geneva Convention.
- To refer a patient to specialist/consultant as and when needed on taking prior consent.
- To take an X-ray of injured part (unless trivial) for ruling out fractures.
- To maintain a perfect professional secrecy.

PROFESSIONAL MISCONDUCT

(Synonyms: Infamous Conduct)

Definition

Professional misconduct can be defined as something done by a doctor in profession, which is considered as *disgraceful* and *dishonourable* by his or her professional brethren of good repute and competence, after the enquiry by the State Medical Council.

Punishments

Under *disciplinary control* the State/Indian Medical Council decides this and *depending on the type of misconduct* punishment can be:

- Warning
- Suspension
- Penal erasure (professional death sentence).

Examples

The potential reasons for *penal erasure* or *suspension* from the *register* are limitless. However, a few examples are enumerated below. Each of these is also called as *ingredients* of *professional misconduct* and listed under **6 A's.**[8,11]

- Association with unqualified persons
- Advertising
- Adultery
- Abortion (illegal)
- Addiction
- Alcohol.

Apart from these, the councils can also consider any other form of alleged *infamous conduct*, which is not in the above list for deciding the punishment.

Association with Unqualified Persons

Following may be considered as suitable examples:

- Employing *unqualified* or *unregistered* assistants.
- *Assisting an unqualified* person for some purpose.
  - *Ghost surgery:* Here a qualified surgeon performs a surgery on a patient on behalf of an unqualified, who enters the operation theater after the patient is anesthetised and leaves prior to returning to consciousness.[8]
  - *Covering of unqualified persons:* Here a qualified doctor covers unqualified persons as to enable them to practice midwifery by issuing a certificate to them, which enables them to conduct such practice. However, this is not applicable where in proper training is offered to bonafide medical students/dispensers/surgery attendants and skilled technical assistants, under the supervision of qualified and registered medical practitioners. Conversely, the said registered medical practitioner is held responsible for all

mistakes committed by these trainees/students *(vicarious liability)*.

Advertising

Includes three modes, namely *direct advertisement, indirect advertisement* and *canvassing*:

Direct Advertising

- An unusually big name plate or signboard announcing the structure of fees collected from the patient, any concession, etc. (Fig. 4.3).
- Inserting name in a telephone directory in a special place, in bold type prints, etc.
- A prescription paper containing appointments held.
- Notification in the lay press (Fig. 4.4) of his/her addresses or telephone number/consulting hours unless he/she has:
  - Started practice afresh
  - Change of practicing place
  - Resumed practice after a temporary absence from duty, such as on returning from a short vacation or visit abroad
  - Changed the type of his practice.

Note: In each of these instances the advertisement should not appear more than twice, in two newspapers.

Indirect Advertisement

- Contributing articles to lay press (except on public health and allied matters).
- Appearance, frequently in broadcasting media such as radios, television, etc. which have the effect of advertising.
- Allowing the use of his/her name on price list of publicity materials, handbills, etc.

Canvassing

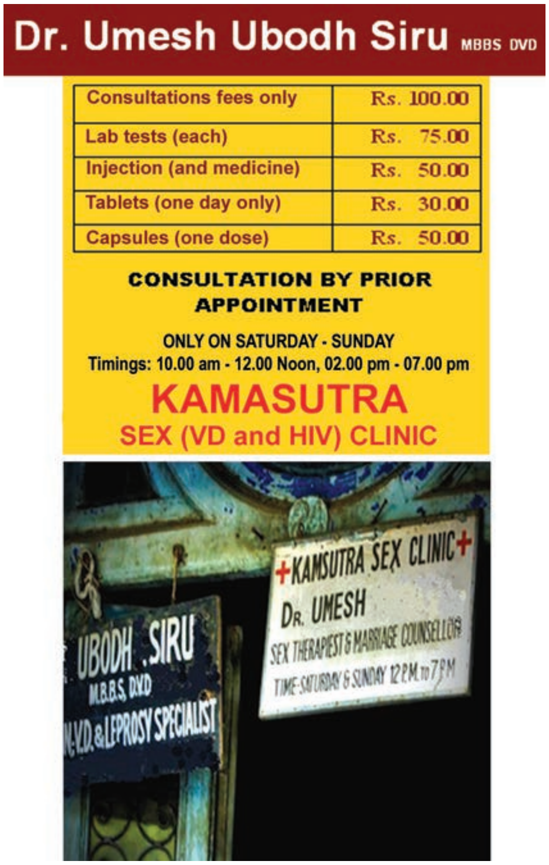
Canvassing includes use of touts or agents for procuring patients (Fig. 4.5).

Adultery

A medical man should maintain the highest professional standard and should not abuse his/her position to seduce a female patient (Fig. 4.6) or some other member of patient's family.

Abortion

Abortion includes procuring, assisting or attempting to procure a criminal abortion.



Dr. Umesh Ubodh Siru MBBS DVD

Consultations fees only	Rs. 100.00
Lab tests (each)	Rs. 75.00
Injection (and medicine)	Rs. 50.00
Tablets (one day only)	Rs. 30.00
Capsules (one dose)	Rs. 50.00

CONSULTATION BY PRIOR APPOINTMENT

ONLY ON SATURDAY - SUNDAY

Timings: 10.00 am - 12.00 Noon, 02.00 pm - 07.00 pm

KAMASUTRA

SEX (VD and HIV) CLINIC

UBODH SIRU
M.B.B.S. DVD
V.D. & LEPROSY SPECIALIST
+KAMSUTRA SEX CLINIC+
DR. UMESH
SEX THERAPIST & MARRIAGE COUNSELLOR
TIME-SATURDAY & SUNDAY 12 P.M. TO 7 P.M.

Fig. 4.3: Illustration of a doctor's name plate/signboard (Unusually large measuring 20' × 5') with consultancy rates and cost of various tests and medicine given etc displayed in the heart of the city in a prominent area.
Note: It shows no address. Another signboard hanging from the roof at the entrance of the clinic

Addiction

Supplying or selling addiction forming drugs to a person *for other than* medical grounds (Fig. 4.7).

Alcohol

Attending patients while under the effect of alcohol (Fig. 4.8).

KAKKAR SEX CLINIC

No: 123, Plaza Building, Opp. Hero Cinema, New Delhi

Do not get disappointed if you are suffering from
any sex problems

DR. MANKUR

MD (Acu), FRCH, Dip. in Sexology (London)

An Experienced Sexologist from London
Will help you

Consultation is only by appointment
Timing: 10:00 a.m. to 12:00 noon
4:00 p.m. to 8:00 p.m.

Monthly Regular Campus : Hotel Jwala, New City Market, Chennai (First Thursday)
Hotel Jalva, Super Bazar Street, Bengaluru (Last Sunday)

Fig. 4.4: Notification in the lay press (A model)

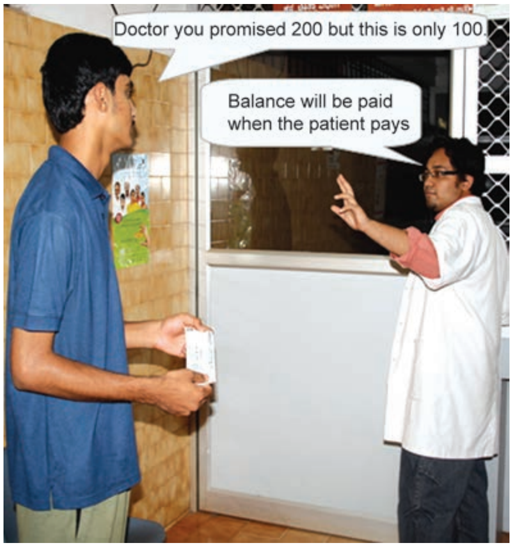

Doctor you promised 200 but this is only 100.
Balance will be paid when the patient pays

Fig. 4.5: Using touts in procuring patients

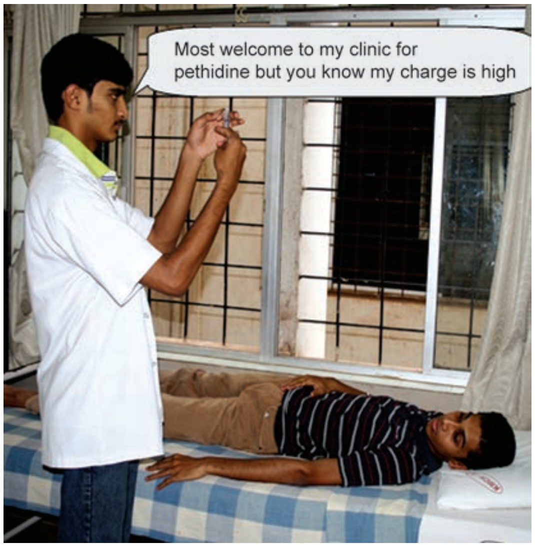

Most welcome to my clinic for pethidine but you know my charge is high

Fig. 4.7: Supplying/selling drugs of addiction

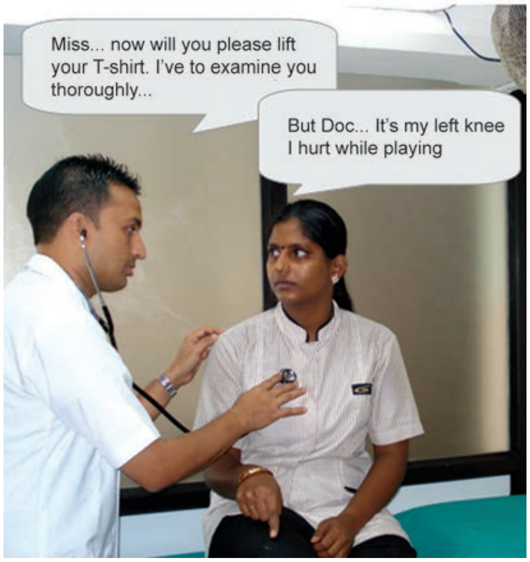

Miss... now will you please lift your T-shirt. I've to examine you thoroughly...
But Doc... It's my left knee I hurt while playing

Fig. 4.6: Doctor seducing a female patient

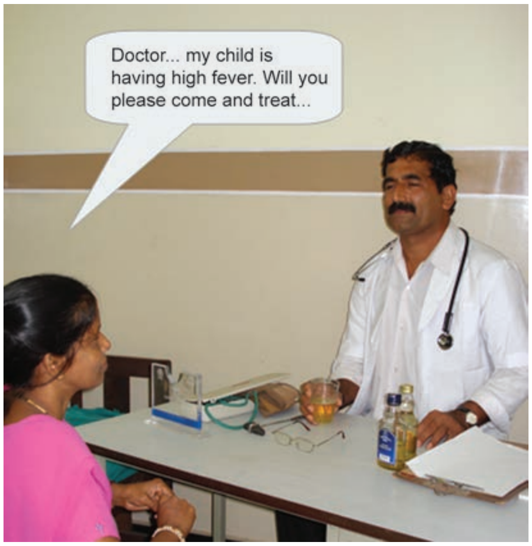

Doctor... my child is having high fever. Will you please come and treat...

Fig. 4.8: Attending patients while on alcohol

Other Examples

The Council can also consider any other form of alleged *professional misconduct*, which is not in the above list for deciding the punishment. Enumerated below are some more examples.

- *Avoiding consultations*: In situations such as a case of poisoning, when a diagnosis is doubtful, when a case had taken a serious turn, when an operation is inevitable and a case is especially one of criminal assault.
- In mutilating surgery case attending a patient who is under the care of another practitioner.
- Issuing false certificates (Fig. 4.9) in respect of birth, death and cause of death, illness, injury, vaccination, mental illness, etc.
- When the doctor is arrested/convicted by criminal court of law for offenses involving moral turpitude.
- Contravening the provisions of the drug Act.
- Selling scheduled poisons to people other than his/her own patients.
- Running an open shop for sale of medicines, dispensing prescriptions of other doctors, or for sale of medical/surgical appliances.

Fig. 4.9: Issuing a false certificate

- Writing prescriptions in a secret formula known to some pharmacy or chemist only.
- Commercialisation of a secret remedy.
- Refusing to give professional service on religious grounds.
- Gross and prolonged neglect of duties.
- Not attending a patient who is already under treatment.
- Receiving/giving commission or other benefits to professional colleagues/a manufacturer/trader/chemist, etc. Giving of a commission by one doctor to another for referring, recommending, or procuring any patients for medical, surgical or other treatment is known as *Dichotomy or Fee Splitting* or *Sharing* (Fig. 4.10).
- *Lack of concern to respond in emergencies* such as traffic accident, railway or air crash, etc.
- *Talking disparagingly about the colleagues* (Fig. 4.11) or doing anything that amounts to unfair competition.

Thank you Doc. You will get your 30% per case. This is a promise. ... Do continue...

Fig. 4.10: Dichotomy/fees splitting/sharing

Oh no... I never thought Dr. Sahani is so poor in surgery - your hand has not recovered even 5% after surgery

Fig. 4.11: Talking disparagingly of a colleague

PROFESSIONAL SECRECY

Definition

Professional secrets are the ones, which a doctor comes to know about his/her patient in his professional capacity as a physician/ doctor.[7-10,13,15-17]

Explanation

Part of the hippocratic oath affirms: 'Whatever in connection with my professional practice or not in connection with it, I see or hear in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret'.

Even if the medical graduate does not totally affirm this oath at qualification, he/she accepts it in its spirit and intention as his ideal standard of professional behaviour. Thus, the doctor should not divulge anything, which he/she has learnt in confidence from his/her patients/found on examining/noticed in the ordinary primacies of domestic life. This is not only a moral obligation but also a legal one, and practitioner is liable to damages if this is neglected.

Case Example

The classic case example is case of Kitson vs. Playfair: Dr Playfair, a well-known Gynecologist, examined Mrs. Kitson, while her husband was abroad. Mrs Kitson was receiving an allowance of £500 per year from her husband's brother. Dr Playfair regarded the pregnancy as illegitimate and communicated the circumstances to the brother-in-law, which led to his stopping payment of her allowance. Mrs Kitson brought an action for slander against Dr Playfair and the court found Dr Playfair as guilty for revealing the professional secrets and made him pay the **damages** of £12,000 to Mrs Kitson.

Medicolegal Significance

Thus, every practitioner should be very cautious to reveal statements confided in him by his/her patients outside the professional milieu. However, professional secrets may be disclosed and in fact it may become the duty of a doctor to divulge the same at times. Divulging a patient's secrets on these occasions is called ***privileged communication***. The doctor is said to get *immunity* for the communication on occasions described below:

- *Consent of patient to the disclosure of relevant information:* Where disclosure is to be anything but informal, such as to relatives, it is wise to obtain written *consent*, or at least, *witnessing oral consent*.
- *Disclosure on the order of a court of law:* When a Judge, Magistrate or Coroner orders a doctor to divulge the information about a patient that he/she has obtained out of his or her professional relationship, the doctor has to provide the details. However, he/she may continue to *refuse* at risk of *fine or imprisonment* for the *contempt of court*. Where a doctor honestly believes that disclosure would be a breach of confidence, *he/she may request* that he give the information *in writing* so that it is not made public. If these requests are not met, he/she has no other choice but to divulge or risk imprisonment.[5-10] *All matters voiced in court are absolutely privileged, and carry no risk of subsequent action for defamation of breach of confidence.*
- *Divulging in the interests of the community:* The most difficult situation of all for the doctor is where his/her ethical inclination towards silence battles with his/her conscience concerning the welfare of the community. Such instances arise when a *patient* holds some position in society and that, his/her illness may prove a *public hazard*[6-10,21] as in following:
  - A *hotel waiter* diagnosed as *typhoid carrier.*
  - A *barmaid* diagnosed to be an *open case of tuberculosis.*
  - A *bus driver* who has pronounced *hypertension* or *brittle diabetes.*

  - A *railway engine driver* who is *colourblind* or *epileptic.*
  - A person suffering from *venereal diseases* who is working as an attendant for a *public swimming-pool/bath*.
  - An individual suffering from fatal disease of *AIDS (HIV-positive cases*). The doctor here has a *legal as well as ethical responsibility* to warn the partners of AIDS patients. Since AIDS is invariably fatal, the physician-patient relationship confidentiality becomes secondary, when it involves potential harm to another individual.
- *Disclosure of criminal matters on the order of a court of law:* It may happen that a practitioner is called to treat a patient who is found to be a criminal, guilty of an offense of serious nature against law, in the due course of his/her examination. Mentioned below are few examples with instructions as how the doctor should act.[5-8]
  - If the patient is a *murderer,* doctor can *treat* his or her ailment but should *inform* proper authorities.
  - When the patient happens to be a *victim of rape,* call/inform *police* immediately.
  - *Dying declaration:* Doctor should arrange for *dying declaration/deposition* if the patient is about to *die*, by calling police/magistrate accordingly. Always try to obtain a *second opinion* about the case whenever possible.
- *Disclosure about the servant to the master:* When a master has sent a servant for medical examination, master would naturally like to know the results of examination, as to assess the servant about his/her capacity to work. Here, if the communication is supposed to be privileged it is made only to the employer, and only in cases where the employer has an interest in knowing. However, such reports should be confined to those matters only, which have an immediate bearing on the question at issue, viz. *fitness for service/ necessity for leave/extent of disability, etc.* In all such cases, it is better to *obtain the consent* of the servant to divulge the information to the *master* or *refuse to examine* him or her.[8-11]
- *Disclosure about the life insurance medical examiners reports:* A doctor here can inform anything found on examination, as for example, high blood pressure, and the proposer cannot take any action against this, as such reports are privileged. *It is taken for granted that the condition of his/her health will have to be reported, if he/she wants to be insured*. However, exceptionally the case now examined *may be an old patient of yours* for whom you have treated for certain ailments, which he or she is now completely cured of. There shall be no sign of the disease now on examination. *Under such circumstances, the doctor need not report the disease* from previous knowledge, *as it would amount to breach of professional secrecy*. In such cases, it is better to refuse examination on account of previous knowledge.[7,8]
- *Professional secrets and enquiries: Queries* may be addressed to a doctor on a person examined earlier, by insurance company/solicitor/any one else even the nearest relative of the patient. Except when the patient is a child below age of 12 years age, the doctor, *without the prior consent of the patient to do so*, answers no information.
- *Divulging information on cause of death:* The doctor, without taking prior consent of the nearest surviving relative should not answer any enquiries about the cause of death of a deceased person.

CONSENT IN MEDICAL PRACTICE

Every person has the right to have his body integrity protected against invasion by others and only rarely this can be compared (e.g. during arrest). Consent is the ethical precept that allows the patient to make invasion lawful–whether that invasion is into their body or their confidential information.[2] Every human being of adult years and sound mind has a right to determine what shall be done with his own body, and a surgeon who performs an operation without his patient's consent commits an assault for which he is liable for damages.[13-22]

Definition

Consent is defined as voluntary agreement, compliance, or permission.

Consent and its Validity

It becomes legally valid, when it is given only after understanding:

- What it is given for?
- The risks involved in consequence.
- Fulfills the rules of consent.

Consent from a patient to a doctor is a must for examining or treating especially when the *law demands it* for *any reason*.

Types of Consent

Consent in routine medical practice is basically of two types: *Implied Consent* and *Expressed Consent*.

Implied Consent

It is the most common type of consent observed in routine medical practice. Here the consent is presumed to have been given when the patient enters doctor's consulting room, summons the doctor to his/her house or holds his or her arm for an injection (Fig. 4.12).

Reasons since the patient knows that the procedure of diagnosis/treatment, etc. is simple and straightforward, with little/ negligible/no risk, and the conduct of patient implies the willingness to undergo the treatment.

Expressed Consent

Anything other than the implied consent described above is expressed consent. This may be of two types: *oral* and *written consents.*

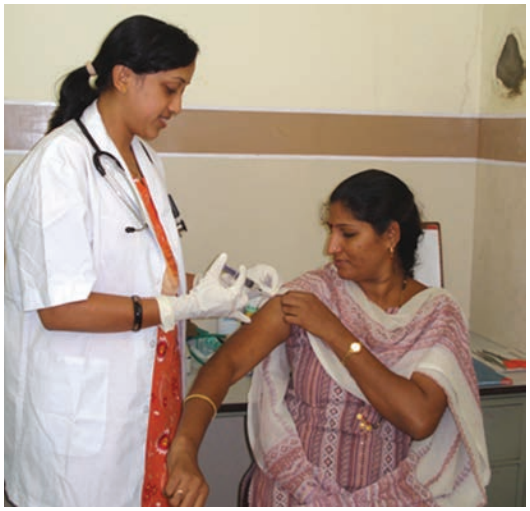


Fig. 4.12: Implied consent for injection

Oral Consent

For majority of relatively *minor examinations or therapeutic procedures*, oral expressed consent is employed, but this consent should be obtained in the presence of a *disinterested third party* (Fig. 4.13). Usually presences of a receptionist or a ward sister/nurse, etc. any one of the following suffice the purpose. However, a person closely related/associated with the patient is *not ideal* for the purpose. Oral consent, when *properly witnessed*, is of *equal validity to written consent*, but the latter has the advantage of easy proof in permanent form or document which avoids further disputes in any subsequent litigation.

Written Consent

It is obtained for all major diagnostic procedures and for surgical operations (Fig. 4.14). However, following may be noted.

- Consent should refer to only one specific procedure. As far as possible consent for more than one procedure i.e. *blanket consent* should be avoided in routine practice.
- Consent should be obtained on a special form provided for the purpose by the hospital/institution.
- A third party with a proper signature should witness consent.
- The doctor should explain the nature of therapeutic or surgical procedures in advance. However, an exception to this is *doctrine of therapeutic privilege (refer below)*.

  Even with consenting patients, where a *female patient* is to be examined by a male doctor, *ensure* the presence of a female nurse /receptionist/female relative of the patient especially when the following has to be done.
- Intimate examination of the patient.
- When anesthesia is to be given, as for minor surgical procedures.

RELEVANCE OF CONSENT IN MEDICAL PRACTICE

As already mentioned every person has right to decide what to do with his/her body.[1,2,5,7-10] A doctor may be charged with the offence of *battery/assault (Section 351, IPC)* or even *medical negligence* if he/she has failed in obtaining consent on giving all instructions about the procedure of treatment, prior to its commencement. However, one may consider following three situations to understand the relevance of consent in practice of medicine.

- *Consent in relation to certain diagnostic and therapeutic purposes.*
- *Consent and certain deviations and exceptions*.
- *Consent in relation to medicolegal purposes*.

Consent in Relation to Certain Diagnostic and Therapeutic Purposes

This is better understood by discussing under the parameters of *rules of consent, Precautions during consent,* and *types of consent.*

Rules of Consent

Legal validity of consent obtained is based on certain rules and formalities maintained in obtaining it. Consent should be always free, voluntary, informed, clear and direct. Apart from this, the person giving consent should be above the age of 12 years. He/she must be mentally sound and should not be under any fear/threat/under any false conception/intoxicated (*Section: 90, IPC*).

OK. So you consent to undergo Blood tests, Stress ECG and Echo. Sister Hilda will help you..

Fig. 4.13: *Expressed consent (Oral)* is must in all minor procedure with disinterested 3rd party

Fig. 4.14: *Expressed consent (Written)* is must in all major diagnostic/ surgical procedures

Doctrine of Informed Consent (Synonym: Rule of Full Disclosure, Written Informed Consent, Informed Consent)

It refers to written consent given by the patient after being informed of nature of illness, nature of operation or procedure to be done, its alternatives, its consequences and complications. This is essential in medical practice, when diagnosing or treating is beyond the *routine methods*, wherein risks are involved. Thus, the doctor should explain *all relevant details* to the patient.

This is called *rule of full disclosure.* However, the *doctrine of therapeutic privileges* and *emergency doctrine* are exceptions for this rule *(refer below).*

Precautions During Consent

These include formalities on how a doctor should act in obtaining consent:

- Explain the object of it.
- Inform the patient that he/she has the right to refuse.
- Explain the complete procedure of treatment.

Consent and Certain Deviations and Exceptions

Rules of consent are though rigid in their legal implications, certain deviations are usually allowed and they are:

- *Doctrine of therapeutic privileges*
- *Doctrine of emergency*
- *Doctrine of locoparentis.*

Doctrine of Therapeutic Privilege

At times it may not be possible to explain everything to the patient. Accordingly under such circumstances doctor can reveal the details to any one of the close relatives of the patient. This is called doctrine *of therapeutic privilege.*

Case Example

A patient who is to undergo the surgery of mitral valvotomy, if scared of a surgery on heart, he/she may not be told about the details of it, but any one of his/her close should relatives should be made aware of the entire procedure, its probable consequences/complications, etc. and the surgery should be performed telling him/her that it is a minor surgery (Fig. 4.15).

Doctrine of Emergency

According to this, a doctor can provide the treatment *without taking* prior *consent* from a patient who is gravely sick, critically ill, unconscious or not able to understand the suggestions or when *mentally ill (Section 92, IPC)*.

However, in such situation law presumes that *consent is deemed to be given* and *protects the doctor's interest* giving him/her *immunity* from proceedings against him/her for *damages or negligence or assault (Section, 92, IPC)*.

Section 92, IPC

Nothing is an offense by reasons of any harm which it may cause to a person for whose benefit it has been done in good faith, even without that person's consent, if circumstances are such that it is impossible for that person to sign consent, or if the person is incapable of giving consent and has no guardian or other person in lawful charge of him from whom it is possible to obtain consent in time for thing to be done with benefit.

Case Example

A victim of road traffic accident who is unconscious and with evidence of intracranial trauma clinically, requiring necessary surgery to save life. Doctor need not wait to take the consent and can proceed with surgery.

Though I've told him this is simple surgery, I must tell you its major surgery...

Fig. 4.15: Therapeutic privilege in mitral valvotomy surgery of the heart

Situations Where Consent May not be Obtained

Enumerated below are certain conditions wherein consent is not required.[13,16]

- *Medical emergencies:* Here the well-being of the patient is paramount and medical rather than legal consideration come first.
- *Notifiable diseases:* In case of AIDS/HIV positive patients, the position in India is not yet clear. However, Supreme Court has held that hospital/ doctor would be under duty to inform about it to his/her 'would be spouse' of the danger of getting infected.[25] Rather for not doing this would render doctor/ hospital punishable with *participiens criminis* under section 69 and 270 of IPC.[21] In England, the Public Health (Infectious Diseases) Regulations, 1998 extends the provisions of modifiable diseases to AIDS but not to the person who is HIV positive.
- Immigrants
- Members of Armed Forces
- Handlers of Food and Dairymen
- New admission to prison
- Incase of person where a court may order for psychiatric examination or treatment
- Under Section 53 (1) of Code of Criminal Procedure, a person can be examined at request of police, by use of force. Section 53(2) lays down that whenever a female is to be examined, it shall be made only by or under the supervision of a female doctor.[20]

Doctrine of Locoparentis

In emergency situations involving *children*, when their parents/ guardian are not available, according to this doctrine, consent can be obtained from the person accompanying.

Case Example 1

A school teacher can give consent for treating a child acutely ill while on a picnic far away from hometown. Even if parents refuse consent, no blame will be attached to surgeon for a surgery done to save the life of the child.

Case Example 2

If a student inmate in a hostel suddenly turns sick and unconscious, the *warden in-charge of the hostel* can take him/ her to a nearby doctor, who can give immediate treatment without waiting for the consent of the patient or his/her parents or relatives. The reason, which renders doctor to forgo the consent formality here, is, that, *it is a case of emergency* and unless the treatment is given immediately, *life of the patient is in danger*. This evolves the basis for concept of *Emergency Doctrine* (see Locoparentis).

Consent and Age

Rules regarding the age and eligibility to give consent in medical practice have following mandates.[2,5-9]

- Minimum age for giving valid consent for physical/medical examination is *12 years (Section 89, IPC)*. Accordingly, for medical examination of a child below the age of *12 years*, the consent is to be obtained from the parent/ guardian of the child. However, it must be realised that doctrine of 'informed consent' has only limited to 'direct' application in paediatric practice. Only patients who have appropriate decisional capacity and legal empowerment can give their 'informed consent' to medical care. In all other situations, parents or other surrogates provide "informed permission" for diagnosis and treatment of children with the 'assent' of the child whenever appropriate.[12,14] Some relevant examples for this are:

- – Venepuncture for diagnostic study in a nine-year-old child.
- – An orthopaedic device to manage scoliosis in an eleven-year-old child.

- A person who is above 18 years age can give valid consent to suffer any harm which may result from an act in good faith and which is not intended or known to cause grievous hurt or death (*Section: 88, IPC).* Thus, minimum age for giving valid consent to suffer any harm, which may result from an act in good faith, is 18 years.
- In consequence a surgeon operating on victim of an accident is doing it for the benefit of the patient and he cannot be held responsible if the surgery ends fatally, as the doctor is *acting in good faith*.

Consent in Relation to Medicolegal Purposes

- In medicolegal cases where the law requests an examination, consent must be obtained whether it is victim or accused/assailant to be examined. Without consent examination amounts to assault.[21] Examination findings when used in process of investigation can damages the party examined. If later on the party is proved innocent, damages sustained cannot be undone. This is why the right to deny consent for examination is generally given to the party. Here the consent is of informed type and must tell the person to be examined that the examination findings may go against him/her and can be used as an evidence in court. Explained below are certain medicolegal matters having relevance to consent in this regard.[8,13-18,21]

  Marriage and conjugal obligations: Regarding consent in relation to these matters, such as *sterilisation, artificial insemination, etc*. consent of *both the partners* must be obtained.
- *Pregnancy (for examining to confirm it) and delivery:* Consent (oral/written) must be obtained in advance from *concerned woman*. If this is not possible consent must be obtained from her *husband* or *relative* who is accompanying her. During examination of case/delivery, it is better to keep an uninterested third person as witness, preferably a *nurse, receptionist or a female relative* of the patient.
- *Medical termination of pregnancy (MTP ACT: 1971):* Here the consent of the *pregnant woman alone is enough* for MTP. However, she should be above the age of *18 years*.
- *Sexual intercourse:* In India *sexual intercourse* with a *consenting* woman amounts to the legal offence of *rape* if she is *below* the age of *16 years* and this is called as *statutory rape.*
- *Examination of rape victim:* In the examination of a victim of alleged rape to confirm the allegation, the doctor should obtain *prior consent* observing all *formalities*. The consent must necessarily be after telling her that the findings of clinical examination shall be *revealed in a court of law*. Consent must be obtained from her relatives if the victim is a child or minor.
- *Examining any medicolegal case:* In every medicolegal case, whether the patient is a victim or an assailant, consent must be obtained.
- *Consent and negligence charges:* In medical negligence charges against a doctor, consent is *not a valid defense*.
- *Consent in drunkenness and intoxicated cases:* Consent obtained from a person who is drunk/under the influence of alcohol is invalid. However, in such events, examination of the case may be done and findings may be revealed only after obtaining the consent at a later period when the person becomes sober.
- *Consent in examining criminal cases:* Here *no consent is necessary*, provided the requisition is from a *police officer* who is not below the rank of *Sub-Inspector of Police*, and the examination is done in the presence of a disinterested witness *(Section: 53, CrPC)*. However, a doctor as a routine, formally *may obtain consent*, and according to this section if the criminal is not willing to get examined, examination may be done with *mild degree of force.* When the criminal is a female, examination should be done only by or under the supervision of a *lady medical officer*.
- *Consent in unconscious: victims/assailants/any patient:* Examination findings can be divulged to police only after the patient regains consciousness and gives consent for disclosure.

CONSENT IN RELATION TO AUTOPSY AND ORGAN TRANSPLANTATION

Consent in Relation to Autopsy Examination

It is *improper* and *illegal* to perform autopsy without consent or authorisation. Consent differs with type of autopsy to be performed:

- Consent *is not required* if it is a *medicolegal* autopsy. Here autopsy is done only with an authorisation.
- Consent *is a must* from spouse or relatives for *clinical or pathological* autopsy. Failure to get consent here is a ground for charges of *mutilation of deceased and emotional hurt* by legal heirs. It could be ground for punitive damage *(US Law)*.

Consent in Relation to Remove and Retain Parts of the Body

Specific consent must be obtained for this purpose. However, *no civil action* has so far been reported for the removal of tissues from *the body at autopsy* even *without specific consent.*[8,9,11,12]

Perhaps it may be *justifiable* to remove certain tissues for demonstration, even without consent during *bonafide autopsies*, done without visible mutilation of the body.

Consent in Relation to Organ Transplantation

In living: A person can donate *voluntarily his/her organs, tissues, etc*. to another person for *therapeutic purposes*. However, in India the consent given for such purposes becomes legally valid only if the donor is *above* the age of *18 years*[1,7-9,13,17,22] (in United Kingdom it is *16 years*).[22,23]

Note: This is called as informed witnessed consent under Human Organ Transplantation Act, 3.[22] It states that a living person should give his/her consent in writing to donate kidney, for the transplantation purposes, in presence of two or more witnesses, at least one of them should be a near relative of the person consenting.[22]

In dead: *Consent* should be given *earlier by the person in writing* in the form of a *Written Will when* he was *alive*. However, this *consent or will* made by the deceased when he/she was alive becomes *null and void after his/her death* and to remove organs from the dead body, *consent must be obtained from legal possessors of the dead body*. No law of the land can procure organs from the dead body if the legal possessor of the deceased *refuses* to give his/her consent to donate the organs or tissues.[8,9,16,22]

MEDICAL NEGLIGENCE

(Synonyms: Professional Negligence, Malpractice, Malpraxis)

Medical negligence covers those defects in profession by a doctor where the standard medical care given to a patient is considered to be inadequate. It is also true that not all cases with negligent allegations are guilty of careless or incompetent actions in reality. However, every patient will have legal right to expect a satisfactory standard of medical care from a doctor, even though this can never mean that the doctor can guarantee a satisfactory outcome to the treatment. An effort is made to understand the concept of medical negligence.

Definition

Medical negligence is defined as want of reasonable degree of care and skill or willful negligence on the part of medical practitioner in treating a patient leading to injury or suffering or death.[2,5-9]

Explanation

Negligence, medical or otherwise is a *civil wrong* known as ***tort***, means *failing* to do something which one *is* supposed to do *(Act of omission)*, or *doing* something which one is *not* supposed to do *(Act of Commission)*.

The law assumes that a medical man will always use reasonable degree of skill, care and prudence in the treatment of his/her patient. Figure 4.16, illustrates certain circumstances in medical profession resulting in negligence/malpractice.

Extent

The extent of *care and skill* a doctor is required to possess in medical practice is highlighted below:

1. A doctor is only expected to possess such skill and knowledge as possessed by any ordinary competent man practicing at given time.
2. He/She need not possess the highest skill and knowledge, i.e. he/she should follow the standard procedures and established methods of diagnosis and treatment.
3. However, following are exceptions:
   - A specialist is expected to exercise greater skill than a general practitioner.
   - An urban doctor with access to modern gadgets and facilities and up-to-date knowledge in medicine is expected to show better skill than a rural doctor.

If the physician did *not possess or exercise* the *reasonable skill and care* expected of him or her, *he or she fails in his legal duty* causing *damage*.

Doctrine of *Res Ipsa Loquitor*

Res Ipsa Loquitor is a Latin terminology, literally means *the thing speaks for itself.*

Explanation

The doctrine of *Res Ipsa Loquitor* applied in situations where the injury could not have happened, but for the negligence of the doctor.

Usually, in cases where *negligence* is alleged, the *plaintiff* is expected to *prove* that the defendant was negligent. But, when this doctrine is applied, the *doctor* will have to *prove* that *what has happened is not due to his/her negligence*. If the element of due care of the patient as exercised could be proved, the injury or complication developed can be presumed to be a consequence of error of judgement and not of negligence.

Examples

Given below are certain examples (Fig. 4.16):

- Foreign bodies left inside body cavities after operation.
- Slipping of instruments during surgery resulting in injuries.
- Injury of the body outside the field of operation.
- Operation on wrong organ/wrong side/wrong patient.
- Too tight plaster cast resulting in gangrene of foot/toes.
- Giving medicines in overdose.
- Giving injections in wrong site/route.
- Failure to inject anti-tetanus serum (ATS) in case of injury.
- Burns from careless use of X-ray/hot water bottles, etc.
- Breaking needle while injecting, but not informing about it to the patient.
- Mismatched blood transfusion.

Classification

Negligence or malpractice is actionable and a case may be brought against a medical practitioner in a civil or criminal court. Hence, it can be classified into two types.

Civil Malpractice (Civil Negligence)

Civil malpractice is usually is of ***bifid*** nature: Either *a patient bringing charges of negligence or malpractice allegation against a doctor for compensation* towards the *physical damages* suffered by him/her, or a *doctor bringing charges against a patient who fails to pay his/her dues on the grounds of charges of malpractice on the doctor*, during the course of treatment.

Note: The ***damage***, in the sense of harm is quite different from ***damages***, which is *financial compensation* awarded to a successful litigant. Enumerated below are few examples with suitable *diagrammatic illustrations* (Figs 4.17 to 4.22) allowing easy understanding of the concepts. The *liability of doctor does not get mitigated* even when either he/she treated the patient free of charges or in a charitable hospital. The doctor will not be liable for *Error of judgement* or *Error of diagnosis*.

Examples

- Failure to exercise proper care.
- Failure to do essential diagnostic tests (Fig. 4.17).
- Promising 100 per cent cure (Fig. 4.18).
- Failure to give proper pre- and postoperative care (Fig. 4.19).
- Mishaps while giving injection.
- Giving injection in wrong site or by wrong route.
- Failure to count swabs or packs properly at the end of surgery and leaving one inside and closing (Fig. 4.20).
- Do not leave a patient unattended during labor (Fig. 4.21).
- Do not perform additional surgery unless in emergency (Fig. 4.22).

Criminal Malpractice

Usually, *patient's party* or *patient* brings the allegation of criminal malpractice against a doctor. Here the gross negligence of the doctor totally unconcerned with the life and safety of the patient is to be established against the doctor. *Prosecutions for criminal malpractice are rare.*

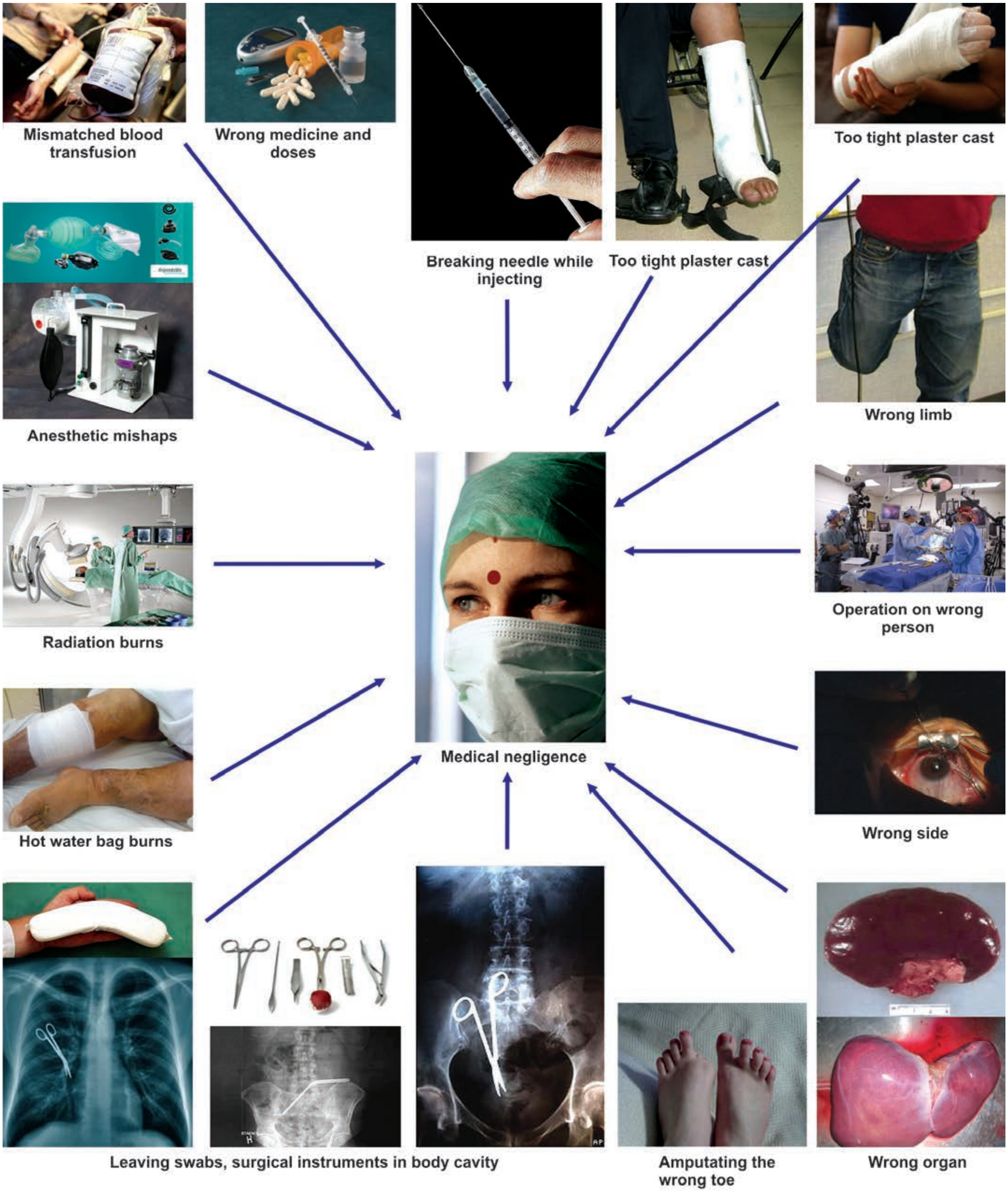

Mismatched blood transfusion
Wrong medicine and doses
Breaking needle while injecting
Too tight plaster cast
Too tight plaster cast
Wrong limb
Anesthetic mishaps
Radiation burns
Operation on wrong person
Medical negligence
Hot water bag burns
Wrong side
Leaving swabs, surgical instruments in body cavity
Amputating the wrong toe
Wrong organ

Fig. 4.16: Certain circumstances/examples in medical profession resulting in negligence/malpractice

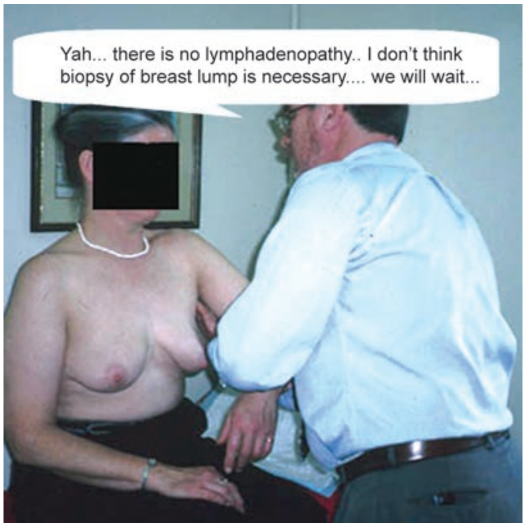

Yah... there is no lymphadenopathy.. I don't think biopsy of breast lump is necessary.... we will wait...

Fig. 4.17: Failure to do/postponement of essential diagnostic tests

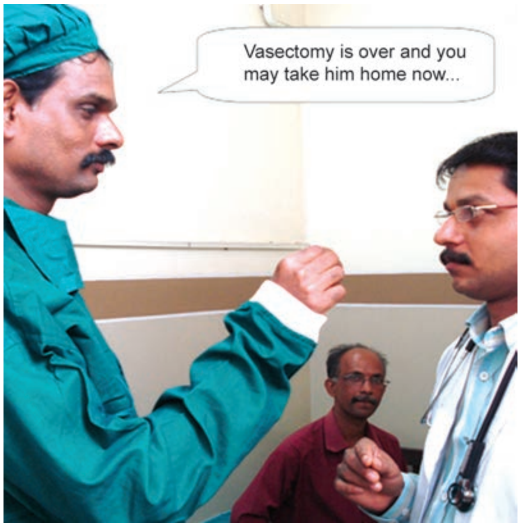

Vasectomy is over and you may take him home now...

Fig. 4.18: Failure to give proper pre- and postoperative care

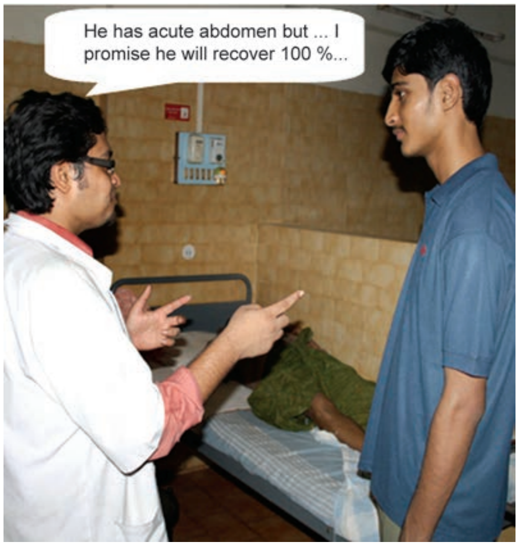

He has acute abdomen but ... I promise he will recover 100 %...

Fig. 4.19: Promising 100 per cent cure

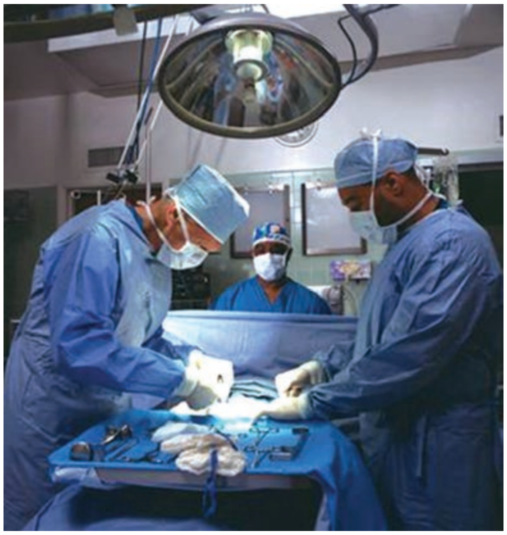


Fig. 4.20: Failure to count swabs, instruments, sponges, etc. at the end of surgery

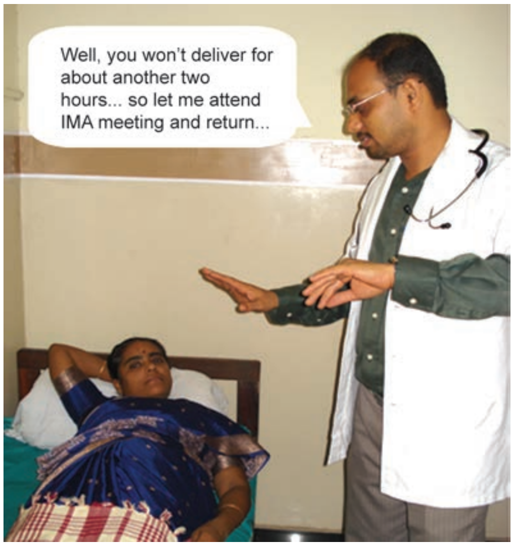

Well, you won't deliver for about another two hours... so let me attend IMA meeting and return...

Fig. 4.21: Do not leave the patient unattended during labour

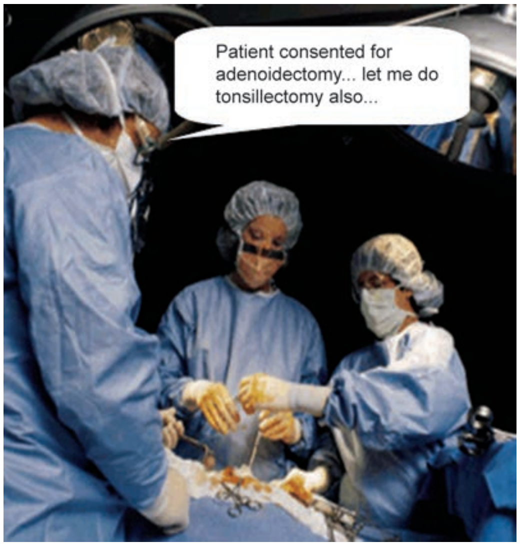

Patient consented for adenoidectomy... let me do tonsillectomy also...

Fig. 4.22: Do not perform additional surgery unless in emergency

Examples

- Cases of *gross negligence* such as removing healthy eye instead of diseased eye, amputating healthy limb instead of unhealthy one, etc.
- Failure to *reveal information to police*, in all medicolegal cases.
- *Treating under* contagious diseases.

Punishments: Case can be tried by *both civil and criminal courts* and punished by *imprisonment* or *fine* as per law.

Burden of Proof and Essential Ingredients of Medical Negligence

The burden of proof of negligence or malpractice of the doctor *lies on the plaintiff, i.e. the complainant.* The essential ingredients for the proof of medical negligence are:

- Duty
- Dereliction/breach of duty
- Direct causation
- Damage.

These *four ingredients* can be better remembered as ***4D's*** *of malpractice*.

Duty

Here the party has to prove that there was an existence of a *duty of care* by the doctor. In other words it must be proved that at the time of causation of the injury, the patient physician relationship already existed and the doctor was thus *duty-bound* to care for the patient. It is immaterial whether the patient was being treated free of charges or not.

Dereliction/Breach of Duty

Dereliction or breach of duty comprises of the element of failure on the part of the doctor to maintain proper care and skill. Injury resulting from diagnostic or therapeutic procedures performed under the effect of alcohol or other intoxicants will be interpreted as lack of care. Similarly, injury resulting from a surgical procedure that is beyond the skill and experience of the doctor will amount to lack of skill.

Direct Causation

The party must prove that failure to exercise a duty of care acted as the proximate cause and led to the injury (damage). In other words, it must be proved that breach of duty was directly responsible for damage occurred.

Damage

Here the party must prove that the damage has actually been caused as a direct consequence of breach of duty. The damage resulted should be such that it permits objective assessment. Conditions such as shock, anxiety, and tension, which do not permit objective assessment, are not likely to be accepted by courts as damage (Refer *Res Ipsa Loquitor)*.

Other Criteria

These include plethora of criteria, and are discussed individually:

1. *Foreseeability of injury:* See malpractice. If the injury could have been foreseen or predicted by a reasonably competent man, then the particular doctor is held guilty of negligence.
2. *Intervention by third person:* If there is an intervention by a third person (i.e. second doctor) between the alleged act of negligence by first doctor and injury, it is difficult to decide the negligence charge on the first doctor who treated first.
3. *Contributory negligence:* See malpractice. At times though the doctor was negligent, if patient also refused to extend his co-operation (by not following the instructions given by the doctor, or failing to give full details of the ailment suffering, etc.) during the course of treatment, charges of malpractice may be examined so as to apportion the damages between the negligent doctor and such contributing patient.
4. *Medical maloccurrence (Synonym: Inevitable Accident, Act of God):* On certain occasion, despite all proper care given by the doctor during treatment, the patient might suffer severe injuries or permanent deformities. This is known as *medical maloccurrence/inevitable accident (Act of God). If the doctor can prove this before the court, it will be an absolute defense against malpractice.*
5. *Therapeutic misadventure:* A misadventure is mischance or accident or disaster, in which an individual is injured or died due to some unintentional Act by a doctor/hospital. It is of three types:
   - Purely therapeutic (when treatment is being given).
   - Diagnostic (where diagnosis is the objective at the time).
   - Experimental (where patient has agreed to serve as subject in an experimental study).

It is a known fact that almost every therapeutic drug and every therapeutic procedure can cause death. *A doctor is not liable for injuries resulting from adverse reaction to a drug.* However, ignorance of the possibility of a reaction, *or condition* of adverse reaction to the drug prescribed to a patient, amounts to *negligence*. Doctor must warn his/her patient about the possible side effects of a drug, such as drowsiness by antihistamines, nausea with *metronidazole* on consuming alcohol, etc. Given below are *certain examples* for therapeutic misadventures.

- Hypersensitivity reaction or anaphylaxis, sometimes severe and fatal as with drugs like penicillin, aspirin, tetracycline, etc.
- Radiological procedures for diagnostic purposes may prove fatal, such as barium enema—resulting in traumatic rupture of rectum, followed by chemical peritonitis.
- Damage by radiations or radioisotopes.
- Injury due to electrical equipment.
- Death during operation.
- Death during blood transfusion.
- Prolonged therapeutic prescription of stilbesterol resulting in breast cancer.

Prevention of therapeutic misadventures

Following precautions may help in reduction or prevention of therapeutic measures:

- Prior to prescribing a drug, learn about the drug on any of its adverse effects and also treat such cases.
- Sensitivity tests should be done before injecting preparations, which are likely to produce anaphylaxis.
- Doctor should warn about permanent side effects of a drug if any, while prescribing if continued to take without further consultation of a doctor.

6. *Error of judgement and negligence:* A doctor *may not be held responsible* or *liable for the deleterious effects* of an Act proved to have *resulted* from *error of judgement*. However, if the error of judgement *was the direct result of Negligent Act* such as failure to do the essentials, it would amount to malpractice.
7. *Professional negligence and infamous conduct:* Professional negligence or malpractice usually does not amount to *infamous conduct or professional misconduct.* Infamous

conduct involves the abuse of professional status, which lacks in negligence. However, allegation of criminal negligence if proved is also considered as infamous conduct in professional respect. The differences between these two are highlighted in Table 4.1.

8. *Vicarious liability (Synonym: Liability for Act of another, principle of respondent superior):* It means an employer is held legally responsible for all. The negligent acts of his/her employees or agents appointed by him/her. It also means, let the superior be responsible. The question of vicarious liability will be raised when doctors, nurses and other staff employed by a hospital are found to be negligent in discharge of their professional duties.

If those staff members are employed under a contract of service on salary, the hospital will be liable for their negligence, irrespective of whether they are necessary or permanent, full-time or part-time, or resident or visiting. The hospital may not be held liable for the negligent acts of a superior if it has exercised due care in selecting them. However, this may not be always true.

Case Examples

- *Failure to utilize diagnostic facilities:* Here the hospital is held responsible for the negligence of doctor, who failed to diagnose fracture of cervical vertebra from which the patient died.
- *Case of surgeon's liability:* A woman suffered from abdominal burns when painted with *iodised phenol* instead of *tincture iodine* during preparation for abdominal surgery. The court held the view that the *surgeon was not vicariously liable* for the *negligence of the nurse* as surgery is considered as *teamwork* and surgeon has to *rely* on certain work done by others in the team.
- *Borrowed servant doctrine:* This refers to *vicarious liability* in relation to patient admitted to a private hospital by a private practitioner, who is not a staff of the hospital. Here, the *private practitioner will be held responsible* for all the *Negligent Acts by any of the hospital staff (Nurse, compounder, etc.)* on the patient admitted by him. The *hospital will not be held here liable*, because all the instructions for the patient admitted is *carried on* by the hospital staff *as told by* the private practitioner.
- *Vicarious liability on negligent acts by interns/ trainee doctors:* Here hospital or unit heads will be liable for all the negligent acts of interns who are appointed by the hospital on salary. However, this will not be so, if the negligence complained of comes under the preview of *doctrine of common knowledge.*

According to this the doctor may be charged for negligent act for failing in involving application of common sense in routine practice. Necessity of fluid therapy for a dehydrated patient suffering from severe gastroenteritis is a matter of common sense or common knowledge. If doctor fails to do needful here, he/she becomes negligent and to prove this plaintiff need not show that doctor did not show reasonable degree of scientific knowledge, care and skill, etc. as it is common knowledge that patient needs fluid therapy for which doctor did not take proper step.

- *Product liability:* This means liability of producer, agent or seller of medicine, an instrument, or appliance, use of which has caused the damage or harm to the patient. Doctor will not be held liable for this. The burden to prove this lies on the plaintiff.
- *Corporate negligence:* It is the failure of the persons who are responsible for providing the accommodation and other facilities necessary to carry out/follow the established standards of conduct in a hospital. It occurs when the hospital provides defective equipment or drugs, selects or retains incompetent employees or fails in some other manner to meet the accepted standards of care, which may result in injury to the patient to whom the hospital owes duty. Thus, a doctor may be charged for negligence in his/her part of duty or a nurse may be charged if she does not take care of the patient according to the directions of the doctor.
- The hospital superintendent may also be charged if he/she does not take steps to make the necessary things available for the patient, in time. Superintendent will not only be responsible for posting qualified persons for specific post, but he/she will also be obliged to see that every employee performs his/her part of duty to patient properly.
- *Vicarious liability and group medical practice.*

Each practitioner will be held liable for the negligent acts of the other, when they practice together as partners.

Procedure to initiate charges of negligence against a doctor The charge of criminal negligence against the doctor is lodged by the public prosecutor. The sufferer patient may lodge the charge of civil negligence. It may come up when doctor sues the patient for non-payment of his/her fees and patient argues that there is no question of paying the doctor's fees, as the treatment of doctor did not benefit him/her, rather caused damage to him/her.

INSTRUCTIONS ON PREVENTION OF CHARGES OF NEGLIGENCE

Box 4.1 presents certain precautions recommended to a doctor in avoiding negligence charges. In addition to these it may be

Table 4.1: Differences between professional negligence and professional misconduct

Professional negligence	*Professional misconduct*
1. It concerns *duties* of a medical man towards his patient	1. It concerns *violation of codes* and *ethics* of medical practice
2. There should be *dereliction of duty* in *treatment* causing *damage* to patient	2. There *need not be* dereliction of *duty* and damage to patient
3. Charges against erring doctor is brought before the court of law	3. Charge is brought before the state medical council
4. May be *punished* as per *Indian Penal Code* as in other criminal cases or may be liable to pay *compensation* as in other civil cases	4. Where applicable, name of the erring doctor gets erased from Medical Council Register/ served warning notice and reprimanded.
5. Appeal cases are lodged before higher courts	5. Appeals are made to the Central Government

Box 4.1: Precautions recommended to a doctor in avoiding negligence charges

- Never guarantee a cure.
- Keep professional knowledge updated.
- Apply due care and skill in treating a patient.
- Take written consent in all steps of treatment if involves risk.
- Take consent of both husband and wife in giving treatment that may result in sterility or impotency.
- Advise laboratory investigations to confirm clinical diagnosis.
- Record the patient's conditions and treatment given regularly.
- Consult professional colleagues whenever necessary.
- Always check the instruments and equipment's prior to its use.
- Always check for the intactness, instructions and date of expiry, etc. of an injection ampoule or vial prior to its use.

- Perform sensitivity test/test dose for a drug known to cause anaphylactic reactions.
- Immunize the patient whenever necessary.
- Injury due to assault and poisoning should be specially dealt with.
- Do not venture a procedure beyond the skills/field of specialisation.
- Avoid experimental treatment with a patient. But if it is necessary, proceed with written consent.

- Write prescriptions clearly, legibly and neatly with proper instructions to the patient.
- Give optimum postoperative care.
- Avoid advises or consultations on phone.
- A qualified and experienced doctor should administer anesthesia.
- Do all needful check-up and give proper premedications prior to administration of anesthesia.

- Do not leave the patient till he/she recovered from anesthetic effects.
- Recommend inquest in case of death from anesthesia on an operation table.
- Choose your assistant with due care.
- Do not stop giving treatment unless the patient desires or agrees to it.
- Do not leave an emergency case unattended.
- Do not criticize a professional colleague.
- Arrange a substitute doctor with prior information if going on leave during treatment.
- Always refer your patient to better doctor or hospital if necessary.
- Always examine a female patient in presence of another female.
- Strictly maintain provisions of medical termination of pregnancy (MTP) Act.
- Always issue medical certificates with due care.
- Do not make statement-admitting fault on your part.

relevant to remember and reproduce here the *nine R's of malpractice prevention*[17] (Box 4.2).

DEFENSES OF A DOCTOR AGAINST CHARGES OF NEGLIGENCE

When charged for negligence, a doctor may plead any one/more of following arguments as his/her defense:

- He/she had no duty to the patient.
- He/she discharged his/her duties in par with existing standards of medical practice.
- The damage caused could be due to other person who was concerned in providing the treatment.
- The damage was the result of third party intervention without his/her knowledge.
- The case is contributory negligence.
- The damage suffering is an expected outcome of the disease the patient suffered from.
- The case is of reasonable degree of error of judgement.
- The case is therapeutic or diagnostic misadventure.
- The case is medical maloccurrance.
- The case is *Res-judicata* (complaint should not be entertained by the court as it has already been tried once in court of law).
- The damage is the result of taking unavoidable risk, which was taken in good faith in the interest of patient with consent.
- Patient persistently insisted on specific line of treatment, in spite of doctors warning.
- The time limit allowed by the law for lodging such a complaint is over (complaints of negligence should be lodged within two years, counting from the alleged date of causation of damage).

Note: Free treatment of patient does not absolve a doctor from his/her responsibility towards his patient and does not give him/her immunity against negligence.

MEDICAL INDEMNITY INSURANCE

After the advent of *Consumer Protection Act*, and the Supreme Court verdict that all medical services by doctors meddling with their patients on payment come under this Act.[16,26]

The doctors are thus held liable for their negligent acts in the course of treatment.

This has threatened the medical profession with uncertain situations of who among the patients treated by a doctor can be the *potential consumer* of the doctor and charge him with negligence charges and demanding huge sum of money for damages suffered by him as compensation.

Box 4.2: Nine R's of malpractice prevention

Rapport	Maintain healthy rapport and communication with:· The patient and his family.· Fellow physicians· Office staff and nurses and other hospital personnel.
Rationale	Physician must understand what he is doing with his patients and why. The diagnostic and therapeutic rationale should be adequately documented. This can help the court in understanding the physician's thought process.
Record	Record should be carefully prepared, complete, accurate, liable germane, timely and generously informative. A good record speaks of good care.
Remarks	This refers to the gratuitous oral statements made to the patient and patient's family as well as to other members of the treatment team. In certain situations a stern warning, a forceful advice, is essential in handling uncooperative patient. But, harshness, excessive criticism, etc. will harm the good patient rapport. A doctor has to be cautious in passing remarks against another doctor who has treated the patient. Doctor should always keep the patients ailments confidential.
R_x	Never prescribe medicine unless indicated. Doctor should be aware of drug reaction, allergy, etc. for a drug.
Res ipsa loquitur	Means 'the thing speaks for itself'. The doctrine is applied in the court of law to refer to situations even an untrained layman will understand the malpractice without the testimony of expert witness. Example – closing the swab/haemostat left in the abdomen after surgery.
Respect	Many malpractice cases are triggered by concurrence of a bad medical or untoward outcome and patient's perception that the physician lacks respect/concern for him as an individual, as a person.
Risks	Risks of treatment, which varies from patient to patient, must be discussed with a patient while taking consent.

This has led the registered medical practitioner loose their confidence in profession placing himself stand in a situation where he cannot predict when and how he can be booked under the charges of COPRA and asked to pay huge compensation.

Something was thus required to fight and regenerate the confidence among the registered medical practitioners to face the consumerism policies and consumerist patients bravely.

With *Medical Indemnity Insurance (MII)* plan insurance companies can provide needful help to a doctor to pay the compensation amount to the patient when legally imposed to pay as penalty for the confirmed charges of medical negligence. This was made available in India only recently since December 1991. The term *indemnity* means, reimbursement to compensate. This insurance scheme works exactly like *Life Insurance Scheme*.

The insurance company will collect proportionate amount on a monthly/yearly/other convenient modes regularly from a doctor who opts for the plan and help to provide the insured doctor protection against the financial consequences of legal liability in his profession. If the insured is legally liable to pay damages to others, the policy help him by indemnifying him subject to the terms, conditions and limitations of contract. Indemnity is also available in respect of legal costs awarded against the insured as well as legal costs and expenses incurred by the insured with the written consent of the insurers in defence of settlement of claims.

Thus, whenever claims arise out of bodily injury or death of any patient, caused by or alleged to have been caused by omission or negligence of medical practitioner, who is covered under the medical indemnity insurance, enjoys the umbrella protection by the company paying compensation to the patient against the legal liability of the member doctor, such as defence cost, fees expenses, etc. Several insurance companies have also introduced facility of individual/joint insurance schemes to single doctor practicing alone or several or all doctors in a hospital or such other organisations recently. In India, currently two insurance companies introduced medical indemnity insurance cover and they are:

- Oriental Insurance Company
- General Insurance Corporation

The amount of premium a doctor has to pay to the company varies depending on the speciality of the doctor.

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Chapter

5

Euthanasia (Mercy Killing)

INTRODUCTION

The advances in medical science and technology have undoubtedly brought relief in several health issues to a great extent. This certainly has altered the pattern of human life and its value along with the upsurge of affirmation of human rights, autonomy and freedom of choice. Amongst these issues, one which has assumed global dimensions is the *right to a dignified death* and the related matter of *voluntary euthanasia.*[1,2] The word *euthanasia* (derived from the Greek—*eu* meaning 'good' and *thanatos* meaning 'death') raises strong emotions and has become controversial as it involves *termination of human life*, which has been unjustifiably equated with *killing.*[1] Advocates of euthanasia consider it as producing release from useless, poor quality life, economic drain on hospital, family and family finances, emotional drain, and caring for handicapped newborn or sick and aging parents.[2-5]

DEFINITION

Euthanasia or *mercy killing* or *assisted suicide* are synonymous terminologies, defined as painless killing of a person who is suffering from incurable disease, senility, or a permanent damage to brain, which cannot be repaired or cured.[6-12] Depending on *act of induction* and *willingness* of the patient, euthanasia is classified into four types.[1-3]

TYPES OF EUTHANASIA

Depending on how it is induced, euthanasia could be *active* or *passive* euthanasia; while depending on the willingness of the patient, it could be *voluntary* and *involuntary*.

Active euthanasia (Positive euthanasia): It is an *Act of Commission*. Death is *induced* by the direct/indirect action, e.g. by *giving a large dose of a drug that hastens death.*

Passive euthanasia (Negative euthanasia): It is an *Act of Omission*. Here there is no active intervention to end the life. The doctor stands by 'passively', allowing nature to take its course. No specific medicine is given or life supporting measures are provided.

Death is induced by the *discontinuation of life-sustaining measures* to prolong the life in desparate cases, e.g. stopping the *heart-lung machine facility* for a *severely defective newborn infant*, or a severe *head injury* case, etc. Thus, it is *refraining* from action that would probably delay the death and permit natural death to occur.

Voluntary euthanasia: It means *euthanasia is induced at the will of an individual by his or her request,* e.g. a patient suffering from incurable disease requesting the doctor to terminate his/ her life.

Involuntary/ non-voluntary euthanasia: It means euthanasia *induced in persons who are unable to express their wishes,* e.g. in person with irreversible coma or severely defective infant, etc.

Euthanasia is always a controversial topic. While the ethics and religion call against it, medical profession is asking for it. Accordingly as per the ethical and religious views *God has made life and God alone should take it*. However, the medical view is that *in incurable disease conditions one shall not try to kill but need not keep alive also.* Though euthanasia is *accepted* legally in certain *parts* of the *United States, Uruguay, Poland, Australia, Switzerland,* it is *not yet permitted legally in India*. Currently a lot of debate is focused on the pros and cons of validating euthanasia legally.[1,2,11,12]

Taken singularly the term euthanasia has no practical meaning, and has been qualified by *voluntary*, *involuntary nonvoluntary* and other prefixes. Medical practice today is oriented to the prime function of *sustaining life* at whatsoever cost, irrespective of the quality of life. The physician treats death as an *enemy* and feels a sense of personal defeat when he fails to avert it. This apprehension of the mind of fighting death, coupled with adherence to outmoded concepts of ethics has led to a *mental and emotional block* in most physicians towards voluntary euthanasia irrationally equated with *killing* and sense of *murder*. Perhaps the *fear of the law* is also contributing to this mental attitude.

EUTHANASIA AND ITS ETHICAL ASPECTS

Medical ethics always emphasized the need of preservation of life. Down the ages it has rejected the act of taking away of life. The intentional termination of life of a human being is contrary to the principles and policies for which medical profession stands. This is irrespective of the situation of the patient.

Hippocratic oath says: *"...neither I will administer a poison to anybody when asked for to do so, nor will I suggest such a course...".*[6-12] Thus, all ethical codes reject euthanasia.

However, it is also a fact that, medical ethics have never been static. Ethics have been undergoing changes or rather evolution frequently. Medical science and technology have produced an impact, which calls for this re-evaluation and evolution of medical ethics systems.[4,11,12]

The prime responsibility of the medical professional is to relieve human suffering. To understand the concepts of ethics, voluntary euthanasia should be viewed in this context. Duty of the physician is *to treat*, *to heal* and *offer an acceptable quality of life* to an ailing patient.[3,4]

Above all, it is the *relief* of *suffering* by all means available to him. When the end point is reached, death by *nature* or via the medium of *voluntary euthanasia* is immaterial. Consequently when euthanasia is the only *good medicine* available for the patient, what is wrong in implimenting it? A physician need not to hesitate or feel *guilty* to practice this, as *final remedy* is for the *patient* or *for the relatives*. Accepting the patient's choice—euthanasia, can certainly be considered as the most intelligent and diligent time-honoured ethics in irremediable conditions or diseases.

A physician respecting the existing practice of medical ethics, such as *patient's right to refuse any treatment* offered; or at times *using doses of pain killer drugs*, which may shorten the life—a physician is ***not*** transgressing any ethical bounds.[4,11] Added to this the patient's *voluntary* and *informed consent* to accept treatment forms all the *legal* and *ethical* basis for offering any form of treatment, be it a conservative line of treatment for prolonging life or means of painless termination of life under the banner of euthanasia.

A doctor, acting in *good conscience*, is *ethically justified* in assisting death which, relieves intense and unnecessary pain or distress caused by an *incurable* illness, greatly *outweighs* the benefit to the patient than further *prolonging* life. This applies to patients whose wishes on this matter are known to the doctor be respected and considered as more significant than any contrary opinions.

However, what is incurable today may not be so tomorrow. And therefore there is always an opportunity to reconsider and re-evaluate the indications and re-adopt the same for the ethical rationale as well.[3,4,11]

INDIAN LAW ON EUTHANASIA

As law stands today, no one has the right to do away with life, whether one's own or that of others, except under certain conditions such as war or after due process of law as punishment.[6,8,10,11,12] No one can take away the life of an innocent person. Life is inviolable. Deliberately causing death of another person constitutes a criminal act (*homicide*), as does co-operating in causing another's death. The law forbids this.

As man has the *right to live*, does he have *the right to die*? Can a physician-aid-in-dying? Legal aspects of euthanasia are difficult to understand. The law on euthanasia, though active and legalised in some of the countries globally, it has been sleeping and done nothing till now in India, as euthanasia goes on unhindered behind the closed doors.[1-3]

The law in 1994 had the first encountre on *right to die* by way of a petition filed by P Rathinam directed against the constitutional validity of *Section 309 IPC*, which deals with punishment for attempt to commit suicide. The Supreme Court ruled in favour of the petitioner, and thereby legalizes and permits suicide and rendering as unconstitutional punishment for helping/abetting of suicide.[1] In this case a consequence was drawn between *euthanasia* and *suicide*. The judgment stated that in cases of *passive euthanasia*, the *consent* of the patient (in sound mental condition) is one of the pre-requisites.[1,2]

So, if one could legally commit suicide, he could also give *consent* for being *allowed to die*. It went on to say that if suicide were held to be legal, the persons pleading for legal acceptance of *passive euthanasia* would have a winning point. This judgment came as a shot in the arm for people supporting euthanasia.

Hence, whatever progress was made, this came to a grinding halt in 1996, and the state of *confusion* returned. The same court now upheld the *constitutional* validity of IPC Sections 309 and 306 thereby legalising the same, totally contradictory to the earlier one. This presented a picture of confusion that prevails in our apex judiciary as far as euthanasia is concerned.[1,2,12]

The basis for this was *Article 21*, which states that all Indians have a *right to life* and *personal liberty*. The judgment accepted the view that in a terminally ill patient (*Permanent Vegetative State - PVS*), *mercy killing* does not extinguish life, but *accelerates* conclusion of the process of natural death that has already commenced. However, it also added that the scope of Article 21 *couldn't be widened* to include euthanasia. In the concluding remarks, assisted suicide and abetting of suicide were made punishable, due to *cogent reasons in the interest of society*.[1,2,11]

So far there has been no reported case of euthanasia *per se*, but if it does come up, the prosecution will have a definite advantage. The law as of now is still pretty *ambiguous* on the topic of euthanasia, but we can hope that some concrete steps shall be taken to resolve this burning problem.

REQUIREMENTS OF EUTHANASIA

No binding guidelines/ rules are suggested till now, as each individual case must be dealt with on its own merits. However, the requirements as laid out in a judgment of the *Nagoya High Court in Japan* may be of some aid. Accordingly, what might be accepted ethically are:[2,3]

- The patient must be suffering from unbearable pain.
- The patient's condition must be terminal with no hope of recovery.
- Euthanasia must be undertaken to relieve suffering.
- It can only be undertaken at the expressed request by the patient.
- A qualified physician must carry out the procedure.
- The method adopted must be ethically acceptable.

ROLE OF THE PHYSICIAN

Besides the above there is a very controversial area where the physician may be called upon to exercise some philosophical and moral judgement. This area is the one concerning *means* used to terminate life where in doctor has multifaceted role to play. It could be the negative means of *allowing death to occur* by withholding treatment and the positive means of *causing death to occur*.[2-5]

The question posed is whether there is a moral difference between these two means? The borderline is certainly blurred when the patient has made a *firm request for euthanasia* and the terminal event is not far away. It is difficult to see the moral difference between the two when in both means the doctor has accepted moral responsibility for actions taken.

It is misunderstanding to believe we are not terminating life when we withdraw life supports. Will it not be more humane and compassionate to bring about rapid and forceful end by positive means such as suitable doses of *narcotics*, rather than *prolong the process of dying* by withdrawing life supports?

The role of the physician in voluntary euthanasia is not only desirable but also almost imperative as he can only make several vital decisions. Thus, involvement of a *qualified physician* is a must to assess several aspects of euthanasia such as: The request by a *competent* patient, ensure if the request is voluntary, the *incurability* of the condition from which the patient is suffering, presence of *caring* at the time of death and a *swift painless death*.

VOLUNTARY EUTHANASIA— INDIAN DOCTOR'S VIEWPOINT

Extracts from a sample survey of 200 doctors carried out by the *Society for the Right to Die with Dignity* in Mumbai,[2,3] do offer some signs. Accordingly:

- Ninety per cent stated they had the topic in their mind and were *concerned*.
- Seventy-eight per cent argued that patients should *have* the right to choose in case of terminal illness.
- Seventy-four per cent believed that artificial life supports should not be extended when death is imminent; but only 65 per cent stated that they would *withdraw* life supports.
- Forty-one per cent argued that living will should be *respected*. Thirty-one per cent had reservations.
- Considerations involved ethics, morality, law and religion in that order of importance.
- More than 70 per cent were apprehensive of the *abuse* of the law if one was enacted to legalise voluntary euthanasia.

VOLUNTARY EUTHANASIA AND INDIAN SOCIETY

The issues of *right to* a dignified death and voluntary euthanasia are not the concern of the medical profession alone, and it should not be so if society has to keep a watch over abuse of the concepts. All sections of society must be vitally involved as the issues transcend any philosophical, moral, legal or theological considerations. It is an issue of *humanism* and *compassion*. Society will need to *change* its value systems in the context of the changing medical scenario, of socioeconomic environment, of increasing *cost* of medical services and their cost-effectiveness.[2,3]

Using knowledge and new power intelligently is better than just adhering to the dogmas and beliefs of the past, which has no relevance for this age of biological revolution and spectacular medical skills. To call ourselves a civilized society, one must understand death, and respect it as much as we respect life.

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Chapter 6

Consumer Protection Act and Medical Profession

INTRODUCTION

Man is a social and rational animal. Throughout his evolution he has tried to improve upon everything he has laid hands on, anything which he has thought about, everything, which he has invented or discovered. The process of thinking gives birth to self analysis as well as to the factors of coherence among various individuals. One cannot survive alone. Interaction is a must. This symbolises the dynamism of civilisation and the more free and fare it is, the more developed we become. Education, trade and commerce, statecraft, research, international policy – all are forms of human interaction. The most subtle and indispensable interaction exists between two entities, if one thinks of minutely keeping in view of commercialisation of every sphere of life, is the interaction between the seller and buyer, the giver and taker, the skilled one and the beneficiaries—the trader and consumer. Is the doctor-patient relationship a special one? Or like any other commercial transaction?

Consumer Protection Act, commonly known as COPRA or CPA came into existence in December, 1986.[1] In April, 1992, the *National Commission*, on appeal from the *Kerala State Commission* decided that the medical services be covered under COPRA.[2] Justice Bal Krishna Erade ruled that the medical services are also covered under COPRA. On November 13, 1995, Supreme Court upheld that the medical services are covered under COPRA. This means a patient (consumer) can make a complaint to a redressal forum in respect of *defective service*, if the service has been paid for.[2] Defective in the context of a doctor's service means *negligent*. Deficiency means fault, imperfection, shortcoming or the inadequacy in quality, nature and manner of performance of medical service rendered by a hospital and/or member of the medical profession. Several amendments in the Act have been passed in the Consumer Protection (Amendment) Act, 1993.[1,2]

Purpose of the Act

Thus, the *statute* has been enacted to provide for *better protection* of the interests of consumers and for that purpose to make provisions for the establishment of the *consumer councils* and *other authorities* for the settlement of consumers' disputes and for matters connected therewith.[1] The complaint is to be lodged before a duly constituted *redressal forum* for easy disposal of cases instead of following the ordinary course of law which take much time and is much expensive to a poor consumer to get justice. It is important to discuss the various provisions of the Act in the true spirit of the legislation to enable one to contemplate its applicability and equip oneself with a grasp on the subject so that as and when the need arises one may invoke the same.

Application of the Act

It has been provided in the Act that it shall apply to all *goods* and *services* unless otherwise explicitly provided by the *Central Government* by notification.[1]

Who is a Consumer?

Any person who *buys any goods against consideration or hires or avail any services for consideration, which has been paid or partly paid or promised to be paid, is a consumer*. For the matter, it also includes any user of such goods where such use is made with original buyer's approval. However, if the goods are purchased for *resale* or for *any other commercial purposes*, then the *buyer is not a consumer* and *cannot avail* the *protection* under *this Act*. Similarly, any person who hires services against consideration is also a consumer and it includes any beneficiary of such services, of course with the approval of original consumer.[1] Thus, any user of goods or beneficiary service has also a legal right and *locus standi* to initiate action under the Act. On 17 Feb, 1994, the Madras High Court while deciding bench of writ petition in *Dr. CJ Subramania vs. Kumaraswamy, I (1994) CPJ 509*, interpreted the provisions of Act *vis à vis* medical practitioners as under:[3]

- The services rendered to a patient by a medical practitioner/hospital by way of diagnosis and treatment both medical or surgical, would not come within the meaning of 'service' as defined in *Section 2(1)(0)* of COPRA, 1986.
- A patient who has undergone treatment by medical practitioner/or hospital by way of diagnosis and treatment, both medical/and surgical, cannot be considered to be 'consumer' within the meaning of *Section 2(1)(d)* of the Act.
- The medical practitioner/hospital undertaking and providing paramedical services of any category or kind cannot claim similar immunity from the provisions of the Act and they would fall, to the extent of such services rendered by them, within the definition of *service* and a person availing of such service would be a *consumer* within the meaning of Act. (This issue stands finally decided by the Supreme Court).[3]

Thus, the *Consumer's Protection Act* has created great *stir* amongst the *medical professionals* on the ground that it would be extensively damaging to the profession and the public service. Though there are many counter arguments of these feelings of the doctors in profession, it can be said that the Act if ultimately keeps medical service under its purview, then must be enforced with full precaution.[4]

Rights of a Consumer[1,5,6,8-10]

As per Consumer Protection Act, a consumer has following six rights being a consumer of a service obtained by him on payment of charges towards or an item bought by him paying its cost:

1. Right to safety
2. Right to choose
3. Right to information
4. Right to education
5. Right to be heard
6. Right to seek redressal

What is a Complaint?

Any allegation in writing made by a complainant is called a complaint.

Procedure for Lodging Complaint and Disposal of Cases[1,6]

The complaint can be lodged at any centre as mentioned below with or without engaging a lawyer and by paying nominal fees of Rs 1.25 *(Rupees One and Paisa Twenty-five only)*. There are three strata for lodging the complaints:

District Level

District Forum/District Consumer Dispute Redressal Forum, to be chaired by a district judge and two other members, one of whom should be a man of eminence and good repute and the other a lady social worker. At district level, a claim for compensation towards *damages* is fixed to a maximum of Rs 1 lakh at the starting which has been enhanced subsequently to Rs 5 lakhs.

State Level

For cases, where compensation is claimed for more than Rs 5 to 20 lakhs, the complaint should be lodged before the *State Commission/State Consumer Dispute Redressal Commission*. The state level forum is to be chaired by a *High Court Judge* and *two other members* as selected in case of district redressal forum.

National Level

When the compensation claimed is *more than 20 lakhs;* the complaint has to be lodged before the national body, i.e. *National Forum/National Consumer Redressal Commission*. A judge of the Supreme Court, selected by the Union Government (who will act as the President of the forum) with four other members including a lady member, constitutes this body.

Proper Procedure

This has been deliberately simplified. If there is a defect of goods or deficiency in service then *the Consumer* or *Any Recognized Consumer Association* or *Central or State Government* can file a complaint before the concerned consumer court in the following manner.[1,6,8-10]

- *Complaint* has to be filed with any of the three forums, within *2 (two) years* from the date of cause of action.
- Any *appeal* preferred from the order of District or the State Commission under the Act, must be filed within *30 (thirty) days* of the order.
- *Admission/no admission:* If admitted, opponent is required to file reply within *30 (thirty) days*. Under Section 23 of the Act, any person, who is aggrieved by an order made by the National Commission whether in its original or appellate jurisdiction, has a right to prefer an appeal to Supreme Court within a period of *30 (thirty) days* from the date of order. The limitation period of 30 days will not stand as a bar, if the Supreme Court is satisfied that there has been a sufficient cause for not filing it in the period.
- No court fees (even if compensation claimed is in terms of crores of rupees).
- Four copies of complaint to be filed (3 for court, one for opponent, if there are more than one opponent then so many extra copies).
- Case is expected to be decided in 90 days (maximum in 150 days). However, no time limit has been laid down by the Act, for the disposal of an appeal or revised petition.
- Only one adjournment is normally permitted.
- Presence of lawyers is not compulsory. Both parties can personally present their case. Lawyers can represent them if they so wish.
- Summons, evidence, principles of natural justice, same procedures as applicable to ordinary courts.
- So ignoring notices, summons, etc. of these courts also invite contempt, fine, imprisonment, etc.

Penalty[1,4,10]

In case of dismissal of frivolous or vexatious complaints—it shall be recorded in writing, dismiss the complaint and make an order that the complainant shall pay a penalty to the opposite party such cost, specified in the order, not exceeding Rs. 10,000.00 (Rupees ten thousand).

After the introduction of *COPRA or CPA 1986*, a large number of cases against doctors and hospitals at different levels have been filed and damages rewarded.

As per Supreme Court of India on 13 November, 1995, the medical services done on payment basis and /or also done free to those patients who can afford to pay, both come under the ambit of the Act. The judgement also states that when a person has a medical insurance policy and all his charges are borne by the insurance company, the service rendered by the medical practitioner is not a free treatment and would therefore, constitute service as defined in the Act and accountable.

Consumers in the country have also won another battle against medical malpractice where in he qualifies one medical system and practices another is considered as quack and a charlatan. Supreme Court has upheld the right of the consumer to haul-up such quacks before the Consumer Courts.

Acts and Apprehensions in Medical Profession

The medical practitioners in general found reasons to be apprehensive for fear of its *misuse* and the obvious and unavoidable *turmoil* it may create in the whole profession. The reasons of apprehension are that:[8-10]

- This act will totally disturb the *doctor-patient relationship*.
- Medical practitioners may opt *Defensive Medical Practice* – i.e. the Act will impose an undesirable tendency in doctors, particularly in general practitioners and in new graduates to be *more evasive* of their responsibilities towards their patients and refer more number of cases to consultant specialists and to advise extensive laboratory investigations which will make their position comparatively safe without the improvement of clinical mind and experience thereby making the treatment for general population to be *costlier*, even unto the extent of being beyond the reach of many.
- Another apprehension is that the doctor will develop a tendency to assure himself or herself free from the danger

Table 6.1: Comments and countercomments on COPRA

Arguments by doctors	*Counterpoints*
1. Professionals are exempt under *contract of personal service*	It is *contract for service* and not *contract of service*
2. There are civil courts, hence, no need of consumer courts	Civil courts have failed in delivery of justice at less expenses.
3. As there is no court fee, etc. any one can appeal here, increasing the litigation and wasting valuable time and energy of Dr and cases.	Consumer courts also follow principles of natural justice may sort out frivolous
4. Consumer courts are manned by nonmedical people. How can they understand and decide technical matters?	Doctors are already being tried by civil courts, for compensation claims. While they are willing to appear before a magistrate in civil court, it is surprising why they oppose appearing before a more senior but retired Judge in Judges in a Consumer Court
5. The cases are hurried through because of time limits.	Both parties can produce their own evidence, lawyer, expert. Frivolous adjournments are not allowed to prevent the delay
6. Indian Medical Council and State Medical Councils are there. Patient can complain of negligence to these bodies.	a. They can try only cases of infamous conduct. b. They can only punish doctors by withdrawing registration. c. They have no power to award compensation d. They are *all doctor bodies—so do* not inspire confidence in patients e. Virtually all professions have their own national bodies and just on this they cannot be exempted
7. A doctor would be punished thrice for the same offence by: a. Consumer courts b. Civil courts c. Medical council	No Consumer Court is additional facility It is consumer choice to go to consumer court or civil court once case is decided by consumer court, he/she cannot be punished for the same offense by a civil court (***Res judicata*)*.* Medical council has an entirely different role to decide *infamous conduct.*
8. No doctor would take *risky cases* for fear of increased litigation.	*Failure of treatment* or *error of judgment* is no offence. All principles of natural justice are followed like in civil courts.
9. Doctors would resort to *defensive practice* and all unnecessary investigations increasing the cost of health care.	This is only partly correct. What is expected is only a reasonable care and skill, not the best care and skill. So, if you maintain contemporary standards nothing to fear.
10. Doctors are practising a profession, not commerce.	There is commerce in all profession. Medicine is no exception. When medical profession is going to the extent of forming corporate bodies, how commercial angle can be denied. And with commercialism there is bound to be consumerism.
11. It would lead to a loss of trust and faith between doctor and his/her patient. A doctor starts seeing a potential litigant in each of his or her patients	With fall in the ethical standards and increasing commercialism there is already a certain degree of loss of faith. In fact due to CPA if doctors become more conscious about their knowledge, ethics and standard of practice some of the faith lost can be regained.

of paying compensation by surrendering to the different professional *indemnity society/insurance firms* which are bound to crop up to make a good business, taking advantage of this situation.

- As a consequence to what will happen as described above, the treating physician will charge from the patients more than what they charge presently.

How the Doctors in General should Deal with the Problem if the Profession is kept within the Purview of CPA (1986)?

The argument by the medical professionals is that:[5,8-10]

- The present law of the land is *not deficient* in dealing with the *erring doctors*.
- An erring doctor should be accountable for his or her Negligent Act.
- A Negligent Act of doctor may come under the purview of *Cr PC* and *IPC,* should continue to be dealt in the same way.
- The civil courts are there to take up *less severe negligent cases* which can be compensated by money.
- *Medical Council of India* and *State Medical Council* are there to deal extensively with the *misconduct and unethical practice* of a registered medical practitioner.

The counterargument by the proact lobbies is that:[9,10]

- The *time consumed* in taking decision in a *court of law* concerning payment of compensation to the aggrieved patient is *too long* and is *expensive* too.
- The common consumer of medical service should be given some relief by way of providing an alternative redressal forum for their grievances.
- The other argument of this section is that the *Medical Council (State and Central)* being bodies of the doctors themselves may have some *natural pardoning tendency* for the faulty doctors. Apart from this, many of the state medical councils are defunct or slow in their action. Thus, generally speaking, justice remains far from the reach of the patients who suffer from the Negligent Acts of the doctor.
- While presenting their case on different platforms, doctors have expressed various apprehensions and reservations about *COPRA, 1986*. However, majority of those arguments look misplaced and only a few sound genuine. Table 6.1 presents the comments and counter comments of the Act.[8-10]

CONCLUSION

Considering the arguments and counterarguments above (Table 6.1) in this respect, following conclusions can be drawn.

- Medical councils can be *reactivated* and can be made *more effective* by allowing them to decide *compensation* to the aggrieved patient, by the *erring doctors*.
- The fact is that the Act of *Negligence* and *Misconduct* of a *doctor* can be better appreciated and assessed by members of *medical profession alone*. However, inclusion of *members* from other concerns of the society in the *medical council* is desirable and no case can be just skipped off without application of mind and attention.
- *Doctors* like all other members of the society *should be accountable for their works*, particularly because they deal with life *and death issues* of other members of the society.
- All the while, as argued by the *doctors* there is nothing wrong in *inclusion* of few medical men *in the consumer redressal forum* as members, particularly when a case of *therapeutic controversy* is to be taken up.
- If the government and the society think that the present legal system is *not sufficient to deal* with the *negligent doctors*, they should make provision of *infallible judgements* from medical point of view.
- In *whichever forum* the Negligent Act of a *doctor is tried*, provision must be made for *inclusion of members of medical profession* in it.
- Effort must be also made to provide *compensation* to the *doctor* when he/she is *proved to be non-negligent*, by the complaining *party*.
- The cases of alleged negligence should *not be made public* through media and press so as to hamper the *professional life of the doctor*, as the case may ultimately be proved to be a *mistake of fact*s without any indication of any wrong by him/her.

Let the profession utilize CPA as a challenge to increase the *standard of medical care* in general and make the government realize its responsibilities like improving the facilities in the government hospitals, reduce patient congestion, etc.

Remember, the old saying: *Prevention is better than cure.* Let us do some *introspection* and *reaffirm* that we shall stand by the *hippocratic oath* and the *Code of Medical Ethics*. That would go a long way in winning over the patient's confidence and faith, in preventing litigation and also salvaging our lost nobility and reputation of *Vaidyo Narayano Hari* to some extent.

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Chapter 7

Human Organ Transplantation: Legal and Ethical Aspects

INTRODUCTION

The Transplantation of Human Organs Bill, 1994, provides for the regulation of removal, storage and transplantation of human organs for therapeutic purposes, and for prevention of commercial dealing of human organs and for matters connected there with or incidental thereto.[1,2] It has been called as *Transplantation of Human Organ Act, 1994,* and came into effect from February 04, 1995, with Government of India Gazette notification. With the Act coming force, brain death has acquired legal status in India. The Act also caused the Ear Drums and Ear Bones Act, 1982, and the Eyes Act, 1982 to be repealed.[1]

Though human tissues or organs, anatomically speaking are of several types, medicolegally they are of only two types:[3-6]

1. Regenerative
2. Nongenerative.

Regenerative Tissues or Organs

Regenerative tissues or organs are human tissues or organs, which, even if removed from their respective places in the anatomy, have the capacity to rejuvenate or replicate within the body.

Examples

Blood, semen, bone marrow, skin, etc. which are regenerative in character. So, in effect if a man or woman donates blood or semen (in the case of man) or other regenerative tissues or organs or body substances, he/she does not stand to lose anything as these tissues or bodily substances have in them an inherent quality to regenerate within the body.

Nongenerative Tissues or Organs

On the other hand, nongenerative tissues or organs such as kidney, heart, lung, liver, etc. if removed from human body will not be regenerated. As a result, in donation of such tissues or organs, the donor undoubtedly, loses a vital organ in the body, which in some cases may result in permanent partial disablement or may even lead to the donor's death.

Concept of Living and Cadaver Donors

At this juncture, an explanation as to cadaver donations seems very necessary. This is because doctors and surgeons are now turning to donors who are *brain dead*. Medical research and technological development have enabled medical practitioners to put patients on an artificial respiratory system or a ventilator, as it is popularly called.

By this process, the person's organs such as the heart and lungs continue to work, while the brain of the person is irreversibly dead. Thus, the concept derives also the terminology "beating heart donor's" for the reasons that the organs can be removed from such brain dead patients who are declared dead but kept ventilated and circulated with oxygenated blood by ventilator and such other artificial means.

THE TRANSPLANTATION OF HUMAN ORGANS ACT, 1994[1-5,7,9,10]

The proliferation of human organ trade for the purposes of transplantation during 1970s and 1980s has influenced World Health Organisation (WHO) to develop a set of guiding principles on human organ transplantation. Accordingly, it has been resolved that, organs and tissues may be removed from the bodies of deceased and living persons, for the purpose of transplantation only in accordance with the following guiding principles:

- Organs may be removed from the bodies of deceased persons for the purpose of transplantation, if:
  - Consent required by law is obtained, and
  - There is no reason to believe that the deceased person objected to such removal in the absence of formal consent given during the person's lifetime. Physicians determining that the death of a potential donor has occurred, should not be directly involved in organ removal from the donor and subsequent transplantation procedures, or be responsible for the care of potential recipients of such organs.
- Organs for transplantation should be removed preferably from the bodies of deceased persons. However, adult living persons may donate organs; but in general, such donors should be genetically related to the recipients. Exceptions are regenerative tissues.
- An organ may be removed from the body of an adult living donor for the purpose of transplantation if the donor gives free consent. The donor should be free of any undue influence and pressure and sufficiently informed to be able to understand and weigh the risks, benefits and consequences of consent.
- No organ should be removed from the body of a living minor for the purpose of transplantation. Under national law exceptions may be made in the case of regenerative tissues.
- The human body and its parts cannot be the subject of commercial transactions. Accordingly, giving or receiving payment (including any other compensation or reward) for organs should be prohibited.
- Advertising the needs for or availability of organs, with a view to offering or seeking payment, should be prohibited.

- Physicians and other health professionals are prohibited from engaging in organ transplantation procedures if they have reason to believe that the organs concerned have been the subjects of commercial transactions.
- Any person or facility involved in organ transplantation procedures is prohibited from receiving any payment that exceeds a justifiable fee for the services rendered. In the light of the principles of distributive justice and equity, donated organs should be made available to patients on the basis of medical need and not on the basis of financial or other considerations.
- In tune with the international understanding, the Indian Parliament has enacted *The Transplantation of Human Organs Act, 1994*.

Restrictions of Removal and Transplantation of Human Organs[1,2,5,6]

Transplantation means the grafting of a human organ from any living or deceased person to some other living person for therapeutic purposes. Any part of human body consisting of a structured arrangement of tissues, which if wholly removed, cannot be replicated by the body, is known as human organ.

Fundamentally speaking, this Act seeks to prohibit transplantation of human organs, except in accordance with the procedure envisaged by the Act. In this regard, *Section 9(1)* mandates that, no human organ removed from the body of a donor before his/her death shall be transplanted into a recipient unless the donor is a near relative of the recipient.

A near relative means spouse, son, daughter, father, mother, brother or sister. Where any donor authorises the removal of any of his/her organs after his/her death, the human organ may be removed and transplanted into the body of any recipient who may be in need of such organ.

If a donor authorises the removal of any of his/her organs before his/her death and then transplantation into the body of a recipient, who is not a near relative, by reason of his/her affection or attachment towards the recipient or for any other special reasons, such human organ shall not be removed and transplanted without the prior approval of the authorisation committee.

The Act enjoins upon the State Government to constitute one or more authorisation committees for this purpose. On an application jointly made by the donor and the recipient, the committee, after conducting a detailed enquiry, subject to compliance of various conditions, may grant approval for the removal and transplantation's, by giving valid reasons.

Authority for the Removal of Human Organs[1,5,9,10]

A donor may authorize the removal, before his/her death, of any human organ of his/her body for therapeutic purposes. If, at any time before his/her death a donor had, in writing and in the presence of two or more witnesses (at least one shall be a near relative), unequivocally authorized for the removal of his/her organs, for therapeutic purposes, the person lawfully in possession of the dead body of the donor, unless he/she has reason to believe that the donor had revoked the authority, may authorize such removal.

In the absence of any such authority, the person who has lawful possession of the dead body may authorise removal, provided if it does not result in any kind of objections from kith and kin, if any, of the deceased.

Where a human organ is to be removed from the body of a person who has suffered brainstem death, the removal shall be undertaken only when a board of medical experts consisting of the *following certifies* such death:

- The registered medical practitioner incharge of the hospital in which brainstem death has occurred.
- An independent registered medical practitioner, who is a specialist, to be nominated by the registered medical practitioner in *clause (i),* from a panel of names approved by the appropriate authority.
- A neurologist or a neurosurgeon to be nominated by the registered medical practitioner specified in *clause (i),* from a panel of names approved by the appropriate authority.
- The registered medical practitioner treating the person whose brainstem death has occurred.

In the case of a person who is less than 18 years of age and whose brainstem death takes place, the parents of such person may authorise the removal of any organ. However, it is to be noted that, if any inquest is to be held on the deceased person, no authorisation can be granted in such cases.

In the case of unclaimed bodies *(those who have not been claimed by near relatives within 58 hours from the time of death, either from hospital or prison),* the authority to remove any organ is vested in the hands of a person of such hospital or prison, who is empowered by the management in this regard.

In the case of a dead body that has been sent for postmortem examination, the person competent *(as notified by the State Government)* may authorise, the removal of organs for therapeutic purposes, subject to formalities as notified by the *State Government*.

Section 7 mandates that, after the removal of any human organ from the body of a person, the registered medical practitioner shall take adequate steps for the preservation of the human organ.

Regulation of Hospitals[1-8]

From the date of the commencement of the Act:

- No hospital, unless registered under this Act, shall conduct, or associate with, or help in, the removal, storage or transplantation of any human organ.
- No medical practitioner or any other person shall conduct, or cause to be conducted, or aid in conducting by himself/herself or through any other person, any activity relating to the removal, storage, or transplantation of any human organ at a place other than a place registered under this Act.
- No place including a registered hospital shall be used by any person for the removal, storage or transplantation of any human organ, except, for therapeutic purposes.

Offences and Penalties[1]

According to *Section 18*, any person who renders his/her services to or at any hospital, for purposes of transplantation, or conducts, or associates with, or helps in any manner in the removal of any human organ without authority, shall be punishable with *imprisonment for a term which may extend to five years and with fine which may extend to ten thousand rupees*.

If such person is a registered medical practitioner, his/her name shall be reported by the appropriate authority to the respective State Medical Council for taking necessary action including the *removal of his/her name from the register* of the council for a period of *two years for the first offense and* *permanently for the subsequent offense.* In addition to this, *Section 19* deals with, whomsoever:

- Makes or receives any payment for the supply of, or for an offer to supply, any human organ.
- Seeks to find a person willing to supply for payment, any human organ.
- Offers to supply any human organ for payment.
- Initiates or negotiates any arrangement in involving making of any payment for the supply.
- Takes part in the management or control of a dead body of person, whether a society, firm or company whose activities include any of those mentioned in the clause.
- Publishes or distributes or causes to be published or distributed any advertisements in this regard, shall be punishable with imprisonment for a term which shall not be less than two years but which may extend to seven years and shall also be liable to fine which shall not be less than ten thousand rupees, but may extend to twenty thousand rupees.

However, in the light of special and adequate reasons, if any, the sentencing judge may award less than two years imprisonment. So far as the cognizance of the alleged offense is concerned, no court shall take cognizance except on a complaint made by:

- The appropriate authority or any authorized officer or
- Any person who has given notice of not less than 60 days to the appropriate authority.
- The legislative effort in combating this kind of exploitative commercial trade, seemingly, appears to be quite comprehensive.

However, whether the implementation of this would result in distribution or availability of human organs on the basis of medical need or is not difficult to assess as of now. In addition to passing a skeleton piece of legislation, primarily, it is expected of all the states to pass resolutions and the appropriate government must notify the relevant rules, without any further delay, as mandated by the Act of 1994.

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Chapter

8

Ethical and Legal Aspects of AIDS

INTRODUCTION

The unexpected appearance of opportunistic infections and an aggressive form of Kaposi's sarcoma amongst apparently healthy homosexual men and intravenous drug abusers, heralded in 1981, the recognition of a new disease known as the Acquired Immunodeficiency Syndrome (AIDS).[1] Immunodeficiency is the hallmark of AIDS.

Ever since 1981, as the incidence and awareness on *AIDS* increased among the public, the ethical, legal and regulatory mechanisms are becoming more significant. Efforts to protect individual rights while safeguarding the public from a fatal communicable virus are presenting many unprecedented legal questions on public health, education, employment, insurance, medical law, family law, civil rights, etc.[2-7] An attempt is made here to discuss briefly on ethical and legal aspects of AIDS in the context of developed countries like UK and USA and a developing country like India.

ENGLISH LAW[2,3]

Addressing the question of conflicting public interests, it was held that courts would restrain breach of confidence unless there is a just cause or excuse for breaching confidence.[7]

However, the duty to disclose information to persons in close proximity of the HIV-infected persons and other patients suffering from contagious diseases has been considered as qualified defense in both USA and UK. The British Medical Council has passed a resolution in 1991 that no physician shall undertake mandatory testing of blood unless expressed consent (refer chapter Consent) has been given by the patient. If the physician does so, he/she is liable for disciplinary proceedings where he/she should justify his action. This is very relevant especially in the context of unemployment problem faced in England today. Almost all corporations in England insist on HIV test certificate to be given by in-house doctors. Therefore, the British Medical Council (BMC) sought to reinforce the view held earlier by medical experts in UK that there is little justification in law for disclosing to an employer that an employee has AIDS or is HIV positive.

US POSITION[2-4]

Medical confidentiality has been statutorily recognised in several US federal laws. They offer privacy protections to public health records containing HIV-related information. Also, a proposal, which would bring all medical records containing HIV-related data under protection, has received significant attention in Congress.

Health professionals and public health officials have endorsed this. This proposal was incorporated into the public health and welfare code, which talks of AIDS, where strict confidentiality has to be maintained with respect to all information connected with HIV-related diseases and patients.

The Florida Supreme Court has recognised the damaging effect of such disclosure of confidential information on public health policy by deterrence. The issue was one of blood donation. The court held that as the populations of HIV-infected individuals grow large, the importance of confidentiality both in and out of the courtroom increases.

Much emphasis is not being given with respect to US decisions because of the abundance of case laws in both quarters. To be specifically noted are a few decisions made by the Supreme Court, USA that upheld the views of *recognition of right of informational privacy*.

Thus, a subpoena request for identity of a school child with AIDS was rejected in part because of reliance on state and city confidential laws. Therefore, even in the United States, the health officials have recognised that protection of sensitive information gathered during tracing would encourage participation and cooperation by eliminating the fear of embarrassing and damaging disclosures. The ultimate object of such contact tracing is testing and counseling to provide public health benefits. Furthermore, failure to provide adequate safeguard to protect such information, they are aware, will certainly discourage voluntaries.

INDIAN POSITION[2,3,5]

The existing AIDS law in India consists of *State Amendments* and a proposed *Central Bill*. In August 1989 a bill was proposed in the Parliament called the AIDS (prevention) Bill.

- *Section 4* of the said bill mandates every registered physician to report each case of HIV positive patient to the health authorities, but does not provide a confidentiality clause.
- *Section 7*, however, requires health authorities to undertake counseling, health education and specialized treatment. But this is of no avail if confidentiality is not guaranteed.
- *Section 5*, talks of precautionary measures to curb the spread of disease.
- *Section 5(c)*, suggests removing the patient to a hospital or such other place "for special care", all at the direction of health authority. Therefore, isolation is a proposed alternative. This piece of legislation was sought to be repealed after vehement protest from Human Rights Groups and the WHO.

Most of the local *municipal laws, public health laws as well as the Epidemic Disease Act, 1897, Section 2,* says that the State Government is empowered if it is satisfied that the State or any part thereof is visited or threatened with an outbreak of any *dangerous epidemic disease*, to take measures which it thinks fit and prescribe temporary regulations which would have to be followed by public or any class of persons, necessary to prevent such outbreak and spread of such disease. Under this Central Legislation, Goa, Maharashtra, Orissa and Karnataka have sought to incorporate provisions relating to AIDS within the scope of local acts.

Examples

- In Maharashtra, *Section 421* of Mumbai Municipal Corporation Act requires every doctor to notify the existence of any dangerous disease.
- In *Goa Public Health Act*, according to Section 53(1)(iv) no person including a foreigner can refuse blood collection if the Health Officer has reason to believe that such person is suffering from AIDS or other infectious diseases.
- In Karnataka, certain hospitals have isolation wards pertaining to HIV-tested positive people. The local Public Health Act in Karnataka talks of mandatory testing of all women who are admitted into remand homes in the State.

ISSUES RAISED

With AIDS there are three basic issues raised[2-4] and they are as follows:[2-4]

- AIDS being a disease with no cure/no remedy, what is the status quo of confidentiality with it?
- AIDS if considered as contagious and dangerous then why is it not a notifiable condition?
- AIDS if not considered as statutorily notifiable, is it constitutionally valid to deal it under Epidemic Diseases Act?

To begin with, one must identify whether AIDS is a contagious condition or not. It is *not* appropriate to use the term *contagious*. AIDS is a disease *communicable* from one person to another in specific context only, i.e. by *sexual contact* or *through blood*. Hence, it is *not transmitted* through air, water, belongings or other usual agents. *Therefore, HIV is a communicable disease.*

The logical deduction to this would be that *AIDS is not an epidemic disease*, *neither it is contagious*, *nor it can be cured or restrained from further spread by isolation and such measures*. Therefore, it is submitted here that all legislative efforts in this regard are constitutionally invalid.

At this juncture, it would be pertinent to make mention of *two policy responses* prevalent in the world today that tackle the problem of AIDS:[2,3,4]

- Isolationist response
- Integrationist response.

Isolationist Response

Isolationist response is adopted by Cuba and Romania and includes two specific features:

- Mandatory testing
- *Isolation of infected victims.*

Though both of these policies adopted are encouraging, the inevitable defects observed with these are:

- The approach of mandatory testing is neither feasible nor economically viable in a country with a population of more than 900 million.
- This means that such mandatory testing is going to be confined to the high-risk groups. The HIV is not related to sexual preferences but to unsafe sexual practice, and such testing on this group is going to reinforce strong prejudice against them.
- This will lead to entire high-risk group going underground. There will be failure in disclosing the ailment for the fear of discrimination. This will defeat the object of approach.

Integrationist Response

Integrationist response is proposed by WHO, and adopted by USA and UK and includes three specific features:[2]

1. No compulsory testing
2. Protecting through confidentiality
3. Ensuring nondiscrimination against them.

These policies are considered to be more encouraging for the merits prevalent in them, which are:

- Right to information about HIV tests being conducted and its implication.
- Right to refuse to undergo HIV test.
- Confidentiality about HIV status of the person who has undergone the test.
- Right against discrimination in employment.

It is better to examine another issue, viz. private interest vs. society interest. How is such balancing possible and in whose favour is it? Nonprotection of confidential information relating to HIV-infected persons involves a public interest, the interest of society to be warned of him/her, his/her antibody status so that no other person is encouraged to having sexual relationship with him/her. Here the privacy of the individual suffers.

On the other hand, protecting confidential information serves two interests:

1. The interest of person (*his/her privacy)* is protected.
2. The public interest is also served by not disclosing such information. If it is disclosed, people will stop taking such tests, for the fear of social stigma, loss of employment, etc.

Nonprotection serves a public interest, but *fails* to protect an individual interest. On the other hand, *protection* serves *two interests* public and private. So, *protection of confidential information* will necessarily serve a better interest at large. It is relevant at this juncture to explain that confidentiality clause is therefore essential given the circumstances of an HIV-positive person. Earlier attempts such as the 1989 Bill was withdrawn due to protest from human rights group and WHO, who vehemently count down the practice of isolation. India has not on policy resolved to undertake the integrationist response, but it has not sought to back it up by way of legislation till date.

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Chapter

9

Medical Records

INTRODUCTION

Medical records comprise of various documentary reference of the care and treatment particulars provided to the patient.[1,2] It is the only valid data available regarding the patient treated by the health care professional, either as a general practitioner or as a hospital-based medical officer, irrespective of whether private or government origin.

It is mandatory that all health care professional should maintain the medical records of the patient examined/and treated by them on outpatient basis or as an inpatient case admitted in the hospital. Such records not only are obligatory in the interest of adequate medical care, but they may also be called upon in the court of law, later on for evidence. Thus, the medical records encompass routinely the *who, what, where* and *when* of the patient care in the hospital.

Customarily, there is a separate department in most of the hospitals with trained/qualified personnel maintaining these formalities. Such department is called *Medical Record Department*, often referred to as MRD.

CONTENTS OF MEDICAL RECORDS

The medical records should contain the following:[1,4]

- Particulars of the patient, e.g. name, age, sex, address brought by (name of the person who has brought him to the hospital), referred by, etc.
- Date and time of arrival and examination in hospital.
- Date and time of admission and discharge from hospital.
- The present complaints by the patient at the time of arrival.
- Relevant past history.
- Relevant family history.
- Relevant personal history.
- Details of physical examination done by the physician and the findings.
- Laboratory examinations and other investigations adviced for and their reports, e.g. blood sugar, blood urea, X-ray, etc.
- Treatment given.
- Duly completed consent form for each and every procedure/s and operation/s performed.
- Prognosis chart.
- Details on cross consultations/references to other specialist doctor/s and his/their opinions and reports.
- In case of discharge from hospital—the condition at the time of discharge.
- Maintain discharge card with discharge summary providing brief information on admission particulars, investigations done, treatment given and follow-up advices given at the time of discharge to the patient. If the patient is referred by a *family* physician a *copy* of the same may better be marked to him as well with all instruction to be carried out by him on discharge from the hospital.
- If the discharge is *against medical advice* (AMA), then record accordingly and take signatures of patient and/or his/her guardian/relative with whom patient leaves the hospital).
- Copy of *Police Intimation Letter* with all details of information given to the police in every medicolegal cases (at admission and/discharge on recovery/on death of the patient).
- In the case of death, note down the cause, date and time of death.
- Name and signature, address, medical council registration number/license of doctor.

In Medical Cases

In addition to routine contents mentioned above, certain additional precautionary measures are to be observed in all medical cases and they are:[1,4]

- The casualty MO must ensure that all the registers are numbered and duly certified.
- All pages of the record should be serially numbered.
- On all pages, laboratory reports and X-ray plates, word MLC should be marked. It should be so even on requisition for laboratory investigations and X-ray.
- All the entries should be correct and in detail and in sequential order.
- Abbreviations should be avoided.
- All corrections done should be initialed.
- All medicolegal documents should be prepared in duplicate.
- All communications with police should be in writing only and copy of all such correspondence should be attached to the case papers/file.
- There is no time limit as to when the medicolegal case records can be destroyed by the hospital.
- All records should be kept under lock and key.
- All entries in hospital papers should carry the signature and name of the doctor concerned.

Property Rights of Medical Records

The medical records and also X-ray plates are property of the hospital.[1,2,4] The patient buys the expertise and the treatment rather than the hospital records and the X-ray films. All records are kept in the hospital for the benefit of the patient, doctor and the hospital. In no situation does a patient own his/her records, though he/she has a legal right to the use of the information contained therein.[2,3]

Patient's Rights

Usually, the patient is given a copy of the investigation reports, treatment advised and the discharge summary. Patient has the right to know the details in his/her records and is entitled to get a copy of his/her hospital record on discharge, on payment of cost of reproduction.

In case of death of the patient the next of kin can have the hospital records. However, if in the opinion of the doctor, making the records available to the patient would be harmful or dangerous to patient (professional or therapeutic discretion); he/she may avoid issuing the records to the patient.

The records cannot be used by the hospital or the doctor, for publication, without the patient's consent.

Medical Records in Court

When the hospital/doctor have been summoned by the court, requesting for production of the case records, they have to be produced before the court without failure.[1,4] The court may require the medical records in all alleged *criminal cases* such as assault, burns, criminal abortion, dowry deaths, injury, murder, poisoning, rape, suicide, and vehicle accidents, etc. In some of the *civil cases* also medical records may have to be procured by the court. Workman's compensation cases, insurance claims cases, malpractice/negligence suits, cases of "contested Will", disputed paternity cases, etc. constitute some of the examples of civil cases required by the court.[4]

Information about the health of a patient given to the law courts is covered under *privileged communication* and the doctor in *immune* to the charges of breaking professional secrecy under such circumstances.[1,2]

However, hospital should arrange for photocopying every page of the case file prior to taking them to the court, as the court usually retains the records.[1,4] Whenever court needs the document to be retained, hospital doctor should demand a receipt from the court specifying clearly the total number of pages withheld by the court.

Submission of Records to Government and Other Agencies

On several occasions' government and other agencies such as LIC place a request to supply the information about a patient treated in the hospital. As per law, they are not entitled to this information without the written consent[1,2,4] of the patient and hence the hospital should not comply with such requests. However, information about name, age, sex, date of admission and date of discharge, etc. can be given as these are not confidential.

Storage and Disposal of Medical Records

Storage and disposal of medical records is an essential matter for every hospital. Ever since computerisation, medical records have become simple. Data can be fed into the computers and preserved easily in computer files or on CDs and using CD writer. Such documents can be preserved for any length of time as CDs require minimum space for preservation unlike hard copies/printed or typed documents of a case. However, following scheme is usually adopted in different types of cases:[1]

- Nonmedicolegal Cases (Non-MLC)
  - The OPD records have to be preserved for a minimum of 3 years, when they can be destroyed.
  - The IPD records have to be preserved for a minimum of 5 years.
- Medicolegal Cases (MLC)
  - There is no specified time limit and hence they cannot be destroyed and must be made available as and when needed.

Medical Records and Research

It must be remembered here that no medical records can be prearranged or provided to any of the research workers without prior written consent of the patient and an approval of hospital ethical committee.

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Chapter 10

Medical and Legal Aspects of Anaesthetic and Operative Deaths

INTRODUCTION

Anaesthesia has its own special dangers. Anaesthetists, along with surgeons presents a common target for litigation: The actual administration of anaesthetic is not usually the cause of complaint, but the many ancillary responsibilities such as transfusion, injections, airways, intravenous catheters, diathermy, and hot-water bottle burns may form grounds for allegation of negligence.[1-5]

In recent years, claims of awareness under anaesthesia have become a frequent complaint. One of the most tragic and expensive anaesthetic mishaps is the production of cerebral damage from hypoxia due to failure to maintain oxygenation during the operation. Inattention on the part of the anaesthetist is a more common cause than failures of equipment and recent surveys have shown that inexperienced junior anaesthetist is a major cause of problems.

Majority of the operations require the administration of anaesthesia to the patient. This being a complex and special procedure demands special knowledge and experience.

As far as possible the anaesthetist should be an expert. He should examine the patient properly prior to the surgery. He should also plan and prepare for administration of anaesthesia depending on the surgery. A written informed consent from the patient or his/her guardian must be obtained in advance.

A great variety of unfortunate events/mishaps can occur during or following the administration of anaesthesias and operative or investigational procedures. These do not necessarily convey an error of judgement or negligence on the part of the surgeon or the anaesthetist and can happen in spite of properly *calculated risk*. Anaesthetic deaths are very rare and *only one* in *ten thousand* person die totally as a result of anaesthesias. Table 10.1 narrates the exact incidence of anaesthetic deaths. Anaesthetist should ensure the safety of the patient. However, in a case of the death under anaesthesia it is better to report the matter to police for conducting inquest and postmortem examination.

At autopsy, known sites of sudden catastrophe should be carefully investigated for, such as coronary disease, pulmonary embolism and inhalation of vomit or blood. All too often the tragedy may be due to a combination of errors in varying proportions rather than one particular mistake.

However, any such death believed to be caused, or contributed to, by any of these procedures may be adequately investigated both from the point of view of the satisfaction of the relations of the deceased and instituting future safety/preventive measures.

ANAESTHETIC DEATHS

Anaesthetic deaths are of two types:

1. Deaths due to anaesthesia and anaesthetic agents.
2. Deaths due to factors other than anaesthesia.

Deaths Due to Anaesthesia and Anaesthetic Agents

Deaths due to anaesthesia and anaesthetic agents could be due to three reasons:

1. Anaesthetic agents
2. Anaesthetists
3. Functional problems.

Anaesthetic Agents

Anaesthetic agents may sometimes result in *hypersensitivity* reaction resulting in death of the patient. Certain anesthetics can directly act with a consequence of *cardiac arrhythmia* and *cardiac arrest*.

The use of certain drugs, which can create myoneural blockage, may give rise to death due to *respiratory inadequacy/failure*.

There is evidence that *halothane* can cause *liver necrosis* resulting *in malignant hyper pyrexia* which is characterised by abrupt rise to dangerous temperature (about 110°F) and also may be associated with tachycardia, hyperpnoea, cyanosis and stiffening of the muscles and may ultimately lead to death. This condition is believed to be genetically determined and likely to occur in families having evidence of subclinical myopathy and an exceedingly high level of serum creatinine phosphokinase (Table 10.2).

Table 10.1: Incidence of anaesthetic deaths[6]

Reported causes	*Incidence(%)*
Disease for which the operation was conducted	56
Shock and inevitable risks of the operation	30
Risks and complications of anaesthesia	08
Over dosage, maladministration or bad choice of the anaesthetic agent	06

Table 10.2: Causes of death due to anaesthetic agents

- Hypersensitivity
- Cardiac arrhythmia
- Cardiac arrest
- Respiratory inadequacy
- Malignant hyperpyrexia

Anaesthetists

Anaesthetists who are using improper technique, improper equipments or one who has no familiarity with the equipment, having no adequate experience, or unable to adopt precautions when indicated, or careless in the methods, etc. can always land up with anaesthetic deaths.

Hypoxia, improper depth of anaesthesia, vagal inhibition, etc. constitutes usual causes of anaesthetic deaths. Basically, all these causes are secondary to obstruction of the airways, or a faulty gas connection, etc. due to mechanical problems consequent to several causal agents (Table 10.3).

However, it is reported that of all these causal agents, *human error* alone was responsible for 82 per cent of the anaesthetic deaths, while *equipment failure* occurred in another 14 per cent of cases and all *other factors* caused death in rest of the 04 per cent patients.

Functional Problems

The common problems relate to vagal inhibition, obstruction of the glottis due to spasm, tube, or vomit; cardiac arrhythmia; and hypotension. The unconscious patient poses a special problem in regard to anesthesia, as he is unable to take corrective reflex action against inhalation of foreign material. Table 10.4, enumerates the functional causes of anaesthetic death.

Deaths Due to Factors other than Anaesthesia

Deaths due to factors other than anaesthesia are enumerated and discussed as follows:

- Disease or injury for which the operation or anaesthesia is being given. Here the anaesthesia or surgery is playing no role in causing the death of the patient. Rather it is the disease or the injury in itself has resulted in death. The surgery is usually accepted as a challenge with the remote hope to save the victim's life.
- Disease or abnormality other than that for which the surgical operation is undertaken.
- Surgical mishaps and/or postoperative events.

Table 10.3: Causal agents and cause of death

Human error (82%)
- Carelessness
- In-experience
- Unfamiliarity with equipment
- Inability to adopt precautions
- Mishaps due to intubation/bronchoscopy

Equipment failure (14%)
- Kinked pipes
- Cross tubes
- Over dosage
- Malfunction of the apparatus
- Explosion

Other factors (04%)
- Inadequate communication between staff
- Haste
- Distraction

Table 10.4: Functional problems of anaesthetic deaths

- Vagal inhibition
- Obstruction of the glottis due to spasm, tube, or vomit
- Cardiac arrhythmia
- Hypotension
- Unconscious patient

- Physical status of the patient, e.g. old age, diabetes, high blood pressure, etc.
- Surgical mishaps such as unintentional accidental tearing or cutting of a major blood vessel during surgery resulting in death and therefore such deaths are detectable only at autopsy.
- Postoperative consequences such as death due to phlebothrombosis, pulmonary embolism, aspiration of the vomit, etc. These cannot be considered as part of anaesthetic procedure or that of surgery. Undoubtedly an important postoperative precaution with an accomplished nursing care maintaining the safe position of the patient can certainly prevent this.
- Unforeseeable problems—patients with haemoglobinopathies, especially sickle cell anaemia, are unduly susceptible to low oxygen tension in blood and this may pose a hazard to the unawarded surgeon or anaesthetist. Coronary thrombosis may supervene in a patient operated upon for injuries. Transfusion hepatitis is not unknown. AIDS infection through transfusion is another possibility.

MODE AND CAUSE OF DEATH

Two important modes of death are *cardiac arrest* and *respiratory failure*.

Cardiac Arrest

Cardiac arrest is the most common mode of death. Basically, this is due to either oxygen deprivation or carbon dioxide accumulation as a result of *failure* of technique or *fault* in technique. Most cardiac arrests occur under relative light anaesthesia and therefore tend to occur at either the start of the operation or conclusion of the surgical procedure. Cardiac arrest, thus supervene in three ways:

1. Asphyxia of myocardium (Flow chart 10.1)
2. Overdosing of anaesthetic agents
3. Reflex vagal stimulation.

Asphyxia of Myocardium

Hypovolaemia and some diseases of the cardiovascular system carry an enhanced risk. Presented below are the various events of effects of asphyxia on myocardium resulting in cardiac arrest.

Overdose of Anaesthetic Agents

Over dosage during anaesthesia, act in two different ways:

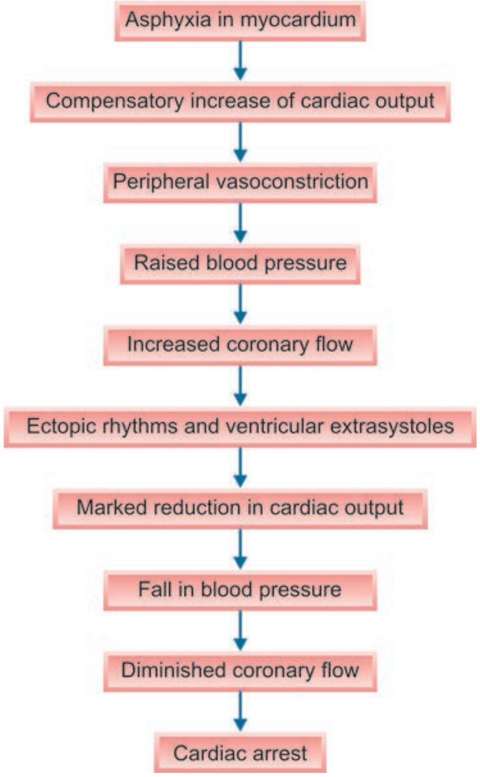
Firstly, there may be an inadvertently high concentration of the agent in the bloodstream. This will produce marked vasodilatation, causing a fall in blood pressure while the heart is receiving a relatively high concentration of the agent via the coronary vessels. The heart may become sufficiently poisoned so that it is unable to produce a compensatory rise in output and will fail ultimately.

Secondly, cardiac arrest may be caused by prolonged administration of an anaesthetic in concentrations sufficiently high to gradually poison the myocardium.

Reflex Vagal Stimulation

Hypoxia, sudden asystole can stimulate vagus nerve resulting in slowing of the heart. Vagal stimulation *per se* is unlikely to occur spontaneously when the circulation is hypoxic, but respiratory irritation causing bronchial spasm results in vagal over activity and hypoxia. It has been postulated that patients suffering from a natural over activity of the parasympathetic system are more likely to have such a reflex arrest.

Flow chart 10.1: Presenting cardiac arrest due to asphyxia of myocardium


Asphyxia in myocardium
Compensatory increase of cardiac output
Peripheral vasoconstriction
Raised blood pressure
Increased coronary flow
Ectopic rhythms and ventricular extrasystoles
Marked reduction in cardiac output
Fall in blood pressure
Diminished coronary flow
Cardiac arrest

Respiratory Failure

Death due to respiratory failure may result during and/ or after the anaesthesia and surgical procedure. Various potential causes are enumerated as below:

- Overdose of premedication drugs such as barbiturates, tranquilizers, morphine, pethidine, etc can depress respiration, leading to hypoventilation and anoxaemia.
- Overdose of anaesthetic drugs/administering deep anaesthesia with consequence of the respiratory muscles paralysis.
- Administration of opiates during postoperative period for the relief of pain may depress the cough reflex causing retention of the sputum leading to secondary infection of lung.
- Obstruction of larynx or trachea by laryngospasm and/or bronchospasm, secretions from the throat, blood, swabs, dentures or gastric contents may lead to hypoxia.
- Hypoventilation and hypoxia due to hyperventilation by the anaesthetic agents may cause depletion of carbon dioxide, during the recovery-period.

INVESTIGATION AND EXAMINATION OF A CASE OF AN ANAESTHETIC DEATH

In investigating a case of an anaesthetic death, a forensic pathologist should take several factors into consideration. A thorough review of hospital chart and discussions with the surgical and anaesthetic team is essential collecting relevant history pertaining to victim about how was his period prior to hospitalisation; during stay in the hospital, at the phase of preparation of anaesthesia, and for how long the anaesthesia affect lasted. Enumerated below are some of the important factors to be evaluated individually:

- *Condition for which surgery was performed:* Look for high-risk surgical conditions, e.g. resection of an aortic aneurysmal repair.
- *Preexisting other conditions:* Some contraindications to operative procedures are not easy to identify. Even if these conditions are identified, their seriousness may not be appreciated. For example, coronary artery disease may prove fatal due to increased anoxia by the anaesthetic agents.
- *Anaesthetic agents:* Inadvertent mixing of the anaesthetic gases may cause death. It is important to get information on anaesthetic agents used, its quantity and method of administration. Note also the duration of time for which the patient remained under anaesthesia.
- *Burn or explosion:* Deaths from anaesthetic explosions occur rarely.
- *Shock and haemorrhage:* Haemorrhage and shock should be evaluated with other findings of the case.
- *Blood transfusion:* Blood transfusion reactions and incompatibilities should be also looked for.
- *Resuscitative measures:* The measures adopted should be noted.
- *Equipment:* With appropriate qualified individuals, all the equipments including the valves and containers should be checked to assure the correct mixing of per centages. Devises attached to and inserted into the body should be examined.

AUTOPSY EXAMINATION IN A CASE OF AN ANAESTHETIC DEATH

This includes three things: *Precautions, Autopsy procedures* and *Chemical analysis*.

Precautions

In order to avoid criticisms it is better take all necessary cautions preventing all harms or dangers.

- Surgical mistakes are gross and anatomical and hence are observable at the postmortem.
- Anaesthetic mistakes being physiological are no longer appreciable after death except where overdose with specific drug is involved.
- Look for or exclude some of the natural disease or mechanical obstruction.
- Autopsy must be preferably done by a forensic expert and it must, however, be remembered that the findings of the autopsy surgeon alone will not be sufficient to explain death.
- It is imperative to hold a discussion across the autopsy table involving forensic expert/autopsy surgeon, anaesthetist and the surgeon concerned.
- It is often stated that deaths under anaesthesia were more often the fault of the anaesthetist than the anaesthetic.

Autopsy Procedure

Autopsy in a case of an anaesthetic death must be performed methodically adopting all the standard procedures. However, care is taken to undertake following unfailingly:

- *Note the odour*: With inhalant anaesthetics, specific odour of anaesthetic agent may be detected at autopsy.
- *Body cavities*: Examine *in situ* all the cavities. Measure the contents or fluids if any and preserve for analysis.
- *Site of surgical intervention*: Examine the site of surgical intervention *in situ* and describe in detail.
- *Surgical sutures and organs*: Dissect all organs and inspect every surgical suture.

- *Signs of prolonged anaesthesia*: Dependent parts of the viscera are usually seen engorged in cases of prolonged anaesthesia.
- *Effect of anaesthetics*: Chloroform and halothane are hepatotoxic and chloroform may rarely produce ventricular fibrillation. Halogenated hydrocarbons like cyclopropane, trichloroethylene and halothane cause cardiac irritability. They sensitize myocardium to the action of adrenaline.
- *Evidence of pulmonary embolism and asphyxia*: Look for presence of pulmonary fat or air embolism or evidence of asphyxia due to aspiration of regurgitated material which are diagnostic about cause of death.
- *Internal findings* of haemorrhage, peritonitis and retained swabs and instruments, or evidence of hypersensitivity reaction are obvious.
- *Evidence unnoticed*: Evidence of vagal inhibition, fall in blood pressure, cardiac arrhythmias, coronaries and laryngeal spasms, etc. could not be detected during an autopsy.
- *Histopathological examinations*: Collect the sample from all viscera for histopathological study.
  - Specimens should be taken particularly to exclude any cardiovascular disorder including occult conditions like *myocarditis* as well as relavent specimens for assessing the *severity of disease* for which the operation was carried out.
  - Histological examination of the brain is imperative which is primarily intended to demonstrate the effects of hypoxia, particularly in the region of *Sommer's area* of the *hippocampal gyrus* and *the cerebellum*, where changes are expected even if the victim suffers hypoxia for a short period.
  - Morphological changes[5] in the brains of victims who suffered hypoxia for a short period but survived for long periods after anaesthesia included-diffuse, severe leucoencephalopathy of cerebral hemispheres with sparing of the immediate subcortical connecting fibres. Demyelination and obliteration of axons was also observed and at times, infarction of the basal ganglia. Damage appeared limited to the white matter, which is explained on the basis of greater glycolysis in the white matter during hypoxia as compared with the grey matter.

Chemical Analysis

- A lung is removed and collected by clamping the main bronchus and retained in a nylon bag and sealed so that the headspace gas can be analyzed.
- Collect the alveolar air with a syringe by pulmonary puncture before opening the chest.
- Prior to autopsy to avoid loss of gases due to exposure of the tissues to the air, it may be necessary to obtain samples of every viscera by the *biopsy techniques* and *frozen immediately*.
- At autopsy some portion of fat from the mesentery, skeletal muscle tissue, brain, liver, half of each kidney are retained.
- Blood should be collected under liquid paraffin.
- Urine should also be collected, if available.

All these specimens should be collected in containers with as little headspace as possible, sealed and immediately refrigerated or frozen.

Bacteriological Examination

Adequate blood, urine and other body fluids may have to be collected.

Hazards of Transfusion of Blood and Body Fluids

Blood and various body fluids should be preserved for analysis.

Extraneous Specimens

Like residual solutions, medication containers, samples of gases used for the anaesthesia and samples of the operating room air may have to be collected in occasional cases.

COMMON MISHAPS IN ANAESTHETIC PRACTICE

The most common errors leading to anaesthetic complications are *human error*. It may be due to lack of vigilance due to haste or fatigue. Inexperience or inadequate knowledge may also contribute to this. Some of the common mishaps encountered are enumerated in Table 10.5.

Mistaken Identity

It is quite common that due to mistaken identity, a wrong procedure may be initiated on any individual. Mistaken identity can result in *mismatched transfusion* leading to serious complications. In India, the common practice is to place a leucoplast sticker containing name, age, sex and ward of the patient on the arm or sternum of the patient. This sticker may be accidentally washed or it may come out due to sweat of the patient. In Western countries, special identification bands are available which cannot be removed easily and are washproof. Efforts made in making such identification bands available in the Indian market and it is advisable to use them whenever available. It is advised that before commencing any procedure, identity of the patient may be confirmed again by asking the patient or his relatives.

Incorrect Positioning of the Patient

Incorrect positioning of the patient on the table can create complication like nerve palsy or problems in cardiovascular or respiratory management. It is true that the surgeon decides positioning of the patient, but anaesthetist should take precautions and caution the surgeon when required.

The following precautions are recommended:

- Eyes should be shut and well protected. Pressure on eyeballs can cause retinal vein thrombosis and blindness.
- Elbows should be protected as allowing them to lie unprotected against table can cause *ulnar nerve palsy*.
- Abduction of arms should not be done beyond 90°. Excess abduction can result in traction injury of bronchial plexus.
- Sciatic nerve injury can occur during movement to *lithotomy position,* if both knees and hips are not flexed simultaneously.
- If patient is in prone position, there may be pressure on abdomen, which can cause obstruction of venous return, and there may be restriction in the movement of diaphragm.
- If there is sudden change of movement, it can cause venous pooling and interference with cardiac output. It may also cause sudden *extubation*.

Table 10.5: Common mishaps in anaesthetic practice

- Mistaken identity
- Incorrect positioning of the patient
- Fault in the anaesthetic machine
- Failure of suction apparatus
- Perforation of airway
- Electrocution or burns
- Fault with intravenous equipment,
- Mishaps with drugs
- Monitoring of vital signs
- Human error

- Protect the patient from hazards of electrocution by avoiding contact of the patient with the metal parts of the operating table.

Faults in the Anaesthetic Machine

There may be faulty gas supply due to incorrect connection or leaks. It may be due to cylinders being empty or proper pipeline supply is not there. Flow meters may have leaks or incorrect settings. In vaporisers a wring agent may be there or there may be leaks or errors in calibration.

- *Faulty anaesthetic circuit*: The anaesthetic circuit may be faulty due to disconnection or leaks or improper setting of APL valve and pressure limiting device.
- *Fault in ventilator*: It may be due to disconnection or leak or electric current failure.
- *Laryngoscope* may cause injuries to lips and teeth. There may be failure in light supply.
- *Endotracheal tube* may be leaking, cuff may be ruptured, obstruction may be present due to kinking. There may be bronchial or oesophageal intubations.

Failure of Suction Apparatus

This is grave especially in emergency cases.

Perforation of Airway

Malleable stylet may cause such perforations.

Electrocution or Burns

Electrocution can occur because of leakage in electric circuit commonly with use of cautery. Burns are also reported because of leakage of current.

Fault with Intravenous Equipment

Catheters may be misplaced. Sometimes, air embolism or embolisation of catheter fragments is reported.

Mishaps with Drugs

Improper labeling can lead to wring drug being administered. If by mistake expired drug is used, it can cause overdose or underdose.

Monitoring of Vital Signs

Every anaesthetic procedure requires close monitoring of vital signs at regular intervals. It is one of the most important areas of anaesthetic care. Patient's condition may worsen if there is a failure to recognize signs of impending danger.

Human Error

Three types of human errors are described:

a. Errors due to failure of monitoring and vigilence.
b. Technical errors due to inefficiency in skills required or due to poor design of the equipments and apparatuses.
c. Judgemental errors due to bad decisions by improper/poor training in skills of decision making.

PREVENTING ANAESTHETIC MISHAPS

Proper Maintenance of Equipments

All anaesthetic instruments should be regularly checked for leakage, proper wiring and proper functioning. The following items should be checked before operation:

- Suction apparatus for tubing, catheter.
- Endotracheal equipment like laryngoscope end tracheal tube.
- Intravenous drugs for correct labeling date of expiry.
- Ventilators.
- *Proper vigilance:* It is the cornerstone of anaesthetic practice. Vital signs should be recorded every five minutes. Continuous monitoring of oxygenation and cardiac output should be done.

HANDLING CASE OF ANAESTHETIC MISHAP

- Never panic.
- The situation should be corrected immediately.
- Call for help from other areas or request surgical team to help you out.
  - Document all the procedures done in finest detail as this is the only defense a doctor can have later on that he has done judiciously what was the need of the time.
  - In postoperative period, discuss with patient the circumstances of the case.
  - If patient is dead, talk to the relatives in detail and explain what happened.
  - Not talking to patient/relatives can lead to unnecessary litigation.
  - If you think litigation may follow, inform insurance company from where you have taken insurance policy.

MEDICOLEGAL CONSIDERATIONS

- Code of Criminal Procedure, 1973, Sec. 39—All deaths occurring during the course of anaesthesia and surgery or within a reasonable period thereafter should be reported to the police.[7]
- These deaths, *all too often the tragedy may be due to a combination of errors* rather than one particular mistake or sometimes due to some significant pre-existing disease or some co-existent condition.
- Tendency on the part of the relatives of the deceased to impute negligence on the part of the anaesthetist and/or the surgeon merely because of the fact that the death was closely associated with the anaesthesia and surgical intervention.
- Apportioning relative contribution between the anaesthetist and the surgeon is extremely difficult and both are required to exercise due care and skill. Each one is responsible for negligent acts of oneself and not of the other.
- As the surgeon possesses absolute control over the staff that assists him in the operation he will be liable for the negligent acts of his assistants. However, the surgeon, has no absolute control over the activities of the anaesthetist and the connected staff.
- If the postmortem examination yields negative results and the autopsy-surgeon may not be in a position to express conclusive opinion and the cause of death remains nothing more than conjectural one.
- The role of autopsy surgeon may remain limited, largely to the detection of some natural disease, overt signs of damage by the anaesthetic procedure or errors in the surgical procedures.

DEATH ON OPERATION TABLE

It has been estimated that expectancy of death from all causes during operation and anaesthesia is 1 in 1000.[8] The reasons contributing to death on operation table are mainly due to anaesthesia and surgery. It may be possible that person may die from the injury or disease for which operation is carried out; for example, in repair of ruptured liver or ruptured lung, person may die within the stage of organ being repaired. Death may also due to a disease other than that for which operation is being carried out but was diagnosed or not diagnosed before operation. It is possible that a person may die of pre-existing disease like rheumatic heart disease, rupture of aneurysm or myocardial infarction during surgery. Major surgical complications like shock can also cause death on operation table.

No surgery is without a risk is a famous saying and is true. But maximum number of lawsuits is against surgeons only. While during surgery, he is captain of the team and, by the doctrine of *respondent superior,* is responsible for any mistakes of any member of the team. Negligence attributed during surgical practice can be divided into the following:

- Negligence due to anaesthesia and surgeon has no role to play.
- Negligence primarily by surgeon alone.
- Negligence by operating assistants.
- Corporate negligence during surgery or in postoperative phase.

Negligence primarily due to anaesthesia and surgeon has no role to play (Discussed above).

Negligence Primarily by Surgeon Alone

Described as *Acts of Omission* and *Acts of Commission*.

Acts of Omission

- Failure to assess surgical condition properly.
- Failure to decide whether surgery is required or not.
- Failure to decide correct surgical approach.
- Delay in planning operation leading to complications.
- Failure to use diagnostic techniques properly.
- Failure to take informed consent.
- Failure to carry out operation properly.
- Failure to provide good postoperative care.
- Failure to provide instructions and precautions to patient.
- Failure in follow-up of patient regularly.

Acts of Commission

- Operation more extensively carried out than consented by the patient.
- Operation conducted on wrong patient or on wrong side.
- Leaving swabs or instruments in the body after surgery.
- Use of unsterile instruments or operation theatre.
- Unnecessary cutting of body tissues.
- Applying plaster casts too tight or too light for a longer time than required.
- Committing major blunder like cutting of big vessel or respiratory passage inadvertently.

Negligence by Operating Assistants

Surgeon is fully responsible for the mistakes of his assistants like nurses and other paramedical staff during operation, although they also have to share responsibility for the mistakes they have committed.

Corporate Negligence during Surgery or in Postoperative Phase

Due to faulty instruments, inadequate facilities in operation theatre, etc. patient may receive injuries during operation or operative care.

For example,

- Leaking cautery during operation may electrocute patient.
- Patient may fall off from the operating table due to a defective table.
- Patient may get injured while being shifted from one place to the other.

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Part III: Forensic Pathology

Chapter 11

Forensic Identity

INTRODUCTION

Identification of a person is vital among the *living* and the *dead*. In civil and criminal courts, results of trials often depend upon establishing proper identity. In deaths due to violence, law needs to establish the exact identity of the deceased prior to final verdict. Identity happens to be the part and parcel of *corpus delicti* or body of crime.

Identification of the Living

This is usually done by the police and is essential in both *civil* as well as *criminal* cases. Identification of the living becomes necessary when through *debility, illness, mental confusion* or *unsoundness, immaturity, infancy, unconsciousness* or *true amnesia*, evidence of identity is not forthcoming from a person who has *no relatives* or *friends* immediately available and who carries no *documentary* evidence of identity. Such cases are sometimes seen in *vagrants, residents* of lodging houses or hotels, *victims of accidents, fires and mass tragedies or disasters,* such as rail, air crash or ship wreck. Other circumstances where a living person may require establishing of identity are—*absconded* soldier, a *criminal* accused of assault, rape, murder, etc. cases of swapping of neonates in a maternity home, cases of *false impersonation* for insurance claims, inheritance of property, passport, school admissions, disputed paternity, dubious sex identity, etc.

Identification of the Dead

In a dead body, identity is often of paramount importance in criminal or suspicious death investigation, as in cases of victims of *homicides,* where in the body may be *mutilated*. Identification of the victim is hence a major step towards identification of the culprit responsible for the death. *Decomposition* and *skeletonisation,* may render identity even more difficult. The other important situations where in identity of dead body is vital, are among the victims of mass disasters in natural calamities such as an *earthquake* or a *terrorist bomb blast* or *conflagration* of a crowded building.

Thus, detailed examination of the dead for evidence of identity becomes a specialised task for the *forensic pathologist* and *other forensic experts*.

Pedigree

Pedigree is a method of establishing identity of the criminal involved in a crime in a police station. It include personal details such as name, his/her alias, address residence, colour of the skin, age, height, build, colour of the hairs, colour of the eyes, complexion, presence or absence of moustache, birth place, occupation, etc. This when combined with photographs and finger prints serves in establishing the identity of a criminal.

DEFINITION

Identity is defined as the *recognition of the individuality* of a person, live or dead.

CLASSIFICATION

For all purposes, personal identity is classified into two types: *complete identity and partial identity*. In addition, *legal identity* is described herewith.

Complete or Absolute Identity

Here the exact fixation of individuality of the person, alive or dead, is possible.

Partial or Incomplete Identity

Here the exact fixation of individuality is not possible, but identity to the extent of certain facts about the person is only possible, i.e. age of the person, sex of the person, race of the deceased, occupation of the person, etc. is possibly established.

Legal Identity

Here the exact fixation of the individuality is impossible, because he or she has been unknown to the people around. Such cases are coded by a number or alphabetical letter or such other methods by the police, constituting legal identity.

Examples: A dead body, recovered by the police in the central part of the city, being unknown is labeled and registered as body No: A. Another dead body recovered in some other part of same city, if again unknown, for registering the case may be labeled as body No: B, etc.

Discussed below are various aspects of *personal identity* with respect to its applied medicolegal context briefly.

CORPUS DELICTI (BODY OF OFFENCE, ESSENCE OF CRIME)

In a trial of homicide, it is necessary to establish identity of the person who is dead.[1] Corpus delicti means facts of any predefined criminal offence. Thus in a case of homicide, corpus delicti includes not only the *dead body* but also all *other factors* which are conclusive of death by *foul play* such as a bullet found at the crime scene or in the dead body, a piece of broken knife at the crime scene, a drawing/photograph of the deceased showing fatal injury, etc. all may be included.

The main fact of corpus delicti is the establishment of identity of the dead body, infliction of violence in a particular way, at a particular time and place, by the person or people charged with the crime and none other. The case against the accused cannot be established unless there is convincing proof of these points. If the identity of the victim is not known, it becomes difficult for the police to solve the crime. The identification of a dead body and proof of corpus delicti is essential prior to passing sentence in murder trials. However, there are cases reported where in the death sentence is passed even when the dead body was not found or identified.

Dermatoglyphics or the ridge patterns of skin of the palms and soles are highly individualistic, providing positive identification of mutilated or putrefied remains. The advent of DNA finger printing helps conclusive and complete identity even with a few drops of blood or body fluids, or a bit of tissue, hair, bone, teeth, etc. This is possible even when there is no antemortem record of the dead, since the DNA can be compared with that of the close/blood relatives of the deceased.[2,3,23,24,29]

FACTORS ESTABLISHING PERSONAL IDENTITY

Identification of a person live or dead can be established in general by certain factors,[1-6] which are illustrated in Figure 11.1 and enumerated below:

1. Age
2. Sex
3. Other factors:
   - Race and religion/ communal characters
   - Stature and general development
   - Hairs
   - Complexion
   - Features
   - Deformities
   - Tattoo marks
   - Moles
   - Scars
   - Occupational stigmata
   - Dactylography
   - Poroscopy
   - Footprint
   - Lip prints
   - Palato prints
   - Ear prints
   - Anthropometry
   - Superimposition
   - Personal belongings:
     - Clothes,
     - Pocket contents,
     - Key bunch
     - Wrist watch
     - Jewellery, etc.
   - DNA finger printing
   - Brain finger prints
   - Trace evidence factors

Each of these factors are discussed ahead.

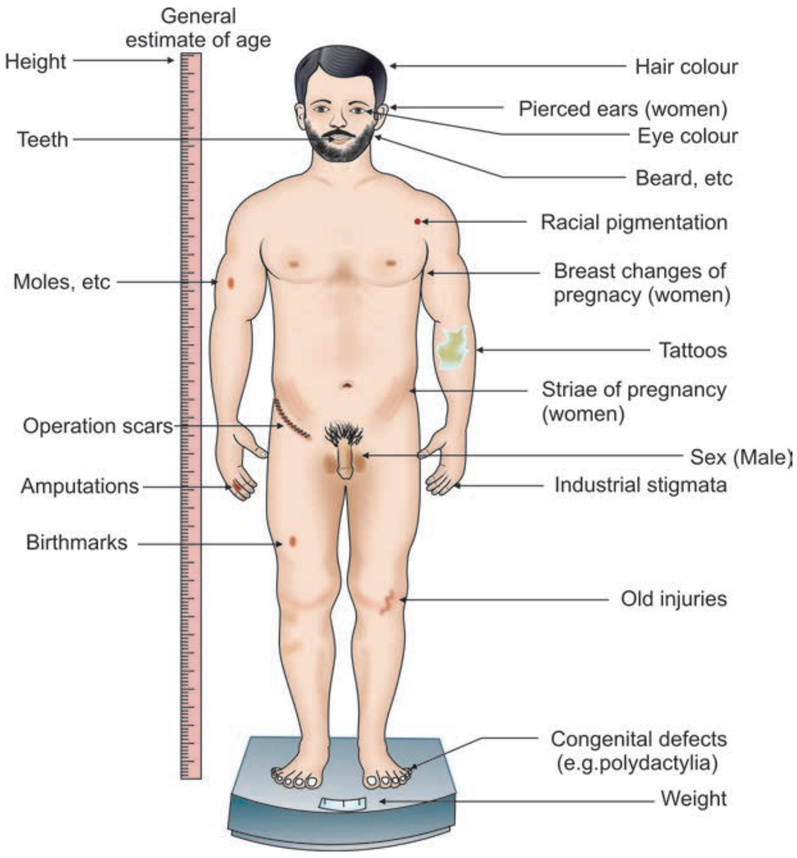

General estimate of age
Height
Teeth
Moles, etc
Operation scars
Amputations
Birthmarks
Hair colour
Pierced ears (women)
Eye colour
Beard, etc
Racial pigmentation
Breast changes of pregnacy (women)
Tattoos
Striae of pregnancy (women)
Sex (Male)
Industrial stigmata
Old injuries
Congenital defects (e.g.polydactylia)
Weight

Fig. 11.1: Factors useful in establishing human identity

AGE AND IDENTITY

Age of a person is an essential factor in establishing the personal identity, and it can be determined by following factors:

- Morphological characteristics
- Dentition (Teeth)
- Ossification of bones
- Miscellaneous data.

These factors can establish the age with a reasonable accuracy almost up to 25 years[3,5] and beyond 25 years, these factors are unreliable.[1-6] However, for the sake of convenience estimation of age is practically considered in three phases of life, namely:

- Intrauterine life (IUL)
- From birth to 25 years age
- Above 25 years.

Intrauterine Life (IUL)

The age of a foetus during IUL can be assessed by studying the developmental morphology, appearance of ossification centres in skeletal bones, and also germination of teeth. A glance at the external and internal foetal autopsy findings emphasize the significance about developmental morphological factors helpful in establishing age during IUL (Tables 11.1 and 11.2). Another method of establishing age during the IUL of the foetus is by Haase's rule.

Table 11.1: External and internal autopsy findings of foetal developmental changes

Features		*Observations*
	Viable foetus (at 7 months)	*Full term foetus (End of 10 lunar months)*
I. EXTERNAL		
Length	35-38 cm	50 – 53 cm
Weight	1060 gm	2.5-5 kg (Average – 3.4 kg)
Head:Circumference	—	33 – 36 cm Head is 1/4th of its whole body length
Fontanels	—	Six (total) and they are: Bregma -(Anterior fontanel) – one at the junction of sagittal with coronal suture (size: 4 × 2.5 cm) Lambda (Posterior fontanel) – one at the junction of sagittal and lambdoid suture. Lateral - two at the sphenoparietal junction on either side Mastoid (posterolateral fontanels) –two at mastoid-occipital junctions on either side.
Eye brows/lashes	Formed	
Eye lids	Open	
Pupillary membrane	Absent	
Face	Wrinkled	Not wrinkled
Nose & Ears	Cartilages not formed	Cartilages formed
Limbs, hands, feet, fingers and toes	Formed	
Nails	Present	Project beyond the tip of fingers of the hands Project up to the tip in toes of the feet
Umbilicus	Cord present	Midway between pubis and xiphoid cartilage
Placenta	Weight: 350-400 gm	22 cm in diameter, 1.5 cm thick at the centre and weight – 500 gm
Genitals	Formed	– Testes are in the scrotum – Vulva is closed and labia minora are covered by fully developed labia majora
Hairs		
a. Body	Lanugo on whole body	Lanugo absent, except on the shoulders
b. Scalp	Present	Dark and 3-5 cm long
Skin	Dusky red, and thick	Pale and covered with vernix caseosa
II. INTERNAL		
Ossification centre appears in	– Talus – Sternum (2nd piece)	– Lower end of femur – Cuboid and – Upper end of tibia
Brain: Convolutions	– Formed	– Well formed
Grey matter	– Not formed	– Begins to form
Intestines, gallbladder - bile	Present	
Meconium	Present in the colon	Dark brownish and present in the rectum
Caecum	In right iliac fossa	
Testis	At external inguinal ring	Present in the scrotum

Table 11.2: Intrauterine age-related changes in the foetus

At the end of IUL (Month)	*Length and weight (cm/g)*	*Sex*	*Skin*	*Hair*	*Eyes*	*Nails*	*Testes*	*Meconium*	*Others*
1.	1 cm 2.5 g	—	—	—	Two dark spots	—	—	—	—
2.	4 cm 10 g	—	—	—	—	—	—	—	—
3.	9 cm 30 g	—	—	—	—	—	—	—	—
4.	16 cm 120 g	Recognized	—	Lanugo on body	—	—	—	In duodenum	Brain convolutions begin
5.	25 cm 400 g	—	Vernix caseosa	Scalp appeared Light	—	Distinct Soft	—	Beginning in large Intestine	Gallbladder contains bile
6.	30 cm 700 g	Well differentiated	Red and wrinkled, subcutaneous fat begins to deposit	—	Eyebrows and eyelashes appear	—	Close to the kidneys, on psoas muscle	—	—
7.	35 cm 900-1200 g	—	Dusky-red, thick, fibrous	Scalp 1 cm long	Eyelids open, pupillary membrane disappear	Thick	At external inguinal ring	Whole of large intestine	Caecum in right iliac fossa
8.	40 cm 1.5-2 kg	—	Not wrinkled	Scalp Thick 1.5 cm	—	Reach tips of fingers	Left testes in the scrotum	—	Placenta weighs 500 gm
9.	45 cm 2.5–3 kg	—	—	Scalp Dark and 2-4 cm long	—	—	Both in scrotal sacs, which are wrinkled	At the end of large intestine	Posterior Fontanels closed, Ossification centre for femur (lower end), Cuboids, Capitates-appeared

Haase's rule: This enables the estimation of the age of the foetus in lunar months from the crown-heel length (in cm) of the foetus. Accordingly, until the foetal length is 25 cm, square root of the length determines the age of the foetus and when the length is >25 cm, age is derived by dividing the length by 5.

From Birth to 25 Years Age

Morphological Characteristics

In both living and dead, age of the person can be determined by the *height* and *weight* referring to *standard height and weight for men and women* and also by the secondary sexual characteristics (Table 11.3).

Dentition (Teeth)

The science dealing with establishing identity by teeth is popularly known as *forensic odontology or forensic dentistry*, which is gaining more popularity these days on account of frequent mass disasters.

Eruption of different teeth has a definite pattern and it occurs at different ages. Human dentitions are of two types:[5,10-12] (i) deciduous dentition, and (ii) permanent dentition.

Deciduous dentition (Temporary/milk teeth): These are the teeth present during early part of life, i.e. in childhood and they are totally 20 in number and begin to erupt at sixth month after birth and begin to shed off by sixth year (Fig. 11.2). The tooth distribution in each jaw is as follows:

- 4 incisors
- 2 canines
- 4 molars

Thus, each jaw has 10 teeth.

Permanent dentition: These are the teeth present during life, which begin to erupt from sixth year of age and remain throughout the life. They are totally 32 in number (Figs 11.3A and B). The tooth distribution in each jaw is as follows:

- 4 incisors
- 2 canines
- 4 premolars
- 6 molars.

Thus, each jaw has 16 teeth. Among these 4 incisors and 2 canines and 4 premolars are called as *successional teeth,* which erupt in the place of *predecessor deciduous teeth,* with 4 premolars (1st premolar and 2nd premolar) erupt in place of 4 temporary molars. The remaining 6 permanent molars (1st, 2nd and 3rd molar teeth) erupt independently without any predecessor teeth.[25] These 6 molars are called *superadded teeth*

Table 11.3: Age by height and weight data (Standard heights and weights for men and women— by Life Insurance Corporation, India)

Height		*Men (Weight)*		*Women (Weight)*	
Metres	*0' 0"*	*kg*	*lb.*	*kg*	*lb.*
1.523	5' 0"	….	….	50.8 –54.4	112 – 120
1.5484	5' 1"	….	….	51.7 – 55.3	114 – 126
1.5738	5' 2"	56.3 – 60.3	124 – 133	53.1 – 56.7	117 – 125
1.5992	5' 3"	57.6 – 61.7	127 – 136	54.4 – 58.1	120 – 128
1.6246	5' 4"	58.9 – 63.5	130 – 140	56.3 – 59.9	124 – 132
1.650	5' 5"	60.8 – 65.3	134 – 144	57.6 – 61.2	127 – 135
1.6754	5' 6"	62.2 – 66.7	137 – 147	58.9 – 63.5	130 – 140
1.7008	5' 7"	64.0 – 68.5	141 – 151	60.8 – 65.3	134 – 144
1.7262	5' 8"	65.8 – 70.8	145 – 156	62.2 – 66.7	137 – 147
1.7516	5' 9"	67.6 – 72.6	149 – 160	64.0 – 68.5	141 – 151
1.7770	5'10"	69.4 – 74.4	153 – 163	65.8 – 70.3	145 – 155
1.802	5'11"	71.2 – 76.2	157 – 168	67.1 – 71.7	148 – 158
1.8278	6' 0"	73.0 – 78.5	161 – 173	68.5 – 73.9	151 – 163
1.8532	6' 1"	75.3 – 80.7	166 – 178	…..	…..
1.8786	6' 2"	77.6 – 83.5	171 – 184	….	…..
1.9040	6' 3"	79.8 – 85.9	176 – 189	….	…..

Note: Maximum weight that one may reach: Up to the age of 30 years, 10% above standard, between 30 and 35 years, standard is optimum weight, above 35 years, 10% below standard.

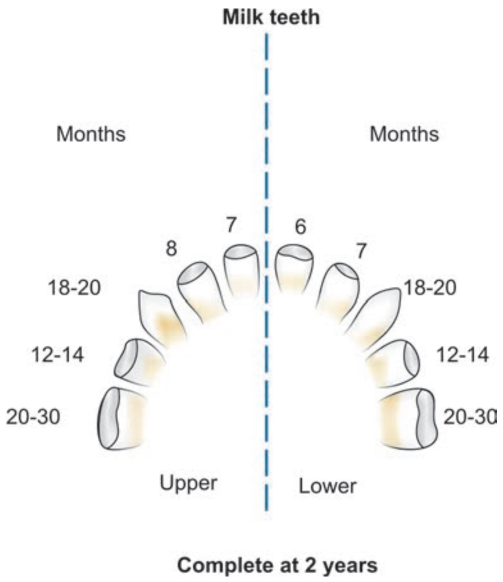

Milk teeth
Months
Months
8
7
6
7
18-20
18-20
12-14
12-14
20-30
20-30
Upper
Lower
Complete at 2 years

Fig. 11.2: Deciduous dentition

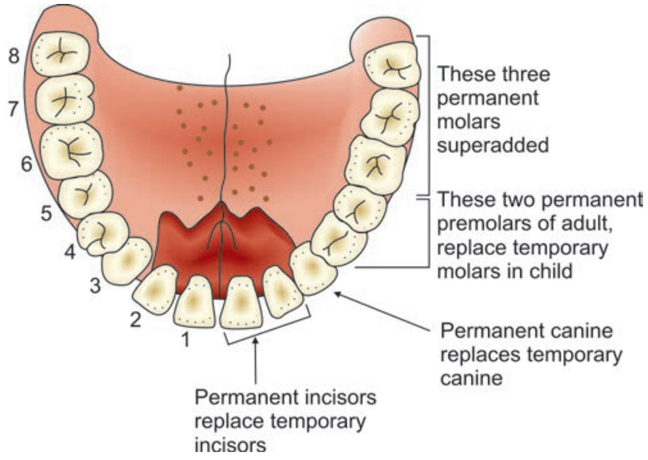

8
7
6
5
4
3
2
1
These three permanent molars superadded
These two permanent premolars of adult, replace temporary molars in child
Permanent canine replaces temporary canine
Permanent incisors replace temporary incisors

Fig. 11.3A: Superadded and successional dentition

(Fig. 11.3A). Table 11.4 enumerates the different ages at which the temporary and permanent teeth erupt, and Table 11.5 gives the important differences between both dentitions. Figure 11.3B, illustrates teeth eruption in a skull of 12 years old age.

Other Factors about Teeth in Determining Age

1. Degree and extent of calcification of roots of teeth - ascertained by X-ray examination.
2. Attrition of teeth, i.e. *wearing off the teeth* on the grinding surface begins in the molars after middle age (Fig. 11.3C).
3. Old age — all teeth are lost and individual becomes edentulous.

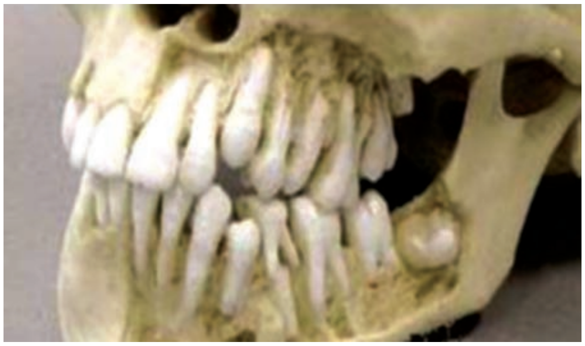


Fig. 11.3B: *Showing tooth development and eruption patterns:* The left side of the maxilla and jaw bone has been cut opened to show adult teeth descending while the deciduous teeth are still in place. The specimen shows two of the teeth are only visible after part of the mandibular cortical bone has been removed. All available evidence suggests an age of 11 to 13 years

Table 11.4: Temporary and permanent dentition eruption ages

Teeth	*Lower/ Upper*	*Temporary*	*Permanent*
Central incisor	Lower	06-08 months	07-08 years
	Upper	07-09 months	07-09 years
Lateral incisor	Lower	07-09 months	07-09 years
	Upper	10-12 months	08-09 years
Canine	—	18-20 months	11-12 years
First premolar	—	Absent	09-11 years
Second premolar	—	Absent	10-12 years
First molar	—	12-14 months	06-07 years
Second molar	—	20-30 months	12-14 years
Third molar	—	Absent	17-25 years

Table 11.5: Differences between temporary and permanent teeth

Characteristics	*Deciduous teeth*	*Permanent teeth*
Size	Smaller	Larger
Colour	Porcelain white	Ivory white
Constriction at the crown-root junction	More prominent	Less prominent
Edge	Sharp	Mammelons/lobes (Serrated)
Cusp	Few and small	More in Number and well developed

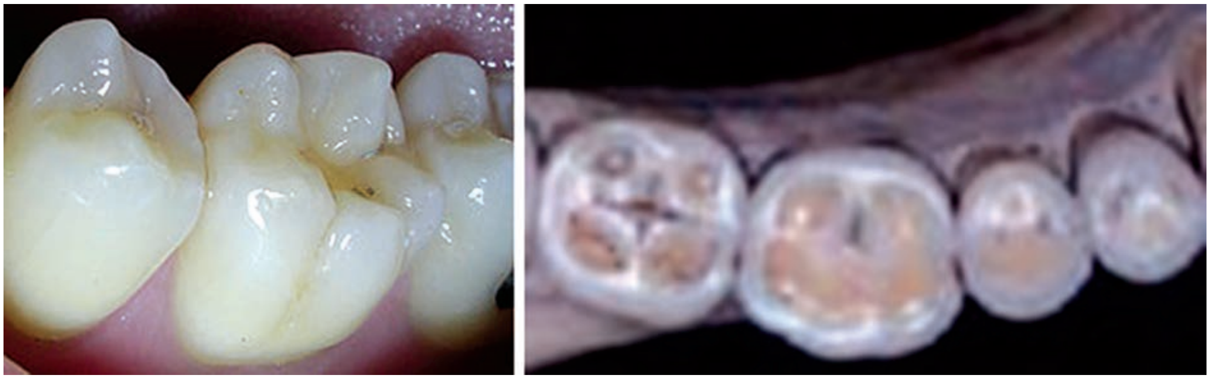


Fig. 11.3C: Normal teeth with prominent cusps (left side), while total worn out teeth (attrition) on right side

Gustafson's Method/formula[1,4,5,11-15]

Gustafson described a method wherein *various criteria* of *normal physiological changes* in a tooth other than dental eruption order are considered in determining the age of a person. Figure 11.4A, Tables 11.6 and 11.7 describe these criteria briefly. The method is useful in determining the age of the victim, when teeth are the only trace evidences collected as in cases of victims of bomb blast or air crash disasters. If the eruption order of teeth could help determine the age up to 25 years, Gustafson's method is helpful in determining the ages beyond 25 years.

Following examination of the tooth based on the aforementioned criteria,[13] the total score obtained is applied to a regression formula and the age is estimated as below:

$$\text{Estimated age (years)} = 11.43 + 4.56 \times (\text{Total score})$$

Other Approved Methods of Establishing Age by Teeth[16-18]

- **Age by cross striations of incremental lines in the tooth enamel**: The age of a person can be determined by counting the number of lines from the neonatal line onwards (Figs 11.4B to D). Accuracy of the method is poor beyond infancy with an error possibility of ±20 days.[16]
- **Age by cemental annulations**: The age is determined by this method at a high accuracy.[17] These are alternating light and dark lines visible under light microscopy of ground section of human tooth root cementum, are believed to be incremental lines and repeat an annual rhythm. Figure 11.4E illustrates transeverse section of human premolar with cemented annulations.
- **Age by racimisation ratio (D/L ratio)**: Here accurate age (± 4 years) estimation is described by the dentin of the teeth using the racimisation ratio of aspartic acid,[18] i.e. ratio between D-aspartic acid (D-Asp.) and L-aspartic acid (L-Asp.) in dentin.

AGE BY CLOSURE OF SKULL SUTURES[1,3,5,6,11]

Skull comprises of two parts — *calvaria* and *face*. Each of these comprises of smaller bones and they are presented in Table 11.8A.[19-22]

Bones of the calvaria are 8 in number, and they are: 2 parietal, one frontal, 2 temporal, one occipital, one sphenoid, and one ethmoid.

Bones of the face and jaws are 14 in number: 2 maxilla, 2 zygoma, 2 nasal, 2 lacrimal, 2 palatine, 2 inferior nasal concha, one mandible and one vomer.

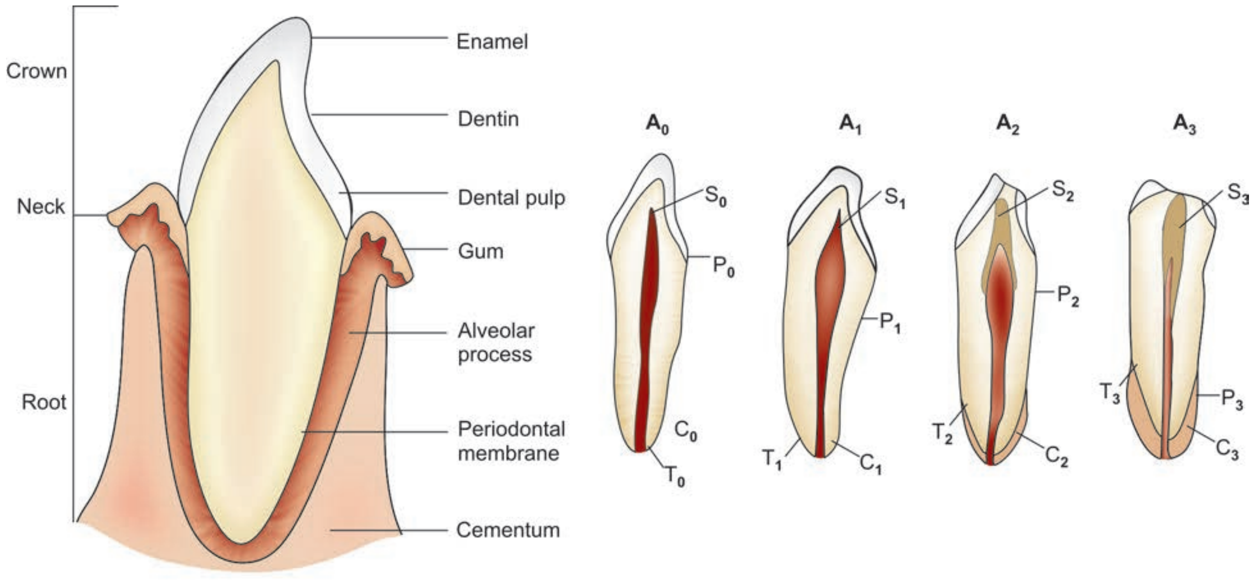


Fig. 11.4A: Gustafson's analysis of age changes in the teeth (left). Normal tooth (right). Criteria and ranking (Refer Tables 11.7 and 11.8)

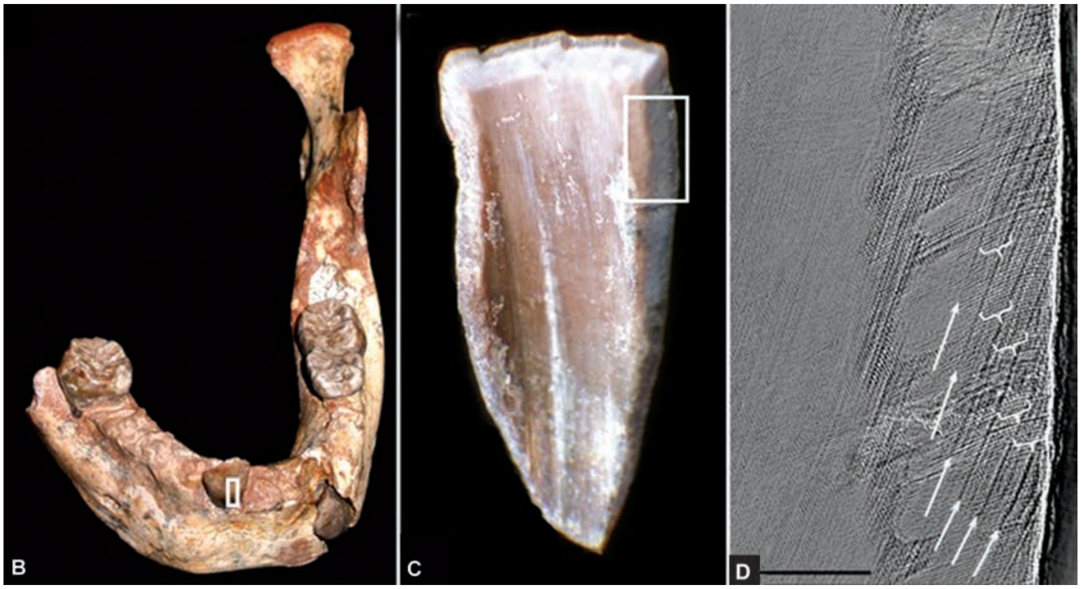
Table 11.6: Criteria of age determination by Gustafson's method/formula[13,15]

Changes assessed	*Descriptions*
Attrition*	Wearing down of incisal or occlusal surface due to mastication (macroscopically and microscopically)
Periodontosis	Retraction of gum margin and loosening of the tooth (macroscopically and microscopically)
Secondary dentin	Seen within pulp cavity, due to aging/ reaction to caries and periodontosis (microscopically)
Cementum apposition	At and around root of tooth (microscopically)
Root resorption	Involves both cementum and dentin (macroscopically)
Root transparency	Is best seen on ground section of tooth

* Cultural, dietary, pathological and traumatic factors affect the occlusal surface wear pattern[44]

Table 11.7: Gustafson's ranking of structural changes in age determination

Changes	*Ranking and details*
A = Attrition	A-0 No Attrition A-1 Attrition lying within enamel A-2 Attrition reaching the dentin A-3 Attrition reaching the pulp
P = Periodontosis	P-0 No Periodontosis P-1 Periodontosis just begun P-2 Periodontosis along first 1/3rd of root P-3 Periodontosis along 2/3rd of root
C = Cementum apposition	C-0 Normal C-1 A layer little greater than normal C-2 A great layer C-3 A heavy layer
R = Root resorption	R-0 No visible restoration R-1 Resorption only on small isolated spots R-2 Greater loss of substance R-3 More cementum and dentin affected
T = Root transparency	T-0 Transparency not present T-1 Transparency just noticeable T-2 Transparency over apical 1/3rd of root T-3 Transparency over apical 2/3rd of root



Figs 11.4B to D: Age by cross striations of incremental lines of tooth enamel: (B) Mandible showing the location of the right incisor tooth enamel (smaller white box) sampled. (C) Close-up of enamel fragment with the area of interest, shown in second bigger white box. (D) Image showing growth lines (white arrows) with 10 daily lines between them (white brackets). The scale bar is 200 microns, or 0.2 mm. *Source:* http://www.esrf.eu/news/pressreleases/homo/ Retrieved on 21.07.2009

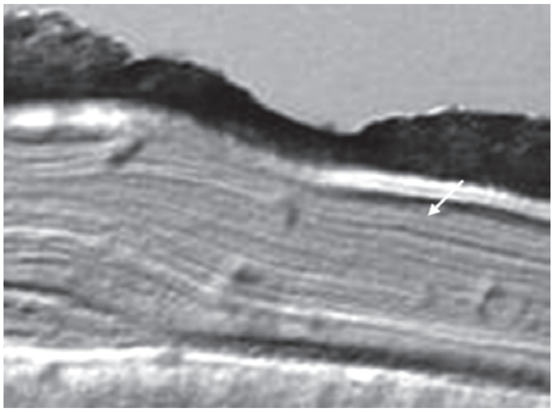


Fig. 11.4E: Transverse section of premolar tooth root with alternate light and dark bands of cemental annulations (Arrow)

Table 11.8A: Bones of the Skull and their number

Skull Bones of Calvaria	*Number*	*Total*
Bones of calvaria		
Parietal bone	02	
Frontal bone	01	
Temporal bone	02	
Occipital bone	01	08
Sphenoidal bone	01	
Ethmoidal bone	01	
Bones of face		
Maxilla	02	
Zygoma	02	
Nasal	02	
Lacrimal	02	14
Palatine	02	
Inferior nasal concha	02	
Mandible	01	
Vomer	01	

All the above bones are kept in position together by the *flexible cartilaginous joints* in early life. These are replaced by *interlocking connections* between bones on *maturity*.

Time of Fusion of Sutures (Table 11.9)[1,3,5,6,11,20-22] (Refer Fig. 11.7A)

Lateral and *occipital fontanelle* usually close within the first two months of birth. The *anterior fontanella* along with *two halves of mandible* closes at the second year. The condylar portion of occipital bone fuses with the squama at the third year and with the basi-occipital at the fifth year. The metopic suture closes at about the third year, but in 5-10 per cent cases it persists and the condition is called *metopism* (Refer Fig. 11.7B). The basi-occipit fuses with the basi-sphenoid at about 18-21 years. In the vault of the skull, closure of the sutures begins on the inner side 5-10 years earlier than on the outer side. The coronal, sagittal, and lambdoid sutures start to close on their inner side at the age of about 25 years. On the outer side, fusion occurs in the following order: posterior one-third of sagittal suture at about 30-40 years; anterior one-third of the sagittal and lower half of the coronal at about 40-50 years; and middle sagittal and upper half of coronal at about 50-60 years. The lambdoid suture starts closing near the lambda and the union is often completed at about 45 years. The squamous part of temporal bone usually fuses with neighbor by the age of 60 years. *Suture closure in skull occurs later in females than in males.* However, estimation of age of skull from suture closure is *not reliable* as it can estimate age in a *range of decade*.

Beginning union in the vault sutures may be identified by irregular radio-opacity on each side of the suture. For age estimation *sagittal suture* is the most reliable, followed by *lambdoid* and *coronal* sutures in order of frequency. A lateral head X-ray film is preferable for the study of coronal and lambdoid sutures. Ectocranial suture closure is very variable. Sometimes, there may not be ectocranial suture closure. This is called *lapsed union*. This occurs most often in the sagittal suture. With lapsing there is slight bony elevation on either

Table 11.8B: Other criteria of assessing the age of the skull

Criteria observed	*Ages*
Both inner and outer surfaces	
— Smooth and ivorine	Young adult age
— Matted granular appearance	40+/- 5 years
Muscular markings become evident on	
— Sides of skull: Along the temporal line	25 years onwards
— On the occiput: Along nuchal line	25 years onwards
— On the lateral side of mandible: Along the mesenteric attachment	25 years onwards
Inner surface of skull: On either sides of the sagittal suture:	
Pits/depressions called	
— Pacchionian depressions develop and become more deeper and more in number	As age advances
— Middle meningeal artery groove becomes deeper	As age advances
Skull diploe: Becomes less vascularly channeled and increase with bone replacement.	> 50 years

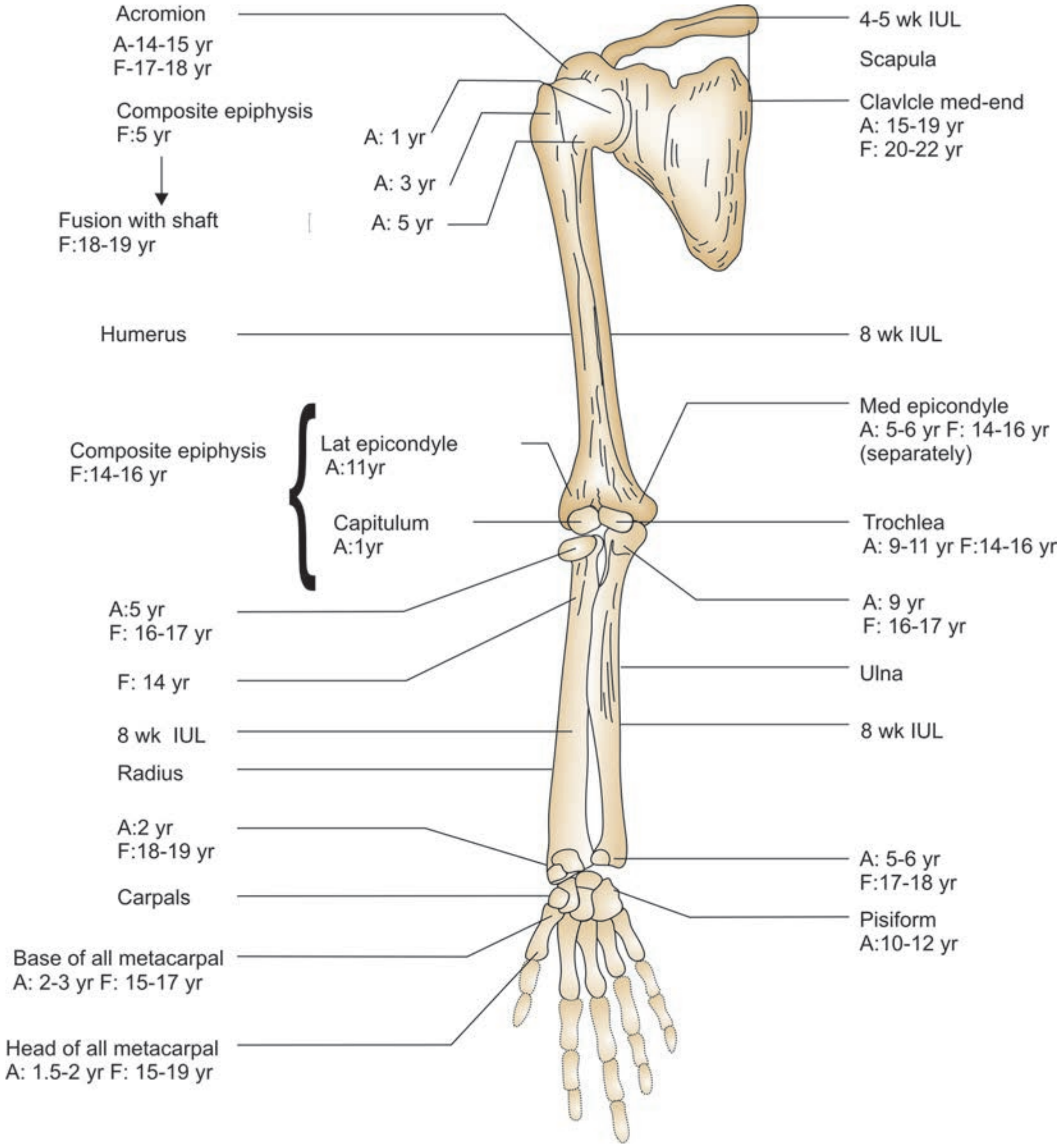

Acromion
A-14-15 yr
F-17-18 yr
Composite epiphysis
F:5 yr
Fusion with shaft
F:18-19 yr
A: 1 yr
A: 3 yr
A: 5 yr
4-5 wk IUL
Scapula
Clavicle med-end
A: 15-19 yr
F: 20-22 yr
Humerus
8 wk IUL
Composite epiphysis
F:14-16 yr
Lat epicondyle
A:11yr
Capitulum
A:1yr
Med epicondyle
A: 5-6 yr F: 14-16 yr
(separately)
Trochlea
A: 9-11 yr F:14-16 yr
A:5 yr
F: 16-17 yr
A: 9 yr
F: 16-17 yr
F: 14 yr
Ulna
8 wk IUL
Radius
8 wk IUL
A:2 yr
F:18-19 yr
A: 5-6 yr
F:17-18 yr
Carpals
Pisiform
A:10-12 yr
Base of all metacarpal
A: 2-3 yr F: 15-17 yr
Head of all metacarpal
A: 1.5-2 yr F: 15-19 yr

Fig. 11.5: Scheme of ossification of bones of upper extremity[11]

Table 11.9: Age by closure of skull fontanelle and sutures

Fontanel and Sutures	*Closing/Fusion**
Anterior fontanelle	2nd year
Mandible two halves	2nd year
Condylar portion of occipital bone with squamous	3rd year
Condylar portion of occipital bone with basi-occiput	5th year
Lateral fontanelle	2nd month
Posterior fontanelle	2nd month/at birth?
Metopic suture	2nd-3rd year
Basiocciput-Basisphenoid	18 - 21 years
Coronal, sagittal, lambdoid begins fusion on inner side of vault	25 years
Outer side of vault – Posterior 1/3rd sagittal	30 - 40 years
– Anterior 1/3rd sagittal and lower 1/2 of coronal	40 - 50 years
– Middle 1/3rd sagittal and upper 1/2 of coronal	50 - 60 years
Lambdoid suture – begins closing at lambda and completes by	45 years
Squamous part of temporal fuses with its neighbour by	60 years

* *The suture closure on outer aspect of the skull takes place 10 years after closure in the inner aspect*

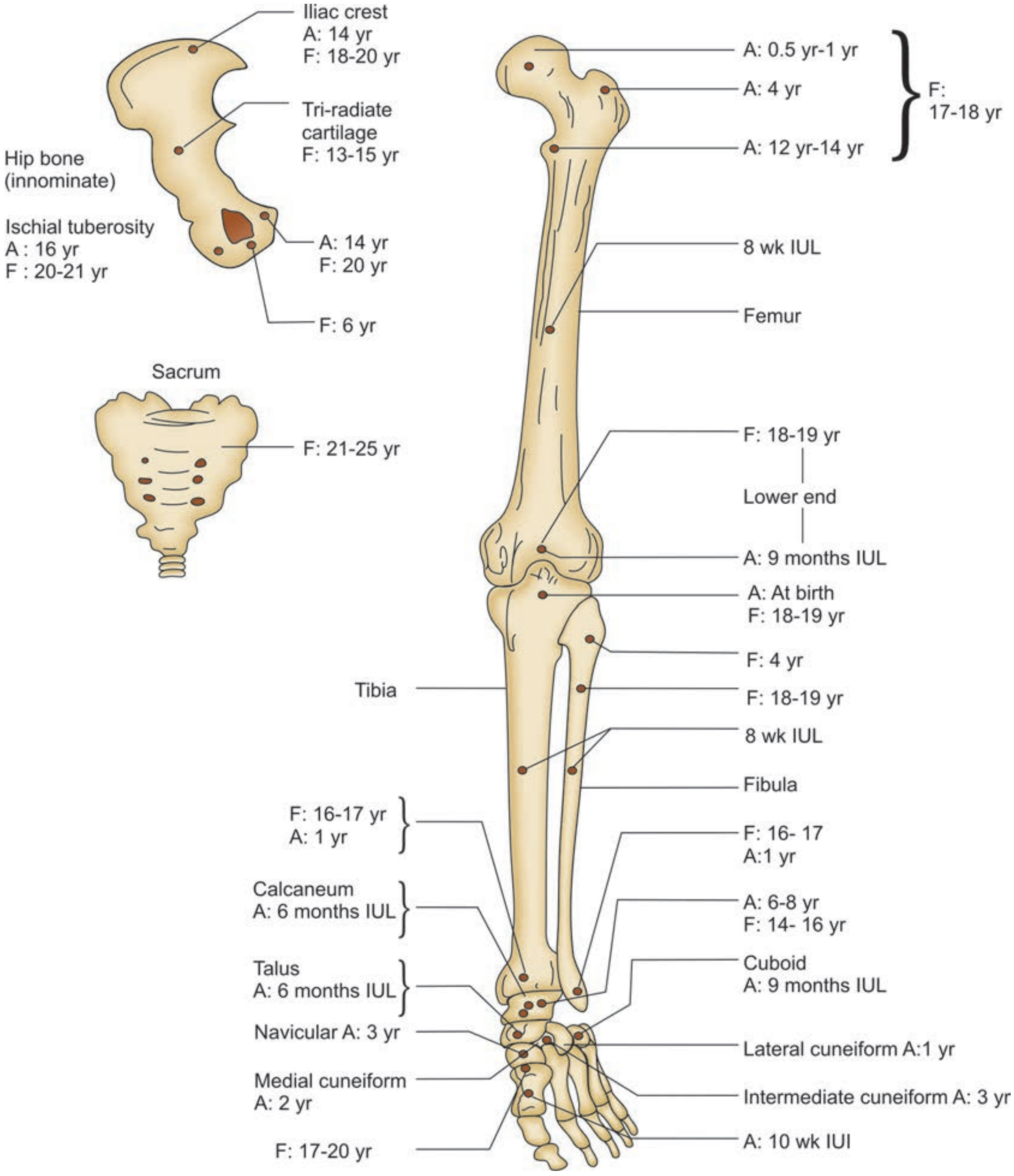

Iliac crest
A: 14 yr
F: 18-20 yr
Tri-radiate
cartilage
F: 13-15 yr
Hip bone
(innominate)
Ischial tuberosity
A : 16 yr
F : 20-21 yr
A: 14 yr
F: 20 yr
F: 6 yr
Sacrum
F: 21-25 yr
A: 0.5 yr-1 yr
A: 4 yr
A: 12 yr-14 yr
F:
17-18 yr
8 wk IUL
Femur
F: 18-19 yr
Lower end
A: 9 months IUL
A: At birth
F: 18-19 yr
F: 4 yr
F: 18-19 yr
8 wk IUL
Fibula
Tibia
F: 16-17 yr
A: 1 yr
F: 16- 17
A:1 yr
Calcaneum
A: 6 months IUL
A: 6-8 yr
F: 14- 16 yr
Talus
A: 6 months IUL
Cuboid
A: 9 months IUL
Navicular A: 3 yr
Lateral cuneiform A:1 yr
Medial cuneiform
A: 2 yr
Intermediate cuneiform A: 3 yr
F: 17-20 yr
A: 10 wk IUI

Fig. 11.6: Scheme of ossification of bones of lower extremity[11]

Table 11.10: Total number of bones in an adult human skeleton

Skeletal structure	*No. of bones*
Skull with mandible	29
Vertebral column (five sacral vertebrae fused together are taken as one and the three coccyx pieces are taken as one)	26
Thoracic cage	25
Upper limb	64
Lower limb	62
Total	206

Table 11.11: Ossification of sternum

Part of sternum	*Appearance*	*Fusion*			
Manubrium sternum	6 months IUL*		50 yr		
1st piece of sternum	6 months IUL	20-25 yr			
2nd piece of sternum	7 months IUL		14 yr		Old age
3rd piece of sternum	7 months IUL	At Puberty		40 yr	
4th piece of sternum	10 months IUL				
Xiphisternum	3 years age				

* *IUL – Intrauterine Life*

side of the incompletely closed suture. Apart from this, there are other approved methods of establishing age of the skull presented in Table 11.8B and Figures 11.7A, 11.10A to C and 11.11A to C.

Ossification of Bones[1-6,19-25,26]

Human skeleton is comprised of 206 bones (Table 11.10). A detailed knowledge of *ossification of bones*, i.e. appearance and fusion of ossification centres can help in determination of the age of a person. Figures 11.5 to 11.18 and Tables 11.10 to 11.12, provide the *scheme of ossification* in human skeleton. Determination of age by skeletal changes constitutes the subject subdivision–*forensic osteology*. This is often useful when skeletal remains are recovered and partial/complete identity of the deceased has to be established from these remains.

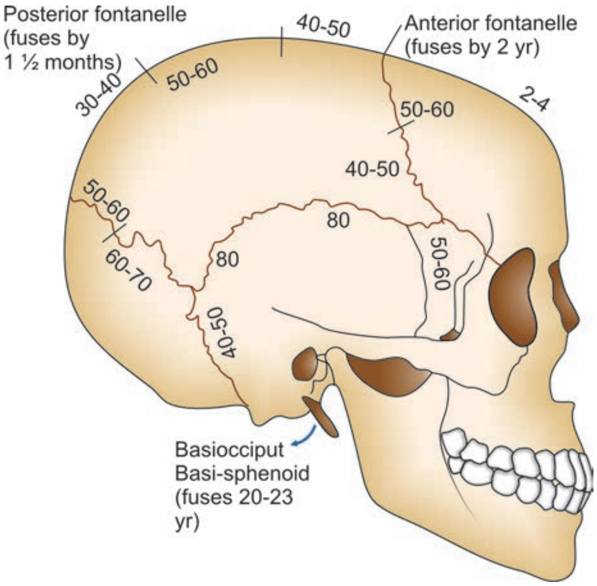

Posterior fontanelle (fuses by 1 ½ months)
40-50
Anterior fontanelle (fuses by 2 yr)
30-40
50-60
2-4
50-60
40-50
50-60
80
50-60
80
60-70
40-50
Basiocciput Basi-sphenoid (fuses 20-23 yr)

Fig. 11.7A: Scheme of ossification of skull by suture closure (Inner aspect)

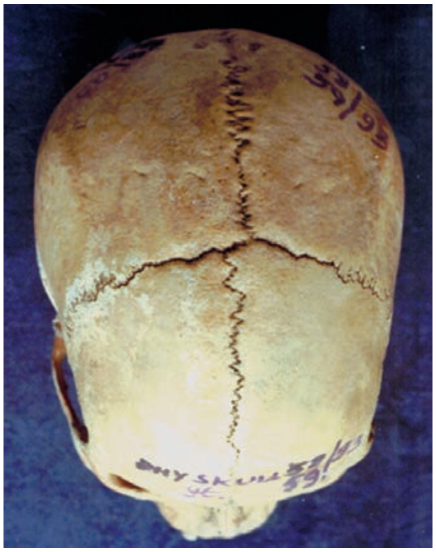


Fig. 11.7B: Metopism. Note: metopic suture not fused (*Courtesy:* Capt. Dr B Santhakumar, Professor and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai, TN)

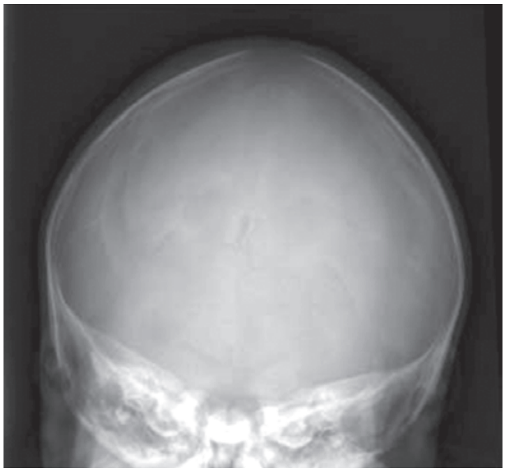


Fig. 11.7C: Radiograph (Towne's view) showing craniostenosis—all skull sutures fused

Table 11.12: Age by changes in articular surface of the pubic symphysis (*Refer Fig. 11.9*)

Changes noticed	*Age*
Markedly irregular and uneven with transverse ridges	20 yr
Ridges disappear, resulting in granularity and formation of the ventral and dorsal margins	24-38 yr
Symphyseal face becomes oval and smoothening of its upper and lower extremities	50 yr
Beaded rimmed margin	5th decade
Erosion of surface and break down of ventral margin	6th decade
Irregularly eroded surface	7th decade

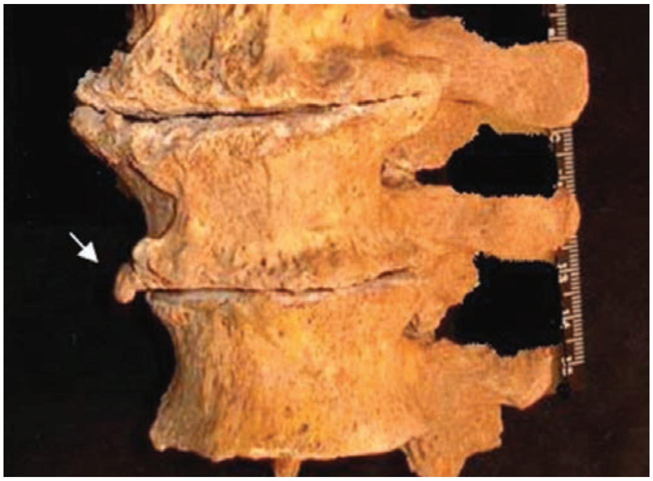


Fig. 11.7D: Lipping of lumbar vertebrae

- Union of Xiphoid process with body of sternum—*after 40 years* (Table 11.11).
- Lipping of lumbar vertebra or bones of the joints of the extremities—*after 45 years* (Fig. 11.7D).
- Union of greater cornu of hyoid bone with body *(40-60 years)*.
- Rarefaction of bone i.e. *senile osteoporosis—after 60 years*
- Age by changes in the articular surface of the pubic symphysis (Table 11.12 and Fig. 11.9).
- Calcification of costal (30 years) and laryngeal cartilage (50±12.7 years).[26]

  However, it may be noted that all these methods are not very reliable.

Confirmation of Ossification Pattern

A radiograph best confirms ossification pattern or union of ossification centres in a bone. Hence, always take an X-ray picture for confirming the age. The subject specialty dealing with this constitutes *forensic radiology.* The ideal site recommended/suggested for ascertaining the various ages of medicolegal importance are enumerated in Table 11.13 and illustrated in Figures 11.10 to 11.18. However, it may be remembered here that extent of ossification of bones varies, depending upon diet, hereditary and geographic factors.[6]

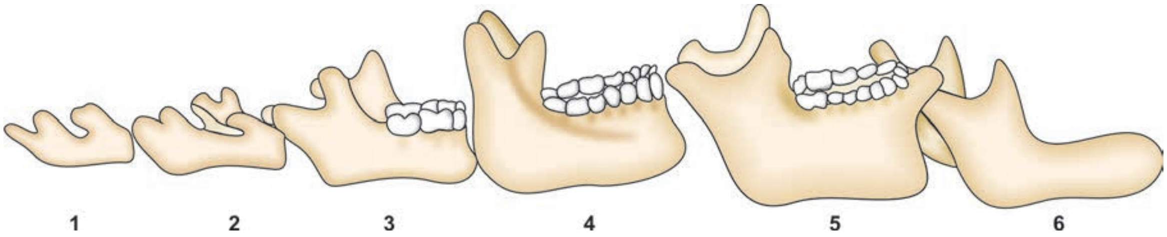


Fig. 11.8: Age changes in the mandible

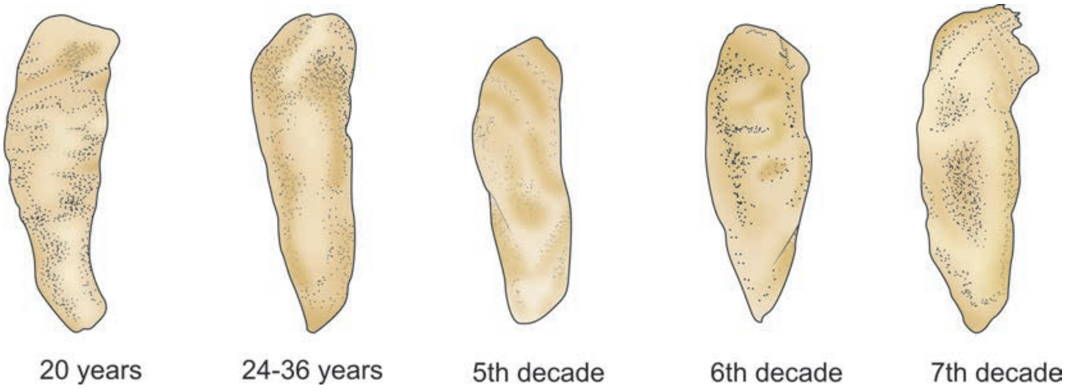


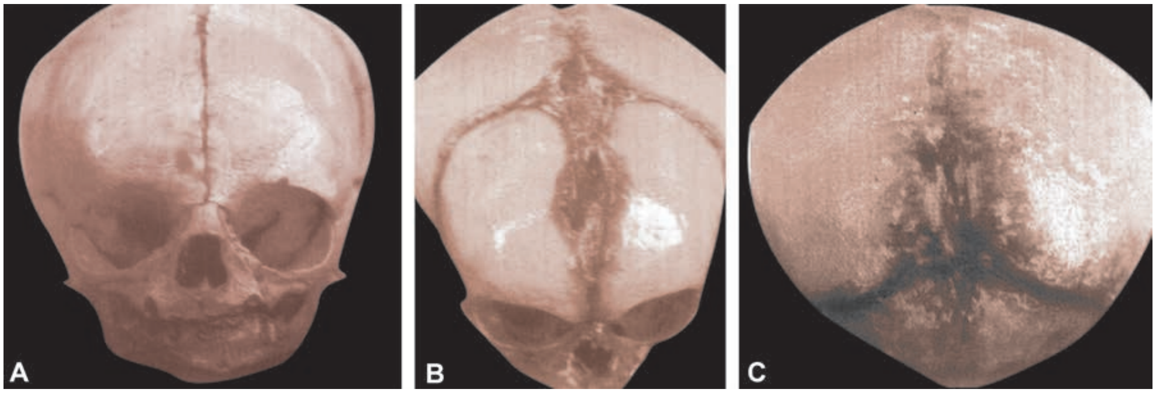
Fig. 11.9: Age changes of symphysis pubis (Refer Table 11.12)

Skeletal and Dental Changes Occurring in Advanced Old Age[3,11,26]

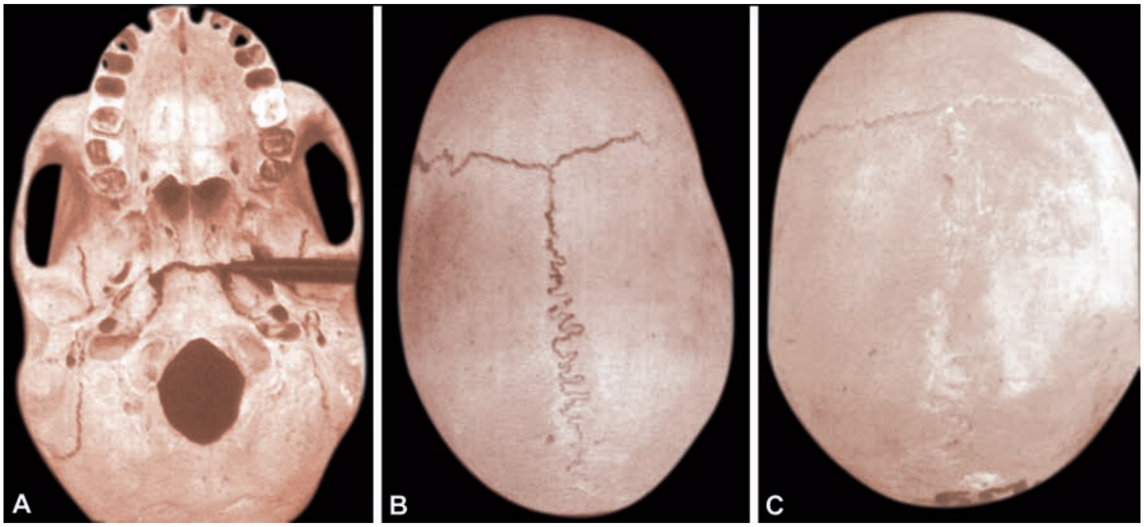
- Disappearance of skull sutures—*after 60 years* (Fig. 11.7C and Table 11.9).
- Attrition or loss of teeth—*after 50 years.*

Table 11.13: Recommended radiograph/X-ray for different ages of medicolegal importance

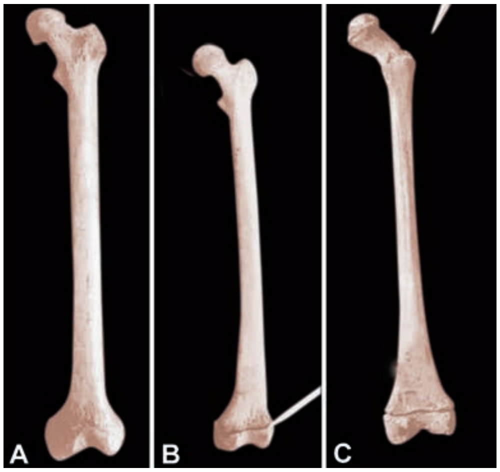
Age group	*Radiograph/X-ray recommended*
Infancy and childhood	Wrist and elbow
Adolescence age	Pelvis, hip and shoulder
Adulthood	Elbow and knee
Old age	Skull
All age groups	Pelvis, hip



Figs 11.10A to C: *Foetal skull:* (A) Frontal suture not fused, (B) Anterior fontanelle not closed, (C) Posterior fontanelle not closed



Figs 11.11A to C: *Adult skull:* (A) Basiocciput—basi-sphenoidal suture not fused, (B) Coronal and sagittal sutures—is not fused, (C) Sagittal suture—posterior 2/3rd fused



Figs 11.12A to C: *Femur:* (A) All centres fused, (B) Lower end not fused, (C) Upper end greater and lesser trochanter centres not fused (missing) and lower end not fused

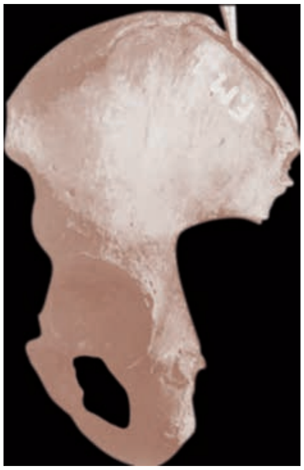


Fig. 11.13: Iliac crest—not fused

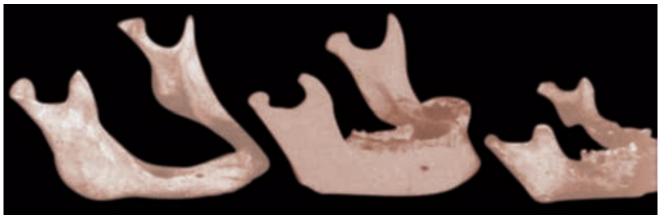
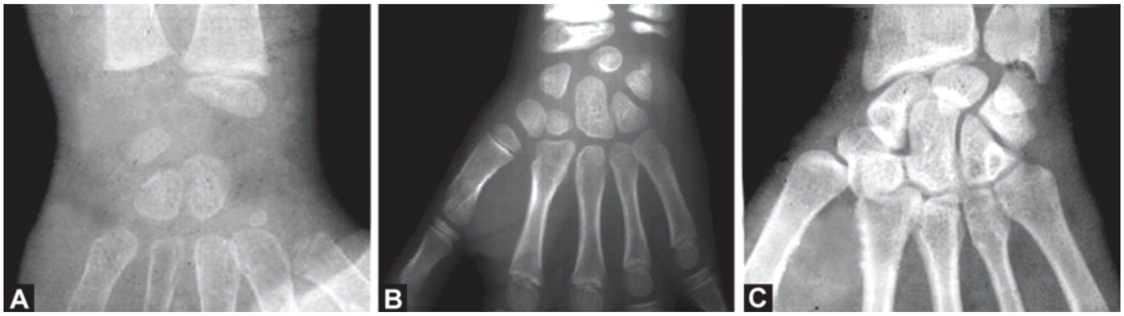
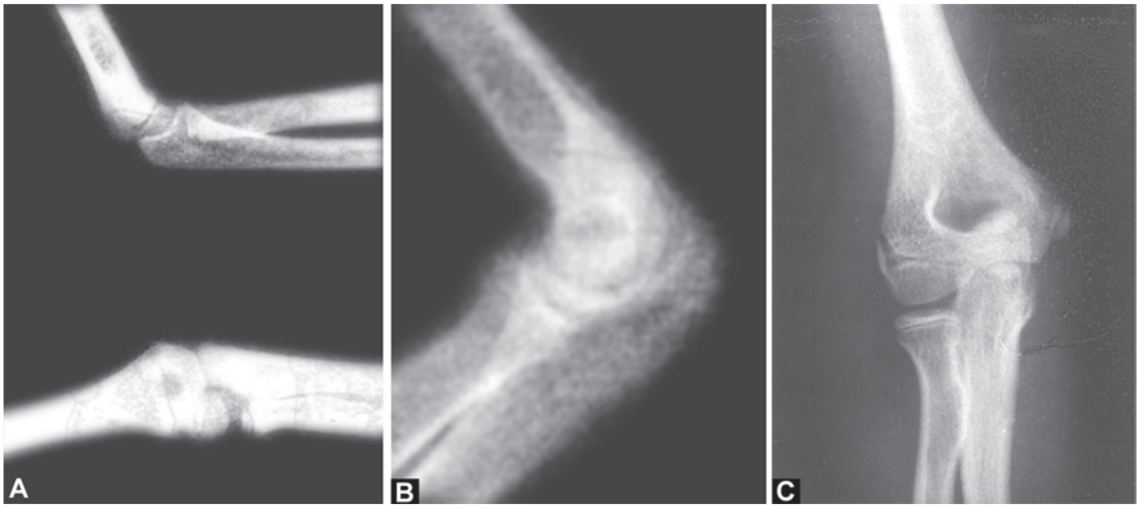


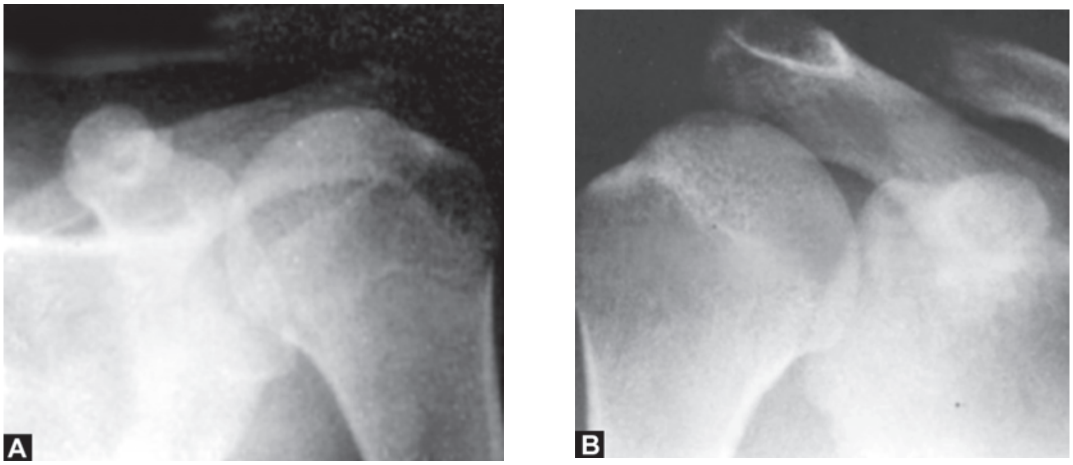
Fig. 11.14: Mandible—age changes



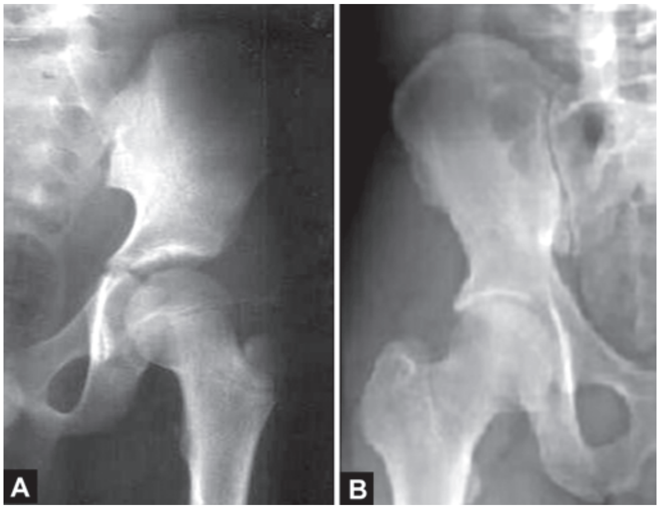
Figs 11.15A to C: *AP views of wrists of age groups:* (A) 2-6 years (Reasons—Lower end of radius appeared: >2 years; Lower end of ulna not appeared: <6 years); (B) 10-17 years (Reasons—Pisiform appeared: >10 years; Base of first metacarpal not fused <17 years). (C) More than 18 years (Reason—Lower end of radius fused)



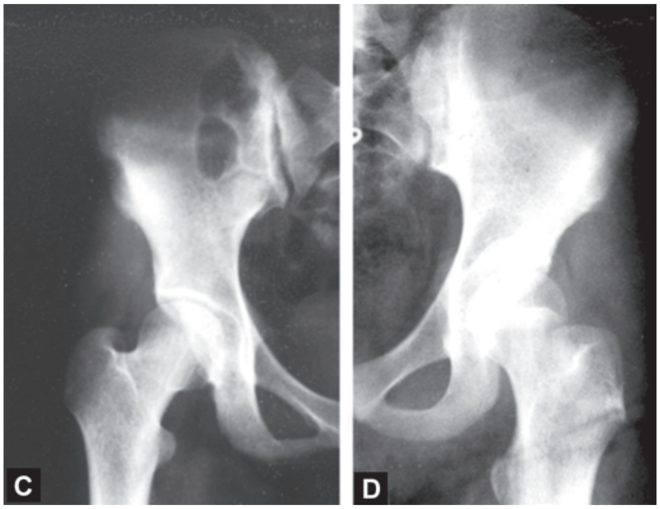
Figs 11.16A to C: *AP veiw of elbows of age groups:* (A) 5-9 years (Reasons: Centre for upper end of radius appeared: >5 years; centre for upper end of ulna not appeared: <9 years); (B) 9-17 years (Reasons: Centre for upper end of ulna appeared: > 9 years; Centres for upper ends of both radius and ulna not fused: <17 years); (C) < 16 years (Reasons: Composite epiphysis for lower end of humerus not formed: <16 years; Centre for lateral epicondyle appeared: >11 years)



Figs 11.17A and B: *AP view of shoulders of age groups:* (A) 14-18 years (Reasons: Centre for acromion appeared: >14 years but not fused: <18 years; (B) Just 18 years (Reasons: Epiphyseal scar formed for the upper end of humerus



Figs 11.18A and B: *AP view of hip joint of age groups:* (A) 12-14 years (Reason: Centre for lesser trochanter appeared: >12 years; Centre for iliac crest not appeared: <14 years) (B) 14-16 years (Reasons: Centre for iliac crest appeared: >14 years; Centre for Ischial tuberosity not appeared: <16 years)



Figs 11.18C and D: (C) 17-20 years, (Reasons: Upper end of femur fused: >17 years; iliac crest not fused: <20 years) (D) More than 20 years (Reasons: Centre for ischial tuberosity fused: >20 years)

Changes in Sacrum and Vertebrae Helpful in Determining the Age

Sacrum

The five sacral vertebrae are separated by cartilage until puberty. Later, fusion of epiphyses takes place and ossification of intervertebral discs extend from below upwards. Sacrum becomes single bone at 21-25 years of age, leaving a gap between S1 and S2, until 32 years due to 'lapsed union'.

Vertebrae

The immature vertebral body has series of deep radial furrows both on upper and lower surfaces. The feature increases in prominence up to the age of ten, and then gradually fades between 21 and 25 years. Later on, due to osteo-arthritic changes in the form of *lipping* of vertebra are seen after the age of 45 years.

HEIGHT AND WEIGHT DATA

Height and weight data are quite useful in establishing age of a person. Table 11.14 presents these facts clearly.[7] This table is rather often useful in determining the *normal height* and *weight* range for a given age.

Miscellaneous Data

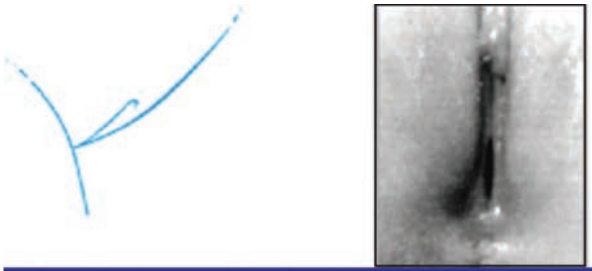
Various other data that may be worth mentioning and beneficial in determining the age of an individual are enumerated below:

Table 11.14: Age by height and weight data (Standard heights and weights for men and women—by Life Insurance Corporation of India)

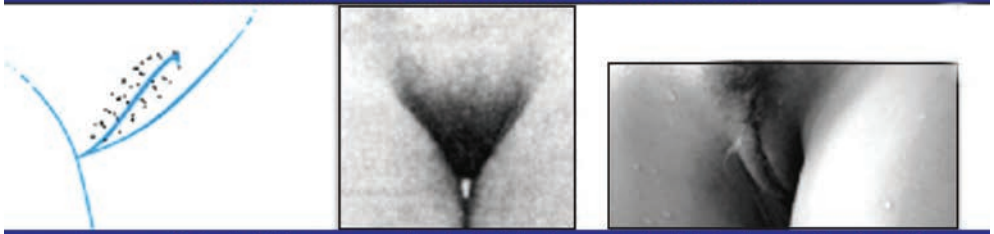
Height		*Men (Weight)*		*Women (Weight)*	
Metres	*0’. 0”*	*kg*	*lb*	*kg*	*lb*
1.523	5’ 0”	….	….	50.8 – 54.4	112 – 120
1.5484	5’ 1”	….	….	51.7 – 55.3	114 – 126
1.5738	5’ 2”	56.3 – 60.3	124 – 133	53.1 – 56.7	117 – 125
1.5992	5’ 3”	57.6 – 61.7	127 – 136	54.4 – 58.1	120 – 128
1.6246	5’ 4”	58.9 – 63.5	130 – 140	56.3 – 59.9	124 – 132
1.650	5’ 5”	60.8 – 65.3	134 – 144	57.6 – 61.2	127 – 135
1.6754	5’ 6”	62.2 – 66.7	137 – 147	58.9 – 63.5	130 – 140
1.7008	5’ 7”	64.0 – 68.5	141 – 151	60.8 – 65.3	134 – 144
1.7262	5’ 8”	65.8 – 70.8	145 – 156	62.2 – 66.7	137 – 147
1.7516	5’ 9”	67.6 – 72.6	149 – 160	64.0 – 68.5	141 – 151
1.7770	5’10”	69.4 – 74.4	153 – 163	65.8 – 70.3	145 – 155
1.802	5’11”	71.2 – 76.2	157 – 168	67.1 – 71.7	148 – 158
1.8278	6’ 0”	73.0 – 78.5	161 – 173	68.5 – 73.9	151 – 163
1.8532	6’ 1”	75.3 – 80.7	166 – 178	…..	…..
1.8786	6’ 2”	77.6 – 83.5	171 – 184	….	…..
1.9040	6’ 3”	79.8 – 85.9	176 – 189	….	…..

Note: Maximum weight that one may reach: Up to the age of 30 years, 10% above standard, between 30 to 35 years, standard is optimum weight, above 35 years, 10% below standard

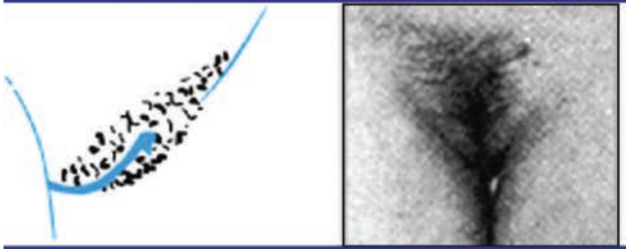
- ***Pubic hair:*** They appear at the age of about *13 years in female* and at the age of *14 years in male*. They are *sparse, soft and light in colour* initially and later on turn *thick, bushy* and *dark* within 2 years of appearance with the onset of *puberty* (Fig. 11.19A).
- ***Axillary hair:*** They appear at the age of about *14 years in female* and at the age of *15 years in male.*
- ***Beard and moustache/facial hair:*** They appear *only in male* by *16 to 18 years.*
- ***Breasts:*** Begin to appear by *13 to 14 years in females only* (Fig. 11.19B).
- ***Voice:*** It become *hoarse in males by 16 to 18 years.*
- ***Scalp hair:*** Begins to *turn gray by 40 years*. Also becomes *less dense as the age advances.*
- ***Axillary and pubic hair:*** Turn *gray* only at advanced *old age*.
- ***Arcus senilis:*** This is a whitish ring that makes its appearance in the periphery of the cornea of the eyes due



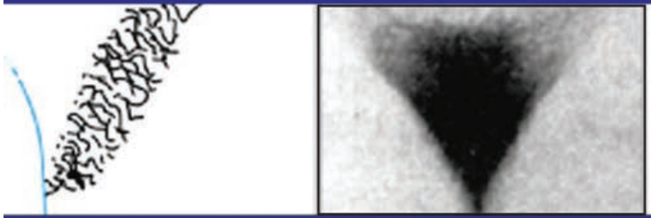
< 12 years (Pre-pubertal): No pubic hairs. Vulva shows flattened labia majora and thin attenuated labia minora which do not meet in the midline



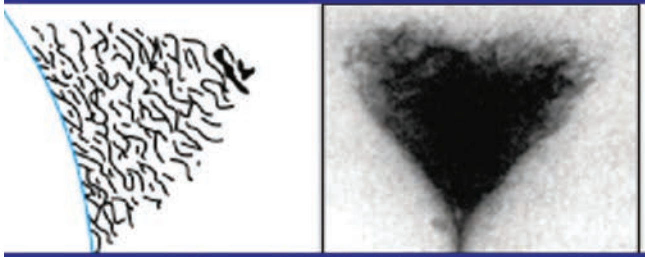
12 years (Pubertal): Sparse growth of slightly pigmented pubic hairs chiefly along the medial border of labia majora, that are now developed and meeting in the midline



12-13 years: Hairs turn darker, increase in amount, begins to curl and spreads over the mons



13-15 years: Increased amount of hairs, which are coarse and curly on the mons



> 15 years: Adult feminine inverse triangular distribution, spreading to the medial surface of thigh

Fig. 11.19A: Secondary sexual characteristics pertaining to vulva and pubic hairs

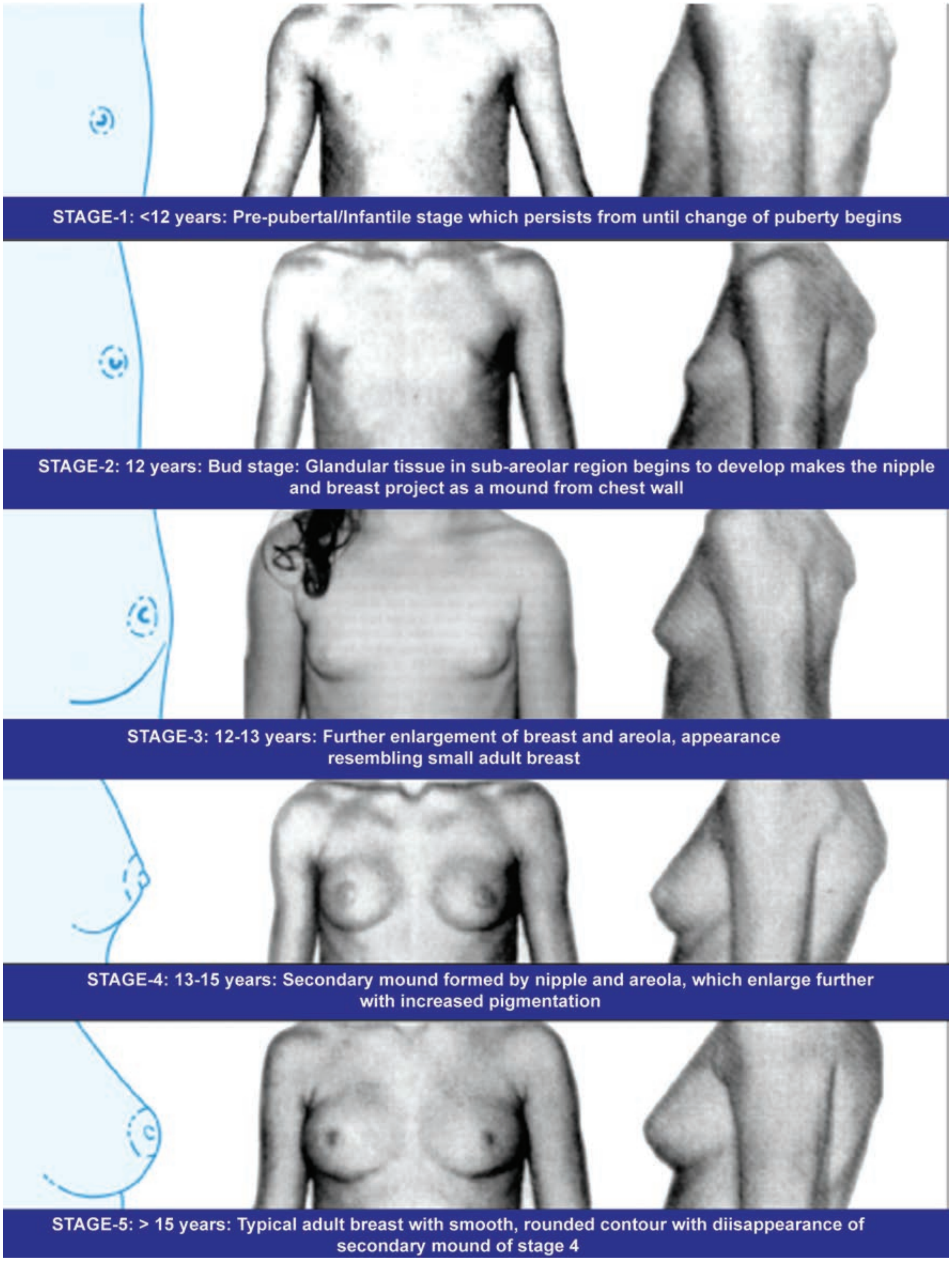
to the degenerative changes and *begins at about the age of 40 years*, is a *normal process of aging* (Figs 11.19C and D).

- ***Cataract (opacity of lense) in the eyes:*** It is seen in *old age (>60s)*.
- ***Wrinkles over the face:*** Are seen in *old age (>60s)*.

MEDICOLEGAL IMPORTANCE OF AGE[1-6,11,23-24,28-33]

Medicolegal importance of age is multifaceted. Enumerated below are certain important ones, which a doctor has to be aware of:

- *In identification of the individual:* Unless the age of a person is determined the identity of the person live or dead stands incomplete.
- *Embryo and pregnancy:* On 7th day of fertilisation, ovum getting impregnated in the uterus, is called as "embryo" and the woman is said to be "pregnant".
- *Foetus and foeticide:* From 2nd lunar month (or 8th week IUL), embryo is termed as "foetus" and killing of a foetus is (foeticide).



STAGE-1: <12 years: Pre-pubertal/Infantile stage which persists from until change of puberty begins

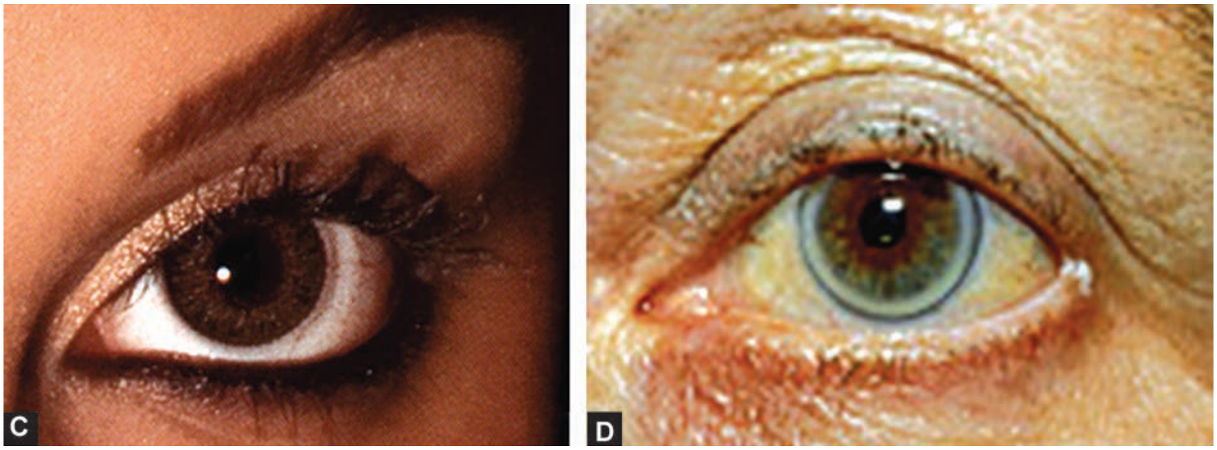
STAGE-2: 12 years: Bud stage: Glandular tissue in sub-areolar region begins to develop makes the nipple and breast project as a mound from chest wall

STAGE-3: 12-13 years: Further enlargement of breast and areola, appearance resembling small adult breast

STAGE-4: 13-15 years: Secondary mound formed by nipple and areola, which enlarge further with increased pigmentation

STAGE-5: > 15 years: Typical adult breast with smooth, rounded contour with diisappearance of secondary mound of stage 4

Fig. 11.19B: Secondary sexual characteristics pertaining to the breast



Figs 11.19C and D: (C) Normal eye (D) Eye with arcus senilis

- *Viable child (Sec 300 IPC):* After 7 calendar months (210 days) of pregnancy, fetus is called as "viable child" as it can live independently if born and killing of a viable child amounts to the offence of "infanticide".
- *Full-term foetus:* Foetus after 10 lunar months of pregnancy is called as "full-term foetus".
- *Medical termination of pregnancy (MTP)-induction:* Inducing MTP, decision by one doctor is allowed up to 3 lunar months (12 weeks IUL) of pregnancy and beyond this period decision is always by two doctors (MTP Act, 1976). However, beyond 5 lunar months (20 weeks IUL) of pregnancy, MTP is not done, unless otherwise mother (pregnant woman) is gravely ill.
- *Still-born:* Foetus after 28 weeks of IUL if shows no signs of life or any breathing is called as "still-born".
- *Criminal responsibility:* In law the crime and punishment is entirely based on criminal responsibility, and this in turn is dependent on age of a person and may be highlighted as follows briefly:
  - Under *Section 82 IPC,* law presumes that *a child below 7 years is incapable of committing any crime.*[27,47]
  - Under *Section 83 IPC,* however, children *between ages of 7 and 12 years* will be guilty of offence and held *responsible for the offenses charged*, only if the trying court is satisfied that the child has attained *sufficient degree of mental maturity of understanding to judge the nature of consequences of their acts on that occasion and unless proved to the contrary.*[27,47]
  - Under *Section 127-130 of Indian Railway Act, 1890*, children above *5 years* of age are *held liable for punishment* if he/she does *anything maliciously to wreck or attempt to wreck a train and passengers in it or to railway property.*[30,49]
  - However, all child offenders above the age of 12 years but below 18 years are called juvenile offenders and tried in a juvenile court and are sent to *Borstal* or juvenile reformatory schools and not to a jail *(Juvenile Justice Act, 1986)*.[50]
- *Infanticide*: Deliberate and unlawful killing of a newborn or a child below the age of 1 year by the act of omission or commission is considered as infanticide. Infant is any live born child up to the age of one year after birth and infanticide is regarded as murder in the court of law and is punishable under *Section 302 IPC.*[47]
- *Marriage*: Any matrimonial alliance entered into by a boy below 21 years and girl below 18 years will be invalid *(Child Marriage Restraint Act, 1978)*.
- *Kidnapping*: To constitute an offence of kidnapping or abducting a minor from lawful guardianship, the age should be 10 years for the purpose of movable property from the body of the child or in its possession *(Section 361 IPC),* below 16 years for a boy and below 18 years for a girl; inducing any girl of Indian origin below 18 years *(Section 366 IPC)*/below 21 years when imported from Jammu and Kashmir or any outside country for the purpose of illicit sexual intercourse *(Section 366-B IPC)* also constitutes the offence of "*kidnapping*" and is punished by 10 years imprisonment and with/without fine.
- *Rape*: Sexual intercourse with a girl below 16 years, even with her consent, legally constitutes rape *(Statutory rape)*. However, as child marriage is still in prevalence in India, even this day, sexual intercourse by a man with his own wife who is of age <15 years, with/without her consent also constitutes the offence of rape *(Section 375 IPC)*.[47]
- *Majority*: A person attains majority on completion of 18 years. He or she can cast vote in general elections, sell or buy property or make a *valid will* and serve on jury only if sound mentally. However, person attains majority at the age of 21 years, when under the *court guardianship (Section 3, Indian Majority Act, 1875)*.
- *Age and child employment*: A child below 14 years cannot be employed in a factory or in any other hazardous employment *(Indian Factories Act, 1948)*. However, this does not mean a child above 14 year age can be employed in a factory and work like an adult. The act allows a person of age 14-15 years (>15 years) of age to work in a non-hazardous work during day time, provided that he has been certified by a physician to be fit physically.
- *Age and government employment*: In India, minimum age limit for entering into the government services is 18 years while the age of retirement from government service, ranges from 55-58 years.

  Note: However, both may vary depending on the job and the State Government Policies.
- *Consent*: A child below 12 years cannot give consent for physical examination and the valid consent is to be given by the parents/guardian *(Section 89 IPC)*.[47] However, valid consent for suffering any harm not intended or known to

cause death/grievous hurt, for example, major surgical operation, can be given only if he/she is above 18 years of age *(Section 87 IPC)*.[47]

Table 11.15 lists various ages of medicolegal importance in an ascending order of age *(from birth to old age)* for an *easy understanding* and *recollection*.

SEX AND IDENTITY[1-6,8,9,11,22-26]

Sex of person is equally important as the age factor in determining the identity of an individual. Sex of a person alive or dead can be determined by: (i) physical examination, (ii) gonadal biopsy, (iii) sex chromatin, and (iv) other methods.

Physical Examination

This includes traits establishing sex identity of an individual. This constitutes anatomical sex component that determines sex and comprise of external appearances inclusive of external genitalia in male and female and internal genital tract in the female (Figs 11.20A to C).

Table 11.15: Medicolegal importance of age

Age in Years	*Medicolegal Importance*
7 days of fertilisation	Embryo, pregnancy
08 weeks IUL	Foetus
12 weeks IUL	Induction of MTP – one doctor can decide
>12 weeks <20 weeks IUL	Induction of MTP – two doctors should decide
>20 weeks IUL	Induction of MTP – Not allowed unless for serious maternal causes
>28 weeks IUL	Called ‘stillborn’ if no sign of life/breathing
07 months IUL (210 days)	Viable child; if killed – charges of infanticide
10 months (lunar) 140 weeks IUL	Full term foetus
0-1 year	Infanticide
5 years	Indian Railway Act
7 years	Criminal responsibility
10 years	Kidnapping for valuables
12 years	Consent for physical examination
14 years	Factory employment Act
15 years	Consent for sexual intercourse in married woman
16 years	Valid consent for sexual intercourse by a woman (Statutory Rape), Kidnapping charges (Boys), etc
18 years	Consent for major surgery, attaining majority, right to franchise vote, kidnapping charges (Girls), marriage (Girls), minimum age for entering Government service, upper limit for juvenility, etc.
21 years	Attaining majority when under court guardianship, marriage (Boys)
25 years	Minimum age for contesting membership of Indian Parliament/Other legislative bodies
28 years	Age limit for entering in to government services
35 years	Minimum age for appointment as the President (Vice-President), Governor of a State in India
41-45 years	Menopause in a woman usually
56-58 years	Age of retirement from government service, government undertakings, statutory bodies, autonomous bodies/ institutions, higher judiciary services

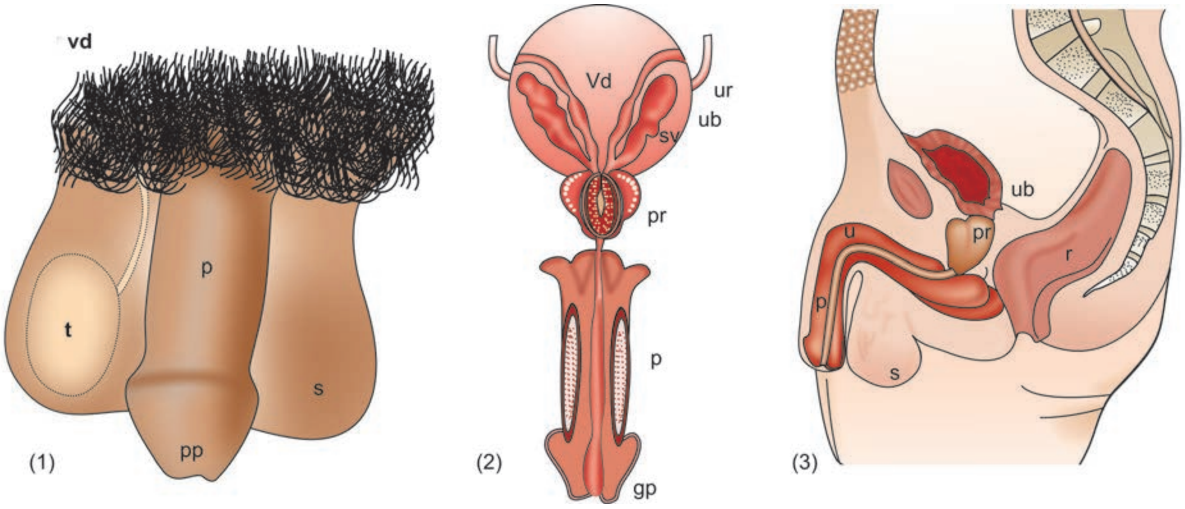
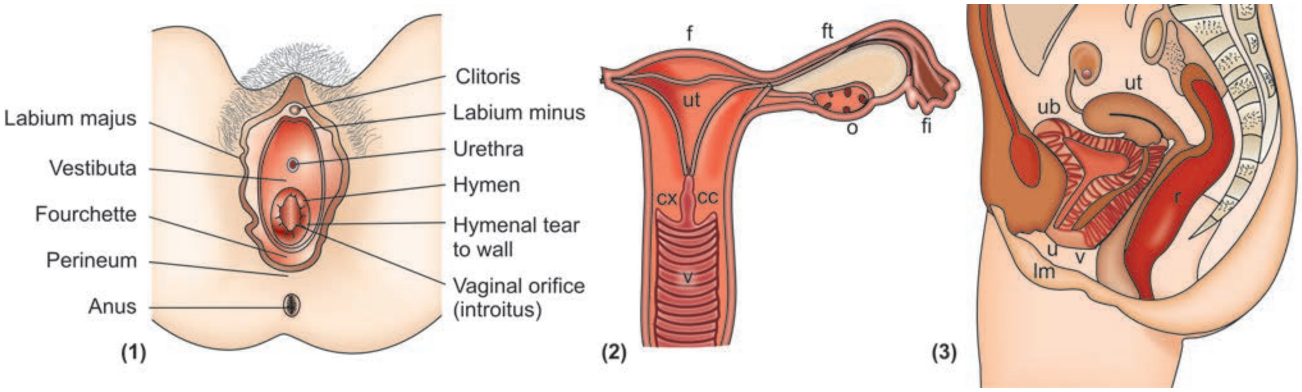


Fig. 11.20A: *Male external and internal genitalia:* (1) Male external genitalia viewed from the front: p—penis, pp—prepuce, s—scrotum, t—testis, vd—vas deferens, (2) Male internal genitalia viewed from the front: gp—glans penis, pr—prostate, sv—seminal vesicle, u—urethra, ub—urinary bladder, ur—ureter, vd—vas deferens, and (3) Midsagittal section of the male pelvis showing external and internal genitalia: p—penis, pr—prostate gland, r—rectum, s—scrotum, u—urethra, uv—urinary bladder


Labium majus
Vestibuta
Fourchette
Perineum
Anus
Clitoris
Labium minus
Urethra
Hymen
Hymenal tear to wall
Vaginal orifice (introitus)
(1)
f
ft
ut
o
fi
cx
cc
v
(2)
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lm
(3)

Fig. 11.20B: *Female external and internal genitalia:* (1) Female external genitalia: c—clitoris, h—hymen, l—labia minora, lm—labia majora, u—urethral meatus, v—vaginal opening, (2) Female internal genitalia viewed from the front: cc—cervical canal, cx—cervix, f—fundus, fi—fimbriae, ft—fallopian tube, o—ovary, v—vagina, and (3) mid-sagittal section of the female pelvis showing external and internal genitalia: lm—labia majora, o—ovary, r—rectum, u—urethral meatus, ub—urinary bladder, ut—uterus, v—vagina

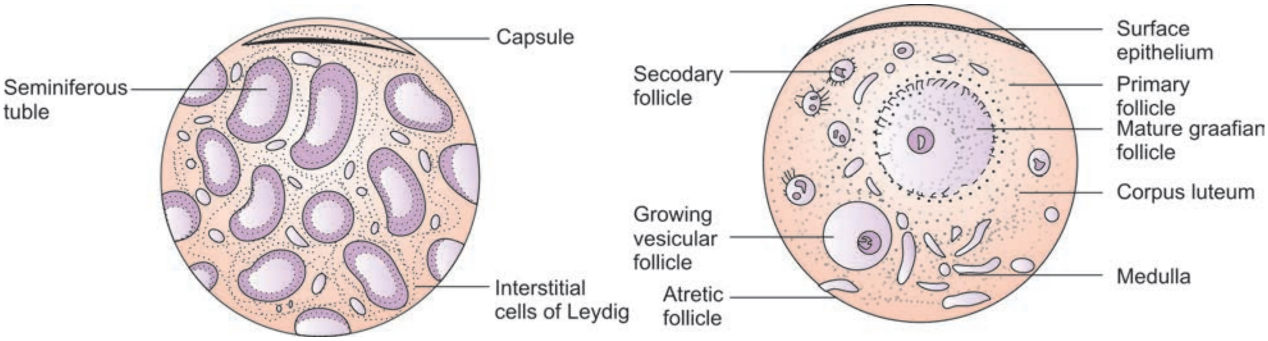

Seminiferous tuble
Capsule
Interstitial cells of Leydig
Secodary follicle
Growing vesicular follicle
Atretic follicle
Surface epithelium
Primary follicle
Mature graafian follicle
Corpus luteum
Medulla

Fig. 11.20C: Histology of testes and ovary

Table 11.16 enumerates the external and internal genital organs in establishing the differences between male and female sex.

Gonadal Biopsy

Gonadal biopsy is a confirmatory method of determining sexual identity histologically (Fig. 11.20C). This constitutes "*gonadal sex*" component in determining sex. In all disputed sexual identity cases, gonadal biopsy is suggested.[20,34]

Table 11.16: Traits establishing sexual identity

Traits	*Male*	*Female*
Testes	Present, functioning	Absent
Penis	Present	Absent
Ovary	Absent	Present
Uterus	Absent	Present
Vagina	Absent	Present
Shoulder	Broader than hips	Narrower than hips
Gluteal region	Flat	Full and rounded
Adam's apple	Prominent	Less prominent
Breasts	Absent	Grows at puberty
Pubic hairs	Thick and extends to umbilicus only	Thin and covers up only the mons pubis

Sex Chromatin (Barr and Davidson Bodies)[8,9,11,35-37]

A chromosome that determines whether an individual is male or female represents sex chromosome. This constitutes *chromosomal sex* or *nuclear sex* component in determining the sex. The sex chromosomes in human beings are designated as *X and Y chromosome.* In humans the sex chromosomes comprise just one pair of the total of 23 pairs of chromosomes. The other 22 pairs of chromosomes are called *autosomes.*

The individual having two X-chromosomes (XX) is female, while individual having one X and one Y-chromosome (XY) is male. The X-chromosome resembles a large autosomal chromosome with a long and a short arm. The Y-chromosome has one long arm and a very short second arm. This change into maleness or femaleness takes place at the moment of meiosis, when cell divides to produce gamete. Thus during meiosis, the male XY sex-chromosome pair separates and passes on a X or a Y to separate gametes, the result is that one-half of the gametes (sperm) that are formed contains the X chromosome and other half contains the Y-chromosome. The female has two X-chromosomes, and all female eggs normally carry a single X. Thus, the eggs with X-chromosome when fertilised by X-chromosome bearing sperm the offspring will become a female with XX-chromosomes, whereas those fertilised by Y-chromosome bearing sperm become males (XY).

The X-chromosome in a female is seen in the form of *chromatin condensation towards the nuclear membrane* microscopically in the nucleus of a cell. This condensed chromatin has been shown to consist of deoxyribose nucleic acid, 1 μ in size and is present in approximately 75 per cent of female cells.

It is called as *sex chromatin* or *Barr body*. Usually, they are better appreciated in the cells of *skin, buccal mucosa, cartilage, nerves, amniotic fluid and lymphocytes*. Barr and Bertram first described this in the nucleus of the nerve cells of female cat, and coined the terminology *Barr body*.[35]

This turned out to be of great significance in identifying the *true sex (nuclear sex)* of a person in case of *intersex* individual with malformed sex organs.[37]

Buccal Smear

From a normal female, *sex chromatin* appears as a *small planoconvex mass*, lying near nuclear membrane (Fig. 11.21A) microscopically. Thus, based on this, to diagnose female sex, the buccal smear must exhibit minimum of 20 to 30 per cent Barr bodies, as against 0 to 4 per cent Barr bodies detected in normal male (Fig. 11.21B).[35]

Vaginal Epethilial Cell (PAP Smear)

Figure 11.21C illustrates Barr body in it is from a normal adult female, is presented here for the purpose of familiarising it to a reader.

Peripheral Blood Smear

Neutrophilic leukocytes in a normal female often presents a similar and distinctive type of nuclear appendage attached to one of the nuclear lobe, resembling a *drumstick* (Fig. 11.22). This is known as *Davidson body*.

However, to diagnose sex as female by this, the peripheral smear examined must show minimum 3 per cent counts.[3,6,38]

Other Methods

Newer methods reported in recent forensic literature are worth mentioning here, adding to the existing list of factors, establishing sex in disputed sex identity cases. Additional factors marking recent advances in sex determination are: (i) costal cartilage calcification pattern,[45,46,57] (ii) footprint ratio,[58] and (iii) mandibular canine index *(MCI)*,[59,60] (iv) cytogenetics and DNA analysis, (v) counting of sex materials within the nucleus.

Costal Cartilage Calcification Pattern

In this methodology radiological appearance of the calcification pattern of costal cartilage (5-12th rib) is considered in determining the sex in age group 16 to 20 years. Three distinct patterns are described under this (Fig. 11.23). This is the first Indian classification pattern ever reported and known as *Rao and Pai's classification of calcification pattern*.[57] Accuracy of the method is >92.3 per cent.

Footprint Ratio (FPR)

This is the ratio between maximum width (MW) and maximum length (ML) of the footprint in millimeters and is reported to be useful in establishing sexual identity of a person alive or dead. Figure 11.24, below gives an idea as to how these parameters are measured and applied in determining the sex.

$$\text{FPR} = \frac{\text{Maximum width of the footprint } (MW)}{\text{Maximum length of the footprint } (ML)}$$

Footprint ratio (FPR) value being derived using this formula is then compared with *standard footprint ratio (SFPR)* value (0.376 for left foot and 0.377 for right foot). Sex is predicted as *female* when the FPR values derived are within the limits of SFPR, and as male when the limits are crossed. Accuracy of the method is 78 per cent.[58]

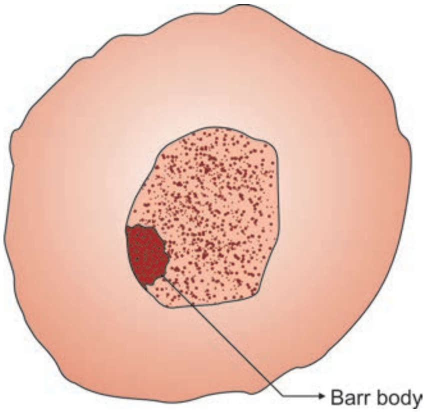


Fig. 11.21A: Barr body in buccal smear

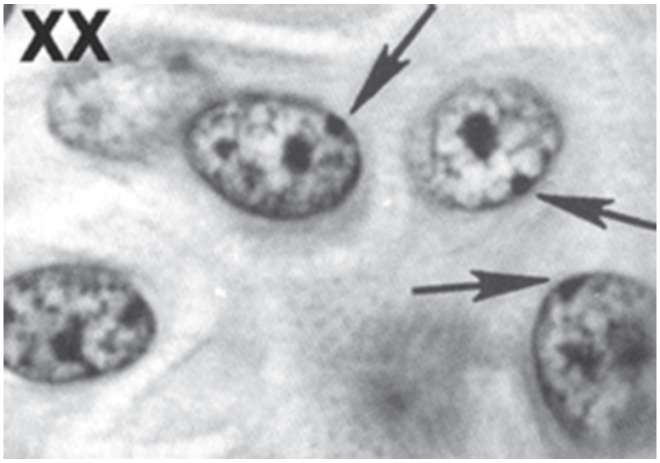


Fig. 11.21B: Planoconvex chromatin mass of Barr bodies (arrows) in buccal smear (40x)

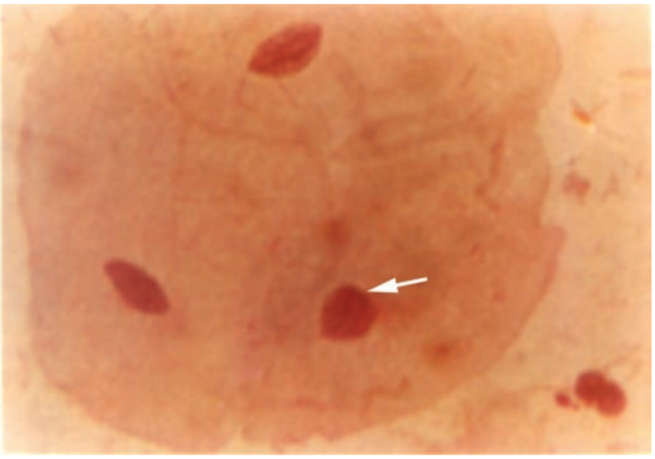


Fig. 11.21C: Vaginal epethilial cell Papanicolaou (PAP) smear–arrow pointing at the bar body (*Courtesy:* Dr Uday Pal Singh, KMC, Warangal, AP)

Mandibular Canine Index

Mandibular canine index (MCI) is again a ratio of maximum width of the *permanent mandibular canine (mesiodistal crown width*) to *arch width*. Figure 11.25 gives an idea as to how these parameters are measured and applied in determining the sex. MCI values being derived thus using this formula are then compared with standard mandibular canine index (SMCI) value

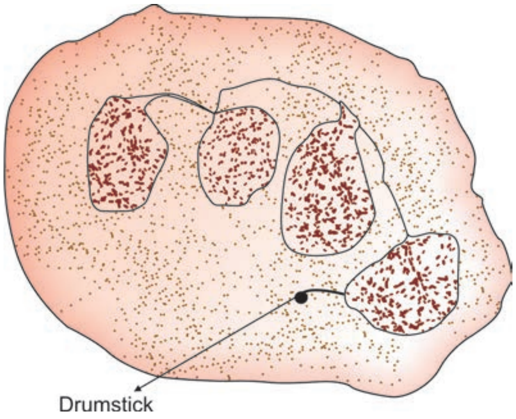
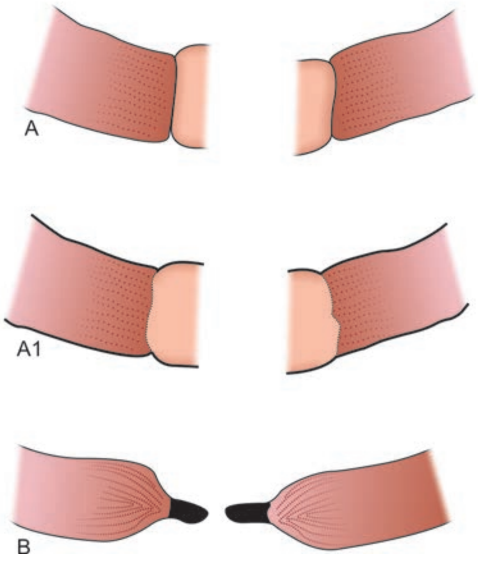


Fig 11.22: Davidson body in peripheral smear



Type A - Marginal square bracket type (Common male type)
Type A1 - Marginal linear type (Less common male type)
Type B - Central tongue shaped type (Female pattern)

Fig. 11.23: Costal cartilage calcification pattern in establishing sex identity[57]

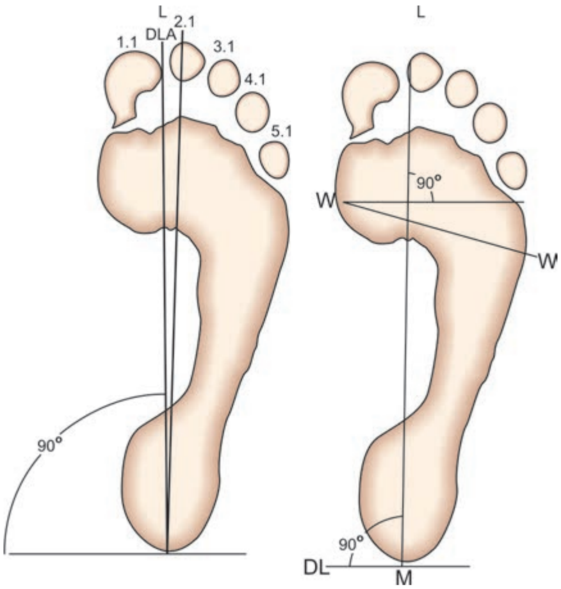


Fig. 11.24: Parameters to be measured in deriving the sex identity by foot-print ratio (FPR)[58]

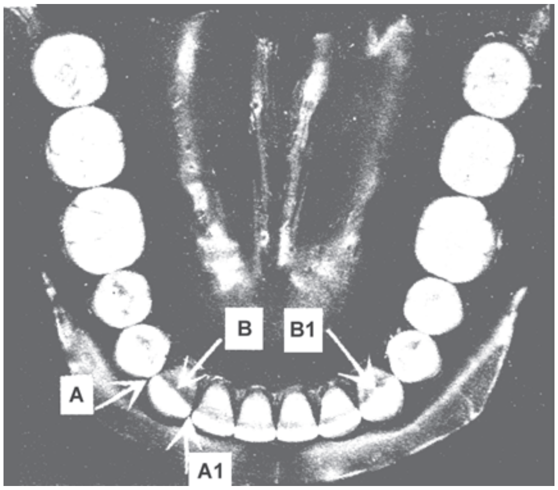


Fig. 11.25: Mandibular canine index in sex determination (AA1-Mesiodistal width; BB1 mandibular canine arch width) in establishing sex identity

(0.274) and sex is predicted as female when the values derived are within the limits of SMCI, and as male when the limits are crossed. The accuracy of this method is reported to be 85 per cent.[59,60]

Cytogenetics and DNA Analysis

This is a powerful tool in sexing which can replace all older techniques in future.[52-54]

Counting of Sex Material within the Nucleus

Stained with a fluorescent dye and viewed with U-V light, performed on the neurons in a brain smear or on cells from the kidneys.

Sex Determination in a Dead Body

Sex determination in dead body is rather easy, provided the deceased is normal physically. However, in dead bodies in mutilated or highly putrefied condition or skeletonized state, sex determination poses challenging task and methods opted routinely are: (i) meticulous autopsy examination of internal genital tract, and (ii) skeletal examination.[1-6,11]

Meticulous Autopsy Examination of Internal Genital Tract

Due to presence of intact *external* and *internal genital system* in a recently dead body, it may not be difficult to establish sexual identity. However, presence of *uterus and* appendages in a nulliparous woman and *prostate* in a man can confirm the sex of an individual, even in a *highly decomposed cadaver.*[1-6]

Skeletal Examination[11,22,23]

Skeletal remains or bones are also helpful in establishing sexual identity. Sexing the skeleton, which is intact and entire, is certainly easier as against only a part of the skeleton that is available.

Table 11.17 as notified by *Krogman,* gives an idea regarding the percentage accuracy of sexing by skeletal remains. Diagrammatic representation of skeletal findings in favour of

Table 11.17: Accuracy in determining sex identity by skeletal remains

Skeletal remains/bones	*Accuracy (%)**
Entire skeleton	100
Skull + pelvis	98
Pelvis + long bones	98
Skull + long bones	95
Pelvis alone	95
Skull alone	93
Long bones only	85

* Adopted from Krogman WM, in 'The human skeleton in Forensic medicine', Charles C Thomas, Illinois, USA, 1962[23]

sex determination is provided in the form of photographs (Figs 11.26 to 11.30) and also in tabular form in Tables 11.18 and 11.19.

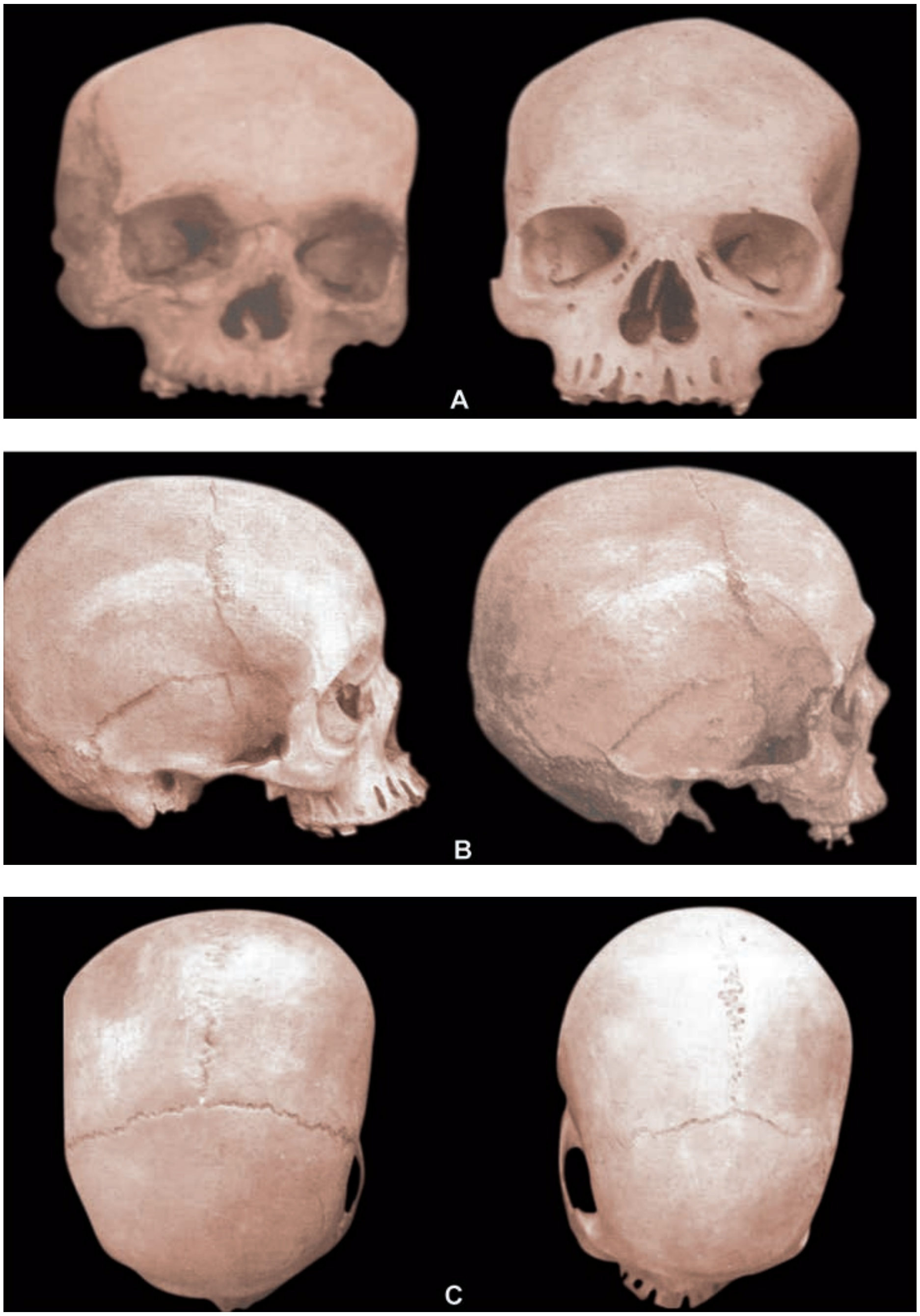
Medicolegal Importance of Sex Identity

Medicolegal significance of sexual identity can be discussed under following subheadings:

- Concealed sex
- Intersex.
- Psychological sex
- Environment and upbringing.

Concealed Sex

Criminals may try to conceal their sex to avoid detection by Police by changing dress and other means. In cases of individuals



Figs 11.26A to C: *Male and female skull:* (A) Anterior view, (B) Lateral view, (C) Superior view

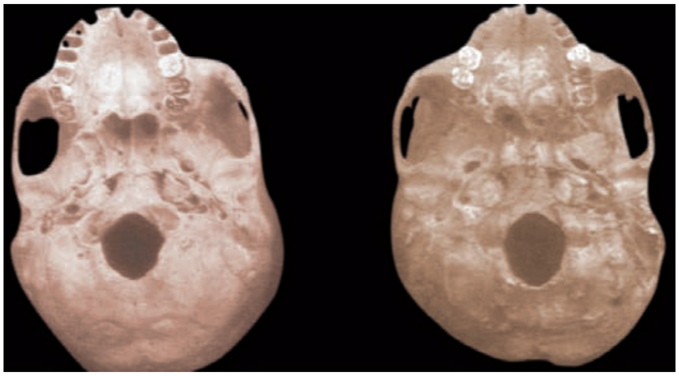
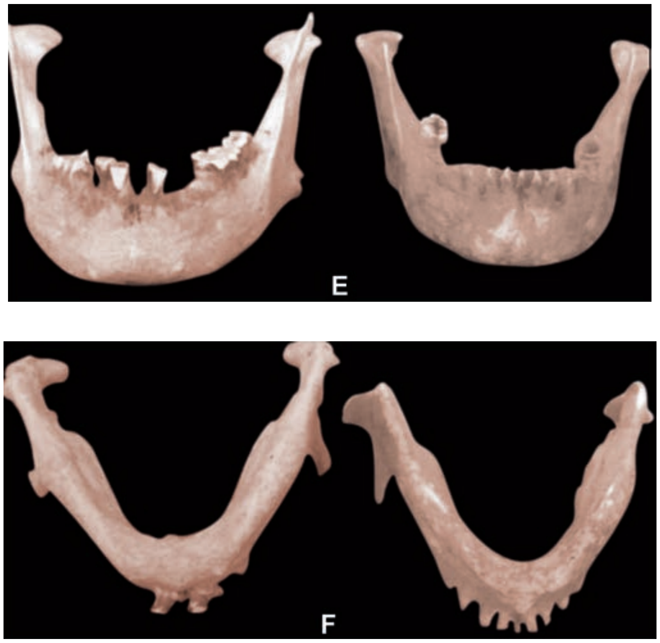
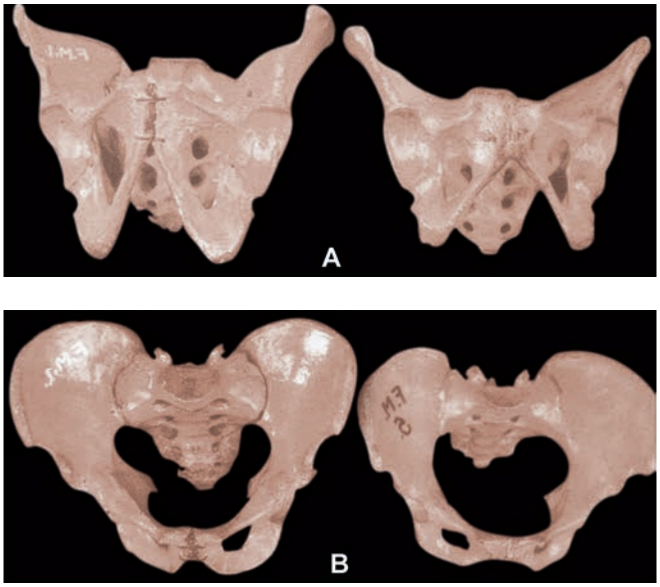


Fig. 11.26D: Male and female skull—inferior view



Figs 11.26E and F: Male and female mandible (E) Anterior view, (F) Inferior view



Figs 11.27A and B: Pelvis in male and female (innominate and sacrum): (A) Anterior view, (B) Superior view

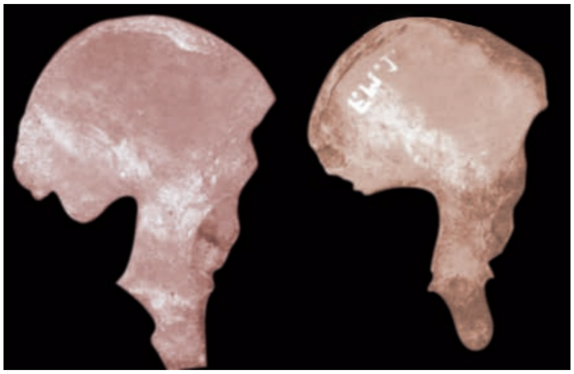


Fig. 11.28: Male and female innominate—posterior view

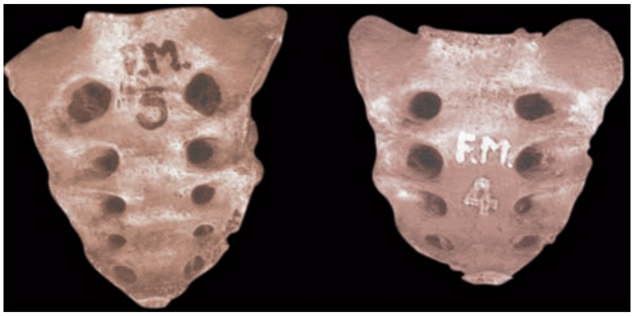


Fig. 11.29: Sacrum—anterior view; male and female

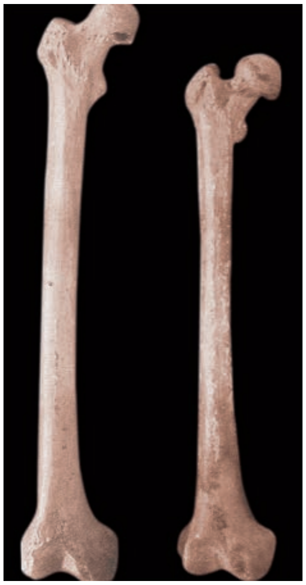


Fig. 11.30: Femur: anterior view male and female

who are suffering from acquired or congenital sexual abnormalities an attempt may be made to conceal the sex. Simple undressing of the person in a doubtful case will be sufficient to know the true sex or otherwise.

Intersex

Sexual differentiation and normal subsequent development are fundamental to the constitution of the human species. Intersex means intermingling of sexual characteristics of either sex in one individual to a varying degree, including the physical form, reproductive organs and sex behavior. There are several types of intersex and four types are recognized medico legally and

Table 11.18: Sex differences in human skeleton (Figs 11.26 to 11.30)

Bone features	*Male*	*Female*
Skeleton in general		
Size	Large, massive	Small, slender
Weight	4.5 kg	2.75 kg
Skull		
Anterior surface		
Frontal surface	Irregular and rough	Smooth and fine
Glabella	Prominent	Less prominent
Supra-orbital ridge	Prominent	Less prominent
Zygoma	Prominent	Less prominent
Supra-orbital margin	Rounded	Sharp
Shape of the orbit	Rectangular and small	Round and large
Superior surface		
Frontal eminence	Less prominent	More prominent
Parietal eminence	Less prominent	More prominent
Inferior surface		
Palate	U-shaped	Parabola
Articular facets	More prominent	Less prominent
Mastoid process	More prominent	Less prominent
Foramen magnum	Large	Small
Occipital protuberance	More prominent	Less prominent
Mandible		
Size	Larger, thick	Small, thin
Chin (symphysis mentii)	Square or U-shaped	V-shaped
Angle: body-ramus	Less obtuse	More obtuse
Angle of mandible	Everted	Not so
Condyles	Larger	Smaller
Pelvis		
Pelvic brim/inlet	Heart-shaped	Circular/oval
Pelvic cavity	Conical and funnel-like	Broad and round
Subpubic angle	Narrow (V-shaped)	Wide: U-shaped
Hip bone (innominate)		
Preauricular sulcus	Faint/absent	Well marked
Greater sciatic notch	Small narrow and deep	Large, wide and shallow
Body of pubis	Triangular	Square
Ischial tuberosity	Inverted	Everted
Sacrum		
Size	Large, narrow	Small, broad
Length	More	Less
Width	Less	More
Curvature	Uniform	Abrupt
Sacral promontory	Prominent	Less prominent
Femur		
Head	Large, 2/3rd sphere	Small, not 2/3rd sphere
Neck-shaft angle	Wider	Narrower
Bicondylar width	More	Less

they are: (i) gonadal agenesis, (ii) gonadal dysgenesis, (iii) true hermaphrodites, (iv) pseudohermaphrodites and (v) hormonal intersex.[8,9]

Gonadal agenesis: In gonadal agenesis testes and ovary have never developed. Nuclear sex is negative.

Gonadal dysgenesis: In gonadal dysgenesis, the external sexual structures are present, but at puberty the testes/ovaries fail to develop, e.g. *Klinefelter's syndrome, Turner's syndrome.*

- *Klinefelter's syndrome*: Anatomically male (Fig. 11.31) but nuclear sex is female. Sex chromosome pattern is XXY *(47 chromosomes)*. Presence of 'Y' chromosome gives resemblance to the male, and these individuals are chromatin +ve, like a female because of the extra 'X' chromosome. Characteristic features are - they resemble male externally in general body conformity, with smaller/normal size penis with smaller testicles normally placed. Sterility is common, gynecomastia is present frequently, with a high pitched voice,

Table 11.19: Mathematical sexing of bones

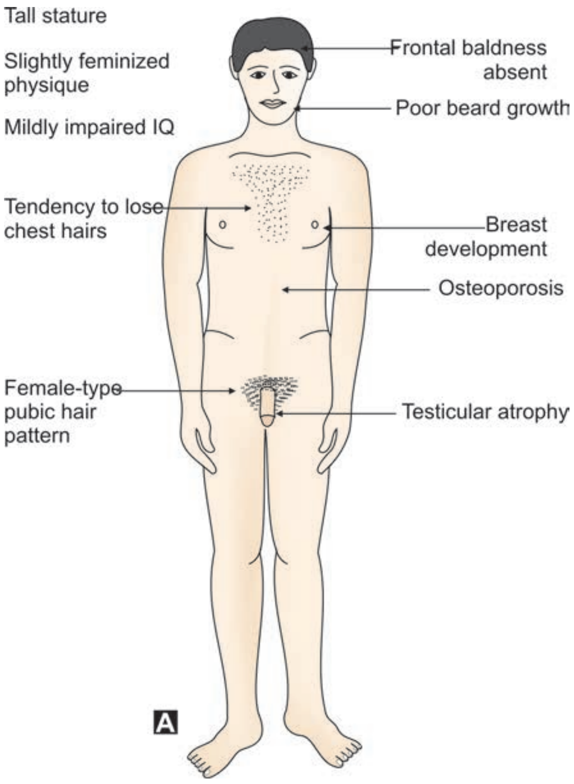
Bones	*Parameters measured*	*Male*	*Female*
Atlas	Breadth	83.00 mm	72.00 mm
Humerus	Length/	322.00 mm	290.00 mm
	Vertical diameter—head	48.00 mm	40.50 mm
	Bicondylar width	60.00 mm	57.50 mm
Radius	Length	242.00 mm	201.20 mm
	Vertical diameter—head	22.50 mm	21.50 mm
Femur	Length	439.00 mm	412.00 mm
	Vertical diameter—head	48.00 mm	41.00 mm
	Bicondylar width	79.50 mm	70.50 mm
Tibia	Length	370.00 mm	358.00 mm
	Bicondylar width	74.00 mm	65.80 mm
Indices	Ischiopubis index	83.60	99.50
	Sciatic notch index	4 - 5	5 - 6
	Sacral index	112.00	116.00
	Corporobasal index	45.00	40.50
	Sternal index	46.20	54.30

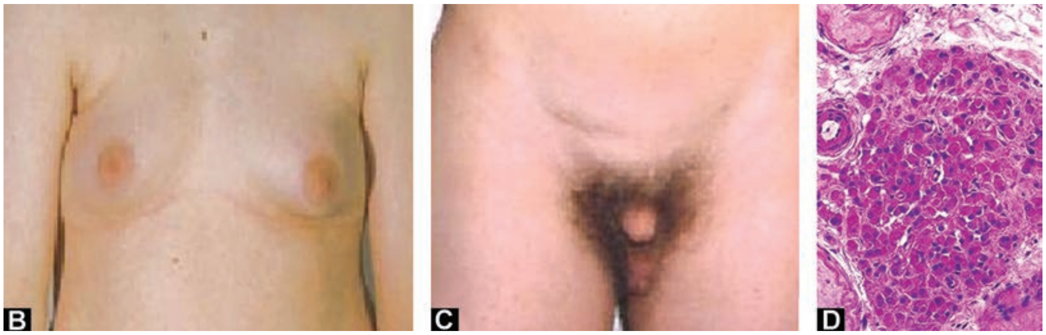
giving a eunchoidal appearance. Testicular biopsy usually reveals hyaline degeneration of the seminiferous tubules as a result of which sterility is the presenting symptom.

- *Turner's syndrome:* Anatomically female (Fig. 11.32), but the nuclear sex is male. Sex chromosome pattern XO *(45 chromosomes)*. Absence of 'Y' chromosome gives the individual resemblance to the female, but individual will be chromatin negative, i.e. there will be no *Barr bodies* or *drum sticks* in the neutrophils. Vagina and uterus if present are undeveloped. Person will be of short stature, will have oedema of hands and feet, wide carrying angle, broad chest with widely placed nipples and webbed neck. Occasionally individual will have associated features such as colour blindness, coarctation of aorta and short metatarsals with deformities of digits (cubitus valgus), etc. As they reach puberty, they fail to present with secondary sexual characteristics, as they posses streak ovaries, which are incapable of producing oestradiol.

True hermaphrodites: This is a condition of bisexuality. Here both ovary and testis (ovotestis) with external genitalia of both sexes exist in one individual. Sex chromatin may be of either male or female pattern. According to Watanabe there are 3 varieties of true hermaphrodism.[62]

1. *Bilateral true hermaphrodism:* There is a testis and an ovary (ovotestis) on each side (Figs 11.33A and B).





Figs 11.31A to D: Klinefelter's Syndrome. *Above:* Illustration of clinical manifestations (A) *Below:* Case of Klinefelter's Syndrome (anatomically male but nuclear sex female)—adolescent male. *Note:* Gynaecomastia and no chest hairs (B); Smaller penis, normally placed but smaller testicles, feminine distribution of pubic hair (C). *Testicular biopsy* — hyaline degeneration of seminiferous tubules (D)

2. *Unilateral true hermaphrodism:* There is a testis and an ovary (ovotestis) on one side and either a testis/an ovary on the other side (Figs 11.33C and D).
3. *Alternating true hermaphrodism:* There is testis on one side and the ovary on the other side.

Pseudohermaphrodite: It could be of two subtypes:

- *Female pseudohermaphrodites* has male external features, but internally has the female gonads (Fig. 11.33E).
- *Male pseudohermaphrodites* has female external features, but internally has the male gonads (Fig. 11.33F).

Hormonal intersex: Sexual variation could be due to the hormones. In the *female pseudohermaphrodites*, an excess production of androgenic hormone by adrenal cortical hyperplasia can modify the external genitalia of a genetic female. Hypertrophy of the phallus, fusion of the labia majora and hirsutism may cause the parents to consider their child to be a male. The virilising tumours of the ovary, such as arrhenoblastoma, can cause hirsutism, hypertrophy of the clitoris, deepening of the voice, masculine body contours and amenorrhoea. The presence of estrogen in the male can cause gynecomastia. These are all examples of how hormones, natural or therapeutic, can modify the sexual organs and secondary sexual characteristics.

Psychological sex: Many men and women are psychologically dominated towards sexual inversion, a persistence of the childhood tendency. Behaviour, speech, dress and sexual inclination proclaim this fact. Transvestism and effeminate behaviour are the most obvious and complete examples, where men dress in women's clothes and assume that gender role, and vice versa.

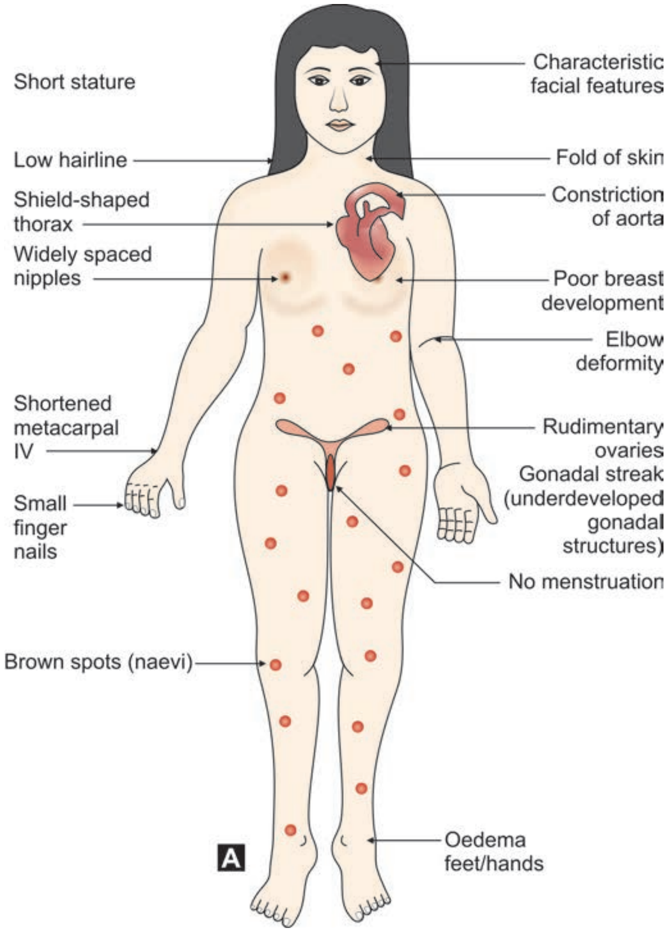
Environment and upbringing: This decides the sex of rearing. There are many examples of genetic males and females being reared by their parents in the mistaken sexual category, and who have acquired over the years the habits and mental inclination of the opposite sex to a sufficient degree to pass off as members of the opposite sex.

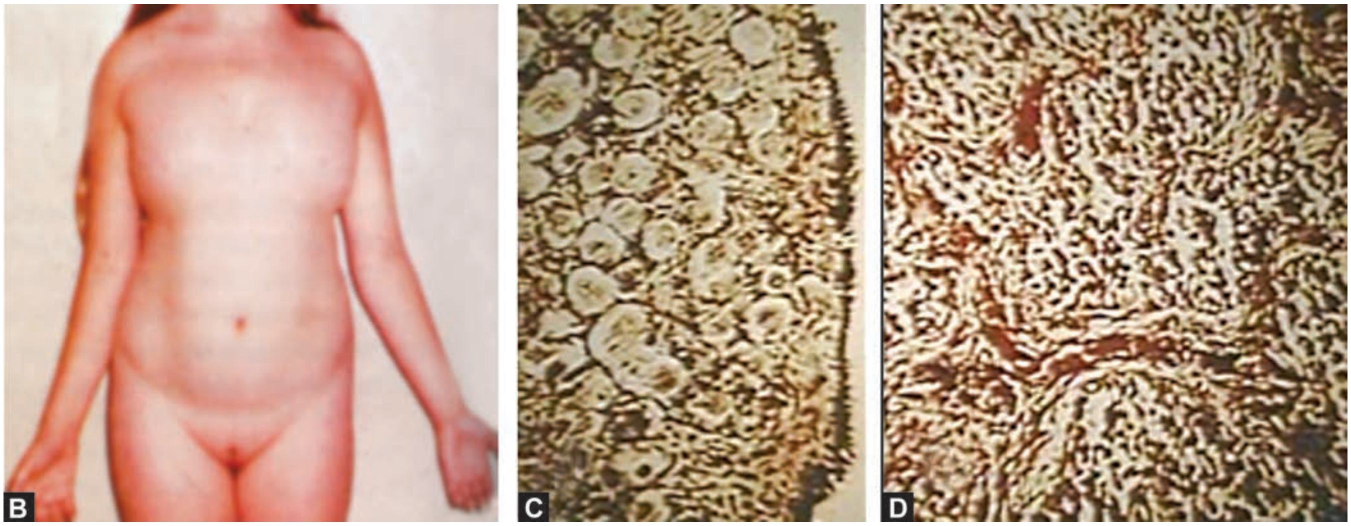
IDENTITY BY OTHER FACTORS

Various important other factors helpful in establishing identity are—racial characters, dactylography, poroscopy, footprint, complexion, features, hair, stature, deformities, tattoo marks, scar, occupational stigmata, anthropometry, trace evidence factors, etc.

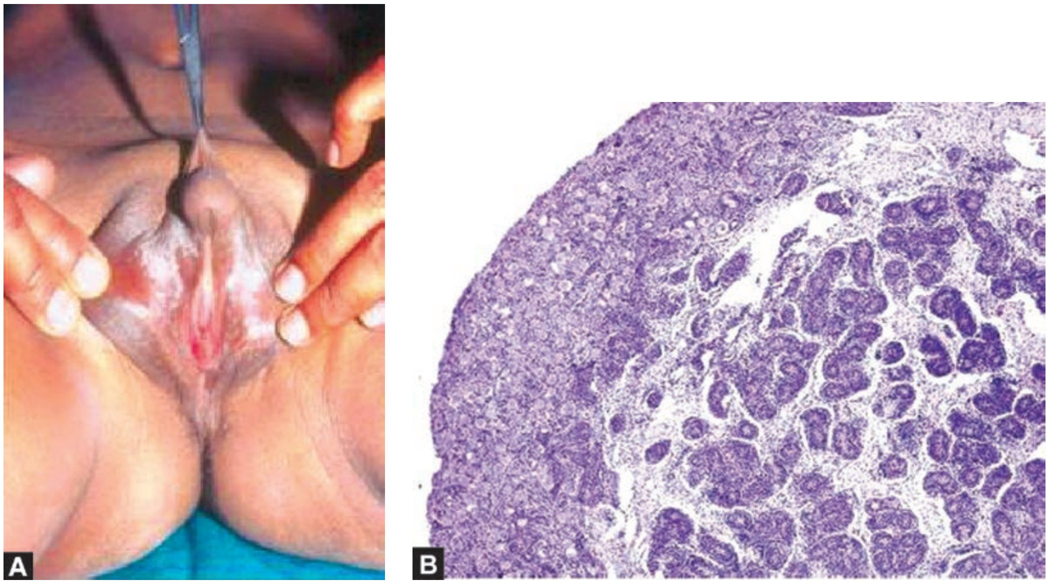
Race and Racial Characters

These are discussed mainly under the speciality field of Forensic Anthropology. Increase in mass disasters often utilises race and

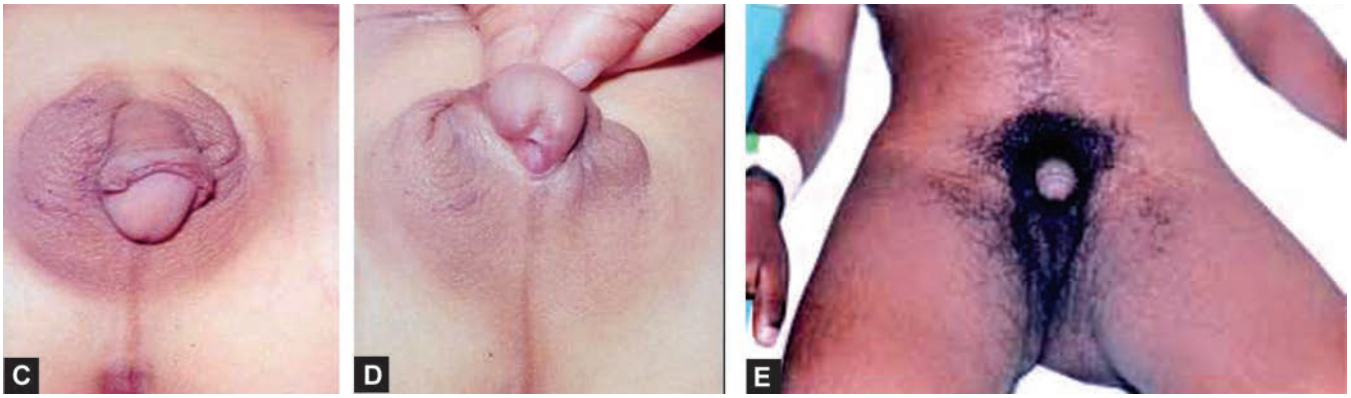

Short stature
Low hairline
Shield-shaped thorax
Widely spaced nipples
Shortened metacarpal IV
Small finger nails
Brown spots (naevi)
Characteristic facial features
Fold of skin
Constriction of aorta
Poor breast development
Elbow deformity
Rudimentary ovaries
Gonadal streak (underdeveloped gonadal structures)
No menstruation
Oedema feet/hands
A

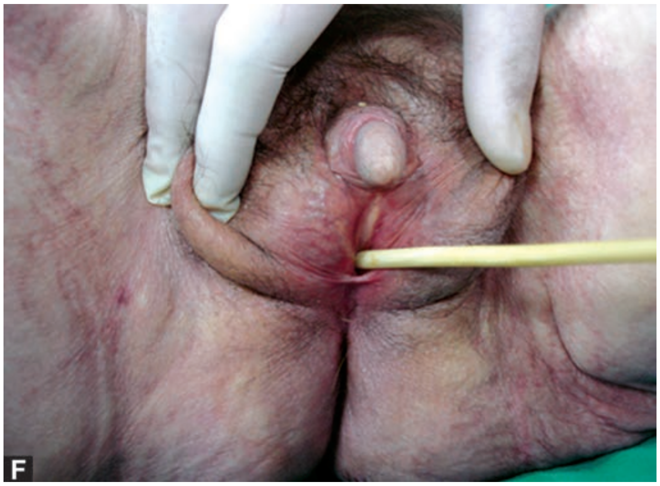


Figs 11.32A to D: *Turner's syndrome: Above:* Illustration highlighting the clinical manifestations (A). *Below:* Case of Turner's syndrome (anatomically female with nuclear sex male). *Note:* short stature, web neck, wide carrying angle, oedema of hands and feet, broad chest with widely placed nipples, cubitus valgus, lack of secondary sexual characteristics at puberty (B). Comparative histopathology ovary—Normal ovary with eggs (C), Streak ovary with no eggs (D)



Figs 11.33A and B: *Bilateral true hermaphrodite:* (A) Well developed phallus, bilateral ovotestes, with fish mouthed urethra below the phallus and vaginal opening in between scrotal sacs (B) Histopathology of ovotestes showing - ovarian component forming a crown around the testicle parenchyma





Figs 11.33C to F: (C and D) Unilateral true hermaphrodite with ambiguous external genitalia. (C) Well-developed phallus and right scrotal ovotestis and testes on left side. (D) Penoscrotal hypospadias. (E) Female pseudohermaphrodite with male external features with a hypertrophied clitoris and fused labia but has female gonads internally; (F) Male pseudohermaphrodite with female external features but has male gonads internally (*Source:* Shilpa Sharma and DK Gupta, Male genitoplasty for intersex disorders. Advances in Urology: Volume 2008 (2008). Article ID: 685897)

racial characteristics in establishing the identity of victims.[23,27,65] According to the anthropological sciences, there are three ***primary races*** in the world – Caucasoid, Mongoloid and Negroid. However, as per Blueman Backs classification there are five human races on the basis of skin colour,[5] and they are:

1. Caucasian (white)
2. Mongoloid (yellow)
3. Ethiopian (black)
4. American (red)
5. Malayan (brown).

Most of the Indians show Caucasoid features. However, Mongoloid features are more common in the North-Eastern population, while, some of the Negroid features are common among the Southern Indian population.[29,63] Race determination usually depends on following:

Table 11.20A: Differences in morphologic feature/traits of three primary races of world population

Traits	*Caucasian*	*Mongoloid*	*Negroid*
Population (Original)	Europeans	Chinese	Africans
Skin	Thin and fair	Pale and yellow	Tought and black
Iris	Blue/gray	Black	Black
Hairs (Scalp)	Thin, straight or wavy, with fair/light brown/ reddish colour	Straight or wavy with black colour	Curley and wooly with black colour
Face		Flattened	Small and compressed
Lips			Big and full
Extremities			
• Upper		Small	Longer forearms than arms
• Lower		Small	Longer legs than the thighs
Teeth		Lower 1st premolars may have 3 cusps and both permanent and temporary molars will have 3 roots	Obliquely placed (proclenated) with outward projection
Skull:			
• Shape	Round	Square	Narrow and oblong
• Size	Mesaticephalic (intermediate)	Brachycephalic (small)	Dolicocephalic (large)
• Cephalic index	75-80	80-85	70-75
• Forehead	Raised	Inclined	Small and compressed
• Orbits		High and roundish	Low and wide
• Nasal aperture	Narrow		Broad and wider

Table 11.20B: Osteometric indices (refer Table 11.20C) helpful in determining the races

Various indices	*Values*		
	Caucasian	*Mongoloid*	*Negroid*
Cephalic Index	(Please refer the Table 11.20D)		
Brachial Index	Average Europeans 74.5	—	Average Negroids 78.5
Crural Index	Average Europeans 83.3	—	Average Negroids 86.2
Humero-femoral Index	Average Europeans 69	—	Average Negroids 72.4
Inter-membral Index	Average Europeans >70	—	Average Negroids <70.5

Table 11.20C: Deriving various osteometric indices

Various indices

$$\text{Cephalic Index} = \frac{\text{Max. Breadth of skull}}{\text{Max Antero-Posterior length}} \times 100$$

$$\text{Brachial Index} = \frac{\text{Length of Radius}}{\text{Length of Humerus}} \times 100$$

$$\text{Crural Index} = \frac{\text{Length of Tibia}}{\text{Length of Femur}} \times 100$$

$$\text{Humero-femoral Index} = \frac{\text{Length of Humerus}}{\text{Length of Femur}} \times 100$$

$$\text{Inter-membral Index} = \frac{\text{Length of Humerus} + \text{Length of Radius}}{\text{Length of Femur} + \text{Length of Tibia}} \times 100$$

1. *Morphological characteristics/traits*: The three primary races that helped in establishing identity are presented in Table 11.20A.
2. *Osteometric parameters*: Osteometry in anthropological sciences comprise of measurements of different bones in the body, assessing its proportionalities to each other and multivariate discriminate functional analysis. This has led to the genesis of several indices (Table 11.20B) and discrimination function techniques.

Cephalic Index

Cephalic index (CI) is defined as the ratio between breadth and length of the skull expressed as below. Table 11.20D illustrates three types of races by cephalic index.

Table 11.20D: Cephalic index values and determination of race

Types of skull	*CI*	*Race*
Dolicocephalic (Long head)	70-75	Pure Caucasians, Negroes and Aborigines
Mesaticephalic (Medium)	75-80	European Caucasians and Chinese Mongoloid
Brachycephalic (Short head)	80-85	Pure Mongolians

$$\text{Cephalic Index (CI)} = \frac{\text{Maximum breadth of skull}}{\text{Maximum length of skull}} \times 100$$

Religious Factors in Establishing the Racial Identity

These are certain religious factors among the Indians that could help in racial identity.[24,25] Religion in India ranks among the worlds most ancient and varied. The Indian subcontinent spawned Indian religions, which compose one of the world's three major groups of religions. Vedic religion gave rise to what is now the oldest extant and World's third-largest religion, *Hinduism*; the Abrahamic religions of *Islam* and *Christianity*, spread by missionary activity and conquest over many centuries, are the two other largest non-native religions.[65-67] Rest of the smaller groups of religions existing in India are: *Sikhism, Buddhism, Jainism, Zoroastrianism and Judaism*. More than four-fifths of Indians practice Hinduism. Islam, practised by around one-sixth of the population, is the most prevalent minority religion. *Christianity* and *Sikhism* are each practised by around 2 per cent of Indians.[68] About 1.1 per cent practice *Buddhism* and 0.4 per cent practice *Jainism*. *Zoroastrianism* and *Judaism* have centuries-long history in India; each has several thousand Indian adherents. Thus Indians represent predominantly two religious people – *Hindus* and *Muslims (Mohammedans)*. Some of features which help in establishing these two religious identities are enumerated below:[2,3,5,6,24,25,65,66]

A typical Hindu male will wear *dhoti, kurta* as traditional dress. Among the Hindus, in certain community namely *Brahmins* will have a total shaved scalp except a tuft of hairs grown long on the occipital protuberance region. These individuals may paint a *tilakum/vibhooti* on the forehead and other parts of the body also such as front of the trunk, side of upper arms, forearms, front of neck etc, made up of sandal wood paste/vermilion/turmeric. They may also wear a *sacred thread* which is a big loop of thread made up of six strands, worn on the chest across left shoulder; a necklace of *Rudraksham* beads worn around the neck.

A typical Hindu female will wear a *sari* and *choli*, etc. Married women will usually wear a beaded necklace made up of red corals and black beads and a thread (Mangolasutra/Tali) with application of vermillion (Kumkum) over the forehead on the hair partition/hairline. They may also wear a nose stud made up of diamond/white stone on the left ala and silver toe rings on the second toe of both feet.

A typical Muslim male will wear *pyjama* and *salwar*. Most of them will have a tapering beard. It is customary/traditional for a male Muslim to submit himself for circumcision of the penis as early as 5-7 years. There will be callosities over the forehead, and lateral malleolus of left leg due to the particular position opted during the practicing of prayers—*Namaz*.

A typical Muslim female will wear a *shalwar* and *kameez*, etc. with a black gown (*Burkha*) covering completely, hiding the person from head to toes, except over eyes. They will wear multiple nose and ear rings; will thus have nose pierced over the ala as well as the nasal septum and on the margins of the helix and lobule of both ears.

Nationality and Language: Characteristics representing nationality of a person and language spoken may also help in establishing the identity of a person. The type of dress worn, articles preserved in the pockets and wallets, passport, a tattoo mark/words inscribed, etc may reveal the nationality and language spoken. In a living person talking to him/her can easily express the language identity.

Dactylography (Dermatoglyphics, Dactyloscopy, Galton System, Finger-print Study)

Federal Bureau of Investigation (FBI, USA), celebrating the centenary (1908-2008) on finger-print identification, proclaims that '…*other personal characteristics may change, but finger-prints do not*…' This is unquestionably true and is approved globally.[67,69]

Criminal identification by means of fingerprints is one of the most potent factors in apprehending fugitives who might otherwise escape arrest and continue their criminal activities indefinitely. This type of identification also makes possible an accurate determination of the number of previous arrests and convictions which results in the imposition of more equitable sentences by the judiciary. In addition, this system of identification enables the prosecutor to present its case in the light of the offender's previous record.

Finger-print identification is the method of identification using the impressions made by the minute ridge formations or patterns found on the finger-tips. No two persons have exactly the same arrangement of ridge patterns, and the patterns of any one individual remain unchanged throughout life. Finger-prints offer an infallible means of personal identification. Finger-prints can be recorded on a standard finger-print card or can be recorded digitally and transmitted electronically. By comparing finger-prints at the scene of a crime with the finger-print record of suspected persons, officials can establish absolute proof of the presence or identity of a person. This study of ridge pattern is applicable to the skins of the palms and soles also. Finger-print study is thus highly individualistic, providing positive identification in not only living person but also in a dead and decomposed or even mutilated hands/feet. Thus, fingerprint is an impression of the ball of a finger and this gives the details of arrangement of the papillary or epidermal ridges on the skin of the finger ball. The friction skin (specialized ridged skins) of the palms and soles differs significantly from the skin covering the rest of the body as follows:[47-49]

- It is hairless and with no oil glands or any pigments. However, it has papillary ridges (Fig. 11.34A) in both epidermis and dermis and arranged in specific pattern, which derives uniqueness for the finger-prints/ impressions in establishing accurate identity.
- This friction skin also has higher concentration of sweat glands and these accrine sweat glands contribute mainly to the finger print residues. The direct contact between the fingers/palms

and the substrate on which the finger-print would be deposited can be avoided by wearing the gloves or any other substances/material intervening. Thus, the absence of a person's finger-prints on an object does not eliminate the person from having touched the object.

Classifications of Finger-print Pattern

Sir Henry Galton (1892), depending on the arrangement of papillary ridges classified the finger-prints into four major or primary types (Fig. 11.34B) (Box 11.1).

1. Loop (65% of population)
2. Whorl (25% of population)
3. Arch (07% of population)
4. Composite/compound is a mixed picture of all three patterns above (02-03% of population).
5. To these four patterns a fifth one called "Accidental variety" is added, wherein no specific ridge pattern is available.[5]

However, finger-prints are further classified as follows:

Loops: Loops usually begin on one side of the finger and end on same side. When this happened from ulnar side it is called ulnar loop end if on radial side — radial loop.

Whorl: This could be having multiple circular/oval ridges, one around the other, or a single ridge, round in multiple rounds.

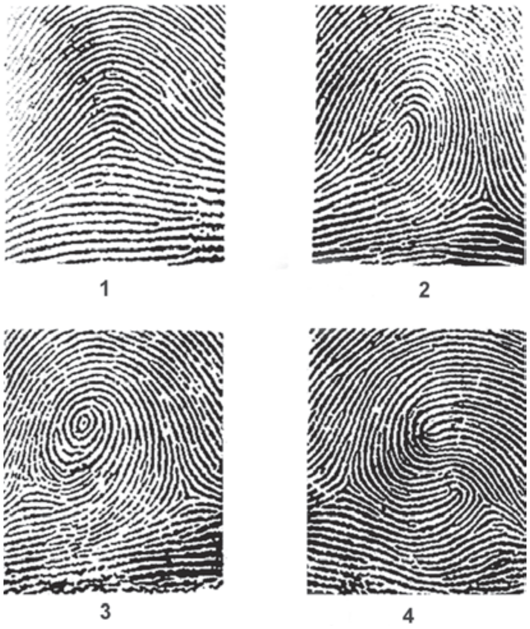


Fig. 11.34B: *Four primary types of finger-prints:* (1) Arch, (2) Loop, (3) Whorl, and (4) Composite

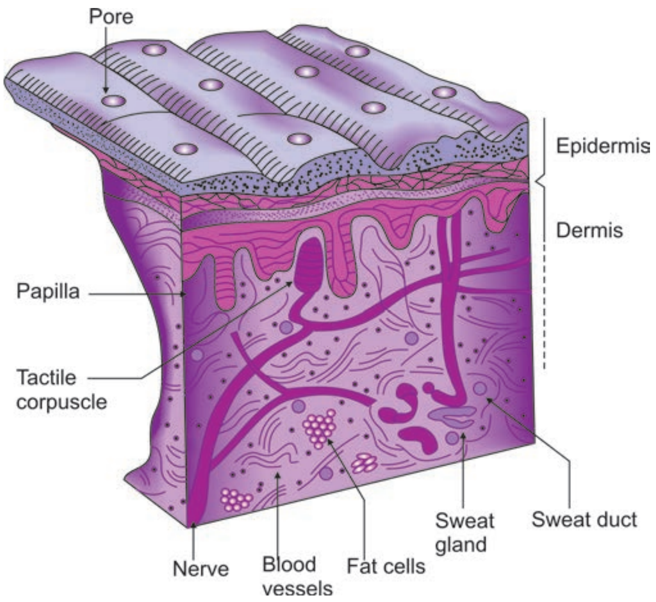


Fig. 11.34A: Skin structure showing finger-print ridges

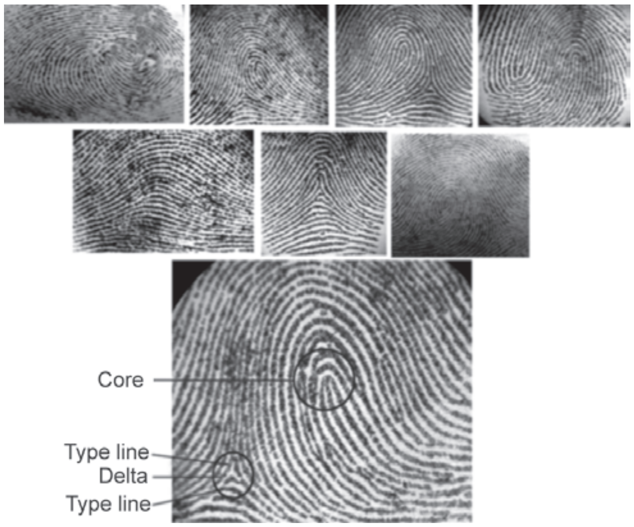


Fig. 11.34C: *Further Classification of finger-prints: Top row (Left to right)*: Accidental type, ulnar loop, radial loop, double loop, *Second row (Left to right)*: Plain arch, tented arch, central pocket loop, *Lower row*: Other terminologies of ridge pattern—Type line, delta and core

Box 11.1: Finger-print: Historical aspects[2,3,5,24,69]

Use of finger-prints in record, in the official documents of China dates as back as 3000 BC. Ancient Indian documents also makes mention of practice of the same in India. On the cliff of Nova Scotia, papillary ridge like carvings of pre-historic age have been discovered, but without any evidence to say its use during that era. In 1880 scientific journal "Nature" published the first paper by Henry Faulds of Tokyo on finger-prints, stating that ridge pattern remains unchanged throughout the life of a man and that ridge pattern could be of help in establishing identity of a criminal by comparing it whenever recovered in the crime scene with suspect/accused. It is also stated that first scientific study on finger-prints was done by Prof. M. Melphige in the year 1680 and Prof. JE Purkinje, classified them into 9 types.[5] "Nature" also made mention about a British officer of East India Company in India, Sir William Janus Harshel, working in the Hoogly District of West Bengal, used palm-print of Indian pensioners and prisoners for identifying them legally. In the year 1892, Sir Frances Galton published his work on finger prints in the form of a book "Finger Print" which later on Sir Edward Henry, the commissioner of Scotland Yard implemented officially in criminal investigation, with little modifications in England and Wales in 1901, and other countries of the World and was popularly known as "Henry Galton System" or "Galton System of Identification".

Arches:

i. Plain arch – when it is wave like (from one side of the finger to other side).
ii. Tented arch – when the arch is sharp and spike like.

Composite: It is a combination of more than one pattern, either a combination of arch, whorl and loop, or two different pattern (twin/double) two whorls and arch or loop.

Other Terminologies of Ridge Pattern

Other terminologies of ridge pattern in technical use in finger-print study are as presented below:

1. *Central pocket loop*: Here, surrounding the central circular/oval ridge, there are other ridges which take loop like course.[5]
2. *Type line*: In loops and whorls, the ridges in the middle/main part are surrounded by divergent ridges and they are called type lines.
3. *Delta*: In loops and whorls, the point of the outer most ridges nearest to damaged type line is called delta. Arches have no deltas.
4. *Core*: In loops and whorls, the apparent central point of the ridge pattern is called core. Thus the central point of starting of whorl type ridge becomes core.

Different types of these ridge patterns are represented diagrammatically in Figure 11.34C.[5]

Maintenance of Finger-print Records

Federal Bureau of Investigation (FBI, USA) maintains record of more than two crores (two billion) of finger prints. Yet whenever necessary for comparative study, a required finger-print can be made available from this within a minute or two. This is possible due to systemic storing of finger-prints in separate files for different types of prints. Filing is done by allocating scores for presence of whorl pattern in different fingers in each hand. The score allotted so, is presented below in a tabular form (Table 11.21A). Thus, if every finger in a person has a whorl then the score is determined as follows:[5,67]

$$= \frac{\text{RI+RR+LT+LM+LL+1}}{\text{RT+RM+RL+LI+LR+1}} = \frac{16+8+4+2+1+1}{16+8+4+2+1+1} = \frac{32}{32}$$

Now multiply 32 × 32 = 1024. This will provide the maximum number of score for a person. If a person has no whorl in any of the fingers than the score is:

$$= \frac{0+0+0+0+0+1}{0+0+0+0+0+1} = \frac{1}{1} \quad \text{i.e. } 1 \times 1 = 1$$

Table 11.21A: Scores allotted for fingers showing whorl pattern

Finger showing a whorl pattern stage	*Score allotted*
Right thumb	16
Right index	16
Right middle	8
Right ring	8
Right little	4
Left thumb	4
Left index	2
Left middle	2
Left ring	1
Left little	1

It is estimated that 60 per cent of world population has no whorls in their hands. Thus by the presence or absence of a whorl in the fingers a primary classification with the total scores can range from 1 to 1024. Based on this, a total number of 1024 boxes are made, which are termed as *pigeon holes*. According to total score, a person's details and his finger-print is preserved in the box, bearing the same number. For people with no whorls, anyway a secondary classification is adopted with further segregation of the scores, distributing the prints efficiently allowing easy and quick accessibility to any of the finger-print as needed.

Methods of Taking Finger-prints

1. Wash the finger properly with soap and water.
2. Ink the ball of the finger with printer's ink.
3. Use an unglazed white paper.
4. Take finger-print in two different types:
   - *Plain finger-print* by just pressing the inked ball of the finger on the paper.
   - *Rolled finger-print* by pressing the inked finger ball, roll it from one side to other side of the finger. Rolled finger-print is always better for reasons such as:
     - It gives wider print, providing wider surface area of the finger.
     - It offers better study of pattern of ridges.

Medicolegal Importance of Finger-print

- Epidermal ridges in the skin of palms and soles are developed as early as 4th month of IUL.
- Finger-print is a 100 per cent accurate method in establishing identity. No finger-prints of 2 individuals, even in case of monozygus identical twins are similar.[48] An estimated scientific report pronounces that chances of resemblance of finger-prints in two people is just :1 in 64,000,000,000 (billions), which renders finger-prints a unique method of establishing human identity. At this juncture, it is good to remember that the DNA technology is incapable of differentiating between the identical twins.
- *Visible finger-prints*: Fingers when smeared with blood, grease, etc. will leave their prints/impression on the weapon used for the crime/ furniture or such other articles that may be touched by criminal at the scene of crime unintentionally and can give clue for identifying the criminals. Such finger-prints constitute *visible finger-prints*.[2,5]
- *Latent finger-prints (invisible finger-prints):* Finger-print experts can render a faint and invisible/latent finger-print visible by special techniques using some *developing agents* which are chemical powders and liquids (Table 11.21B). When these latent finger-prints are obtained at the crime scene by chance, these prints are termed as the *chance finger-prints*.[5] Table 11.21B presents various methods of developing latent finger-prints and these prints being developed can be preserved by lifting them up on an adhesive/gum tape.[2,3,5]
- *Plastic finger-prints*: These are prints of the finger left on soft materials such as wax, soap, dust, etc.[2,5]
- *Finger-prints from dead body*: Tip of the finger may get shriveled in a dead body and then may mask the picture. So to avoid this, soak the fingers in alkaline solution first and then take the print.[3] Finger-print may be even taken from putrefied body where the cuticle of finger ball is pealed off from dermis, which will have the ridge pattern still intact.[3,5]

Table 11.21B: Methods of developing latent finger-prints

Developing agent	*Method*	*Developing features*	*Remarks*
Grey powder (Aluminium dust)	Spread by camel hair brush	Powder adheres to sebaceous secretions in the lines of ridges	Use black/dark background to visualize clearly. Print can be lifted/preserved.
Black powder (mainly charcoal)	Spread by camel hair brush	Powder adheres to sebaceous secretions in the lines of ridges.	Used on light/white coloured surfaces. Print can be lifted/preserved.
Iodine vapour	Surface with print is held over the vapour.	Ridges take yellowish or brown colour.	Preserved by photography.
Nin-hydrin (0.6%) solution	Paper with finger-print is dipped/spread with fluid	Ridges take maroon/ purple colour	Good for old prints
Silicon nitrate (3%) solution	Print with brush on paper with finger-print	Ridges will take black or reddish brown colour	Used only when other tests available

- Finger-prints are important because they can be transmitted easily from one place to another by telegraphic message easily.[2,3,5]

Use of Computer System for Finger-print Study

FBI of USA uses a system called FINDER-II (referred *FINgerprint reaDER*). This can read the data about finger-prints such as ridges, bending, ridge bifurcation, image direction of finger-prints, etc. The light reflected from finger-print is measured and converted into digital data and classified and stored in its memory for future comparative studies with other finger-prints. In the FINDER-II system only 8 finger-prints are taken into consideration excluding the little finger.[5,67]

Age of the Finger-print

How old is a finger-print can also be now reported by determining the chloride ion contents of the finger-prints, which is said to be present in the sebaceous secretions and this vanishes gradually as time passes.[5]

Finger-prints and Scaring Trauma

In case of a blistering – only the epidermis is affected and it heals completely without scaring. If the injury extends further to involve the dermal layers, where the finger-print pattern resides, permanent disruption in the finger-print will occur. However, on the whole, this scar itself can constitute a variable identification mark in addition.[57,63]

Foetal Development of Epidermal Rings

Epidermal rings are said to be formed in a fetus in the 4th month of IUL.[57]

A case example wherein finger-print, acquired deformities and forensic dentistry helped in solving a mysterious crime of identifying a mutilated victim is discussed in the Box 11.2.

POROSCOPY

Poroscopy is further study of finger-prints described by Locard providing absolute identity.[1-6,67] The papillary ridges of the skin of the finger balls and hands will have plenty of *minute pores*, which are the openings of sweat glands. These pores are *permanent and vary in shape, size, position and numbers over a given length of a ridge* in each person, rendering them ideal factors for establishing identity. Method of identification by means of these pores constitutes *poroscopy* (Figs 11.35A and B).

Indications

When the finger-prints available are only a fragment and not complete.

FOOT-PRINTS

Similar to the finger-prints, skin pattern of toes and heels from the sole of the feet are distinctive and permanent-can tell identity.[1-6]

Procedure of Taking Foot-print

It is taken by using printer's ink and a clean paper (Fig. 11.36).

Collecting Foot-print Impression Left in the Soil or Sand

- Spray the impression in the soil or sand with 80 per cent of alcohol solution of Shellac. This hardens the soil or sand.
- Next, smear the hardened surface with lubricant or dust with French chalk.
- Then, pour an aqueous mixture of plaster of Paris and allow it to dry and on drying remove the cast and study the impression.

Note

- Foot-print will be larger on walking than on standing.
- Imprints left on soft and loose material like sand will be always smaller.
- Look for any deformities like — flat foot, webfoot, supernumerary toes, loss of toes, etc.

Box 11.2: Alvander Murder Case[2,3,6,24]

- *Mr. Alvander, aged 42 years was murdered on 28/8/52 by one Mr. Prabhakar Menon with help of his wife Mrs. Devaki, who had illicit sexual relation with Alvander, which evoked Mr. Menon for murder.*
- *On 29/8/52, in a third class compartment of then Indo-Ceylon Express train, a headless trunk was found and later on 31/8/52, a head alone was recovered at Roypuram sea beach, at Madras (Presently Chennai).*
- ***Dr KC Jacob**, Prof of Forensic Medicine and then City Police Surgeon, Madras Medical College, identified the mutilated remains are that of Mr. Alvander by fingerprints, presence of circumcised penis and pierced ear lobes (acquired deformities) and an overriding canine teeth (Forensic Odontology).*

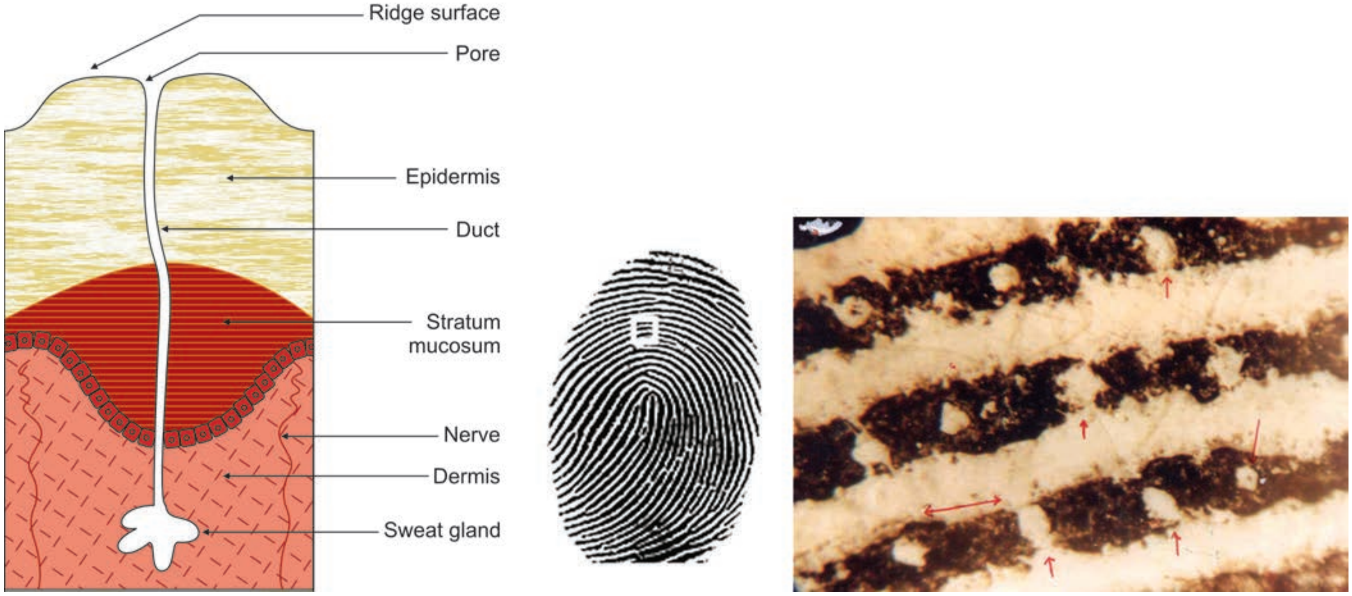


Fig. 11.35A: Poroscopy study: Line drawing on left side illustrating the structure of finger print ridges with pore of the sweat gland duct opening in the middle of the ridge. Finger-print with magnification of the area selected (white box) presenting pores on the ridges (Magnified view)

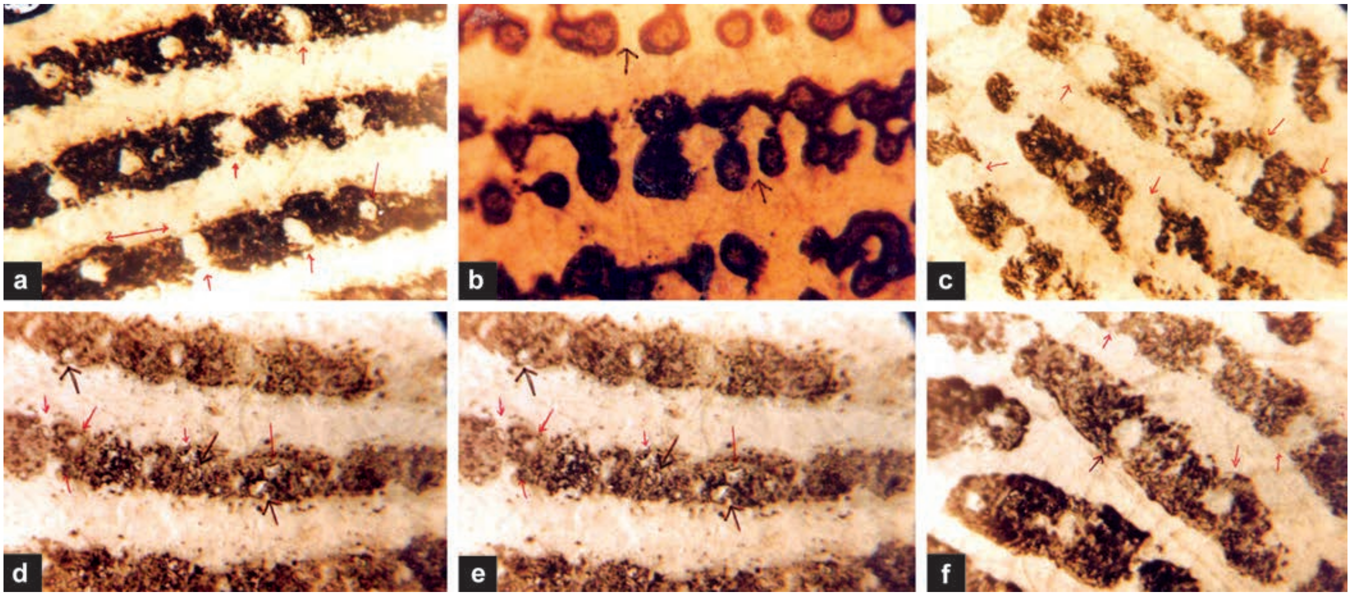


Fig. 11.35B: Poroscopy has been recently studied in detail, classified in to several types by *Bindra B, Jasuja OP and Singla AK* and they are*: (a) Pores of rhomboid and rectangular shape with open mouth, (b) Pores making chain type configuration, (c) Pores of large size with open mouth, (d) Pores of minute size lying in groups and very close to each other, (e) Pores located in the middle of the ridge, (f) Pores of medium size and rounded in shape

(* Bindra, B, Jasuja, OP and Singla AK. Poroscopy: a method of personal identification revisited. Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology, 2000; Vol. 1, No. 1) *Source:* http://www.geradts.com/anil/ij/vol_001_no_001/paper003.html (Retrieved on 21.07.2009)

Foot-print of Newborn Infants

Usually taken in certain maternity hospitals for the simple reasons that foot-prints are very reliable means to identify newborn infants, if there is any mix-up of babies and mothers.[3]

COMPLEXION

Complexion is also an important clue for establishing human identity.[1,6,24,25,27]

Examples: A European is always fair/white skinned, while the Negro is black skinned. However, complexion cannot be appreciated in case of a putrefied body.

BODY FEATURES

Body features also help one's identity.[1-6,27,29] Some of the body features of great medicolegal significance helping in human identity could be enumerated as below:

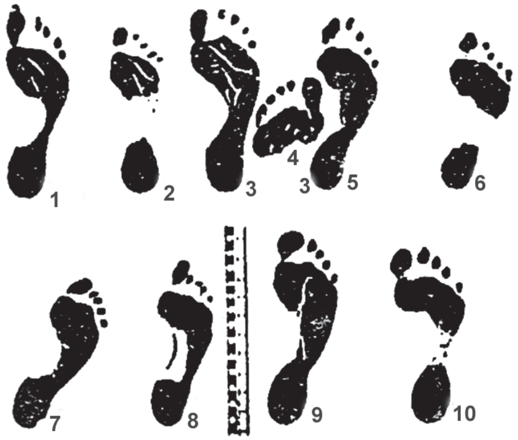


Fig. 11.36: Various types of foot-prints

- Colour of iris of the eyes
- Size and shape of ears and nose
- Shape of the chin
- Presence on the skin of:
  - – A mole (naevus) which could be plain/hairy/big/small/tiny/flat/raised and black/brown/red in colour, etc.
  - – A birthmark which could be an irregular coloured patch on the skin.

    Note: Whenever a mole or birthmark is used as a mark of identification, it is better to describe it as it is, giving its accurate dimensions in millimeters and its exact anatomic location.

A sensational case of establishing the identity by *features* of a person who was declared dead, who returns alive after 12 years and claims the share in his ancestral property – The ***Bhowal Sanyasi Case*** is presented below in Box 11.3.[1-3,6]

Identikit

This is a powerful investigative tool that can create a photorealistic facial composite sketches based on verbal description provided by a witness or victim about the criminal, which can help generating a customized "wanted" posters. Currently, different types of facial composite softwares are available on CD or internet as a hosted application and help police in crime investigation (Fig. 11.37).[42,43]

PRINTS OF OTHER BODY AREAS

Ear Prints

Shape, size, positioning, display, etc of the ears have been used to help in determination of human identity.[28,63,70] Accordingly ears could be of several types and are enumerated below (Figs 11.38A and B):

- Broad, wing like soft/hard
- Lobules of the ear may be wide and look freely hanging/fixed to the face
- Whole ear may be tending to come forward/backwards
- Ears may have hairs on the helix and tragus, seen only in male (linked to Y-chromosome).

Medicolegal importance: Ear prints are chance prints, often seen on the walls, doors, window panels etc at the crime scene, trying to hear what others are talking, behind the closed doors. They vary with pressure applied by the ear on contact surface (Fig. 11.38C).[28]

Nose Prints

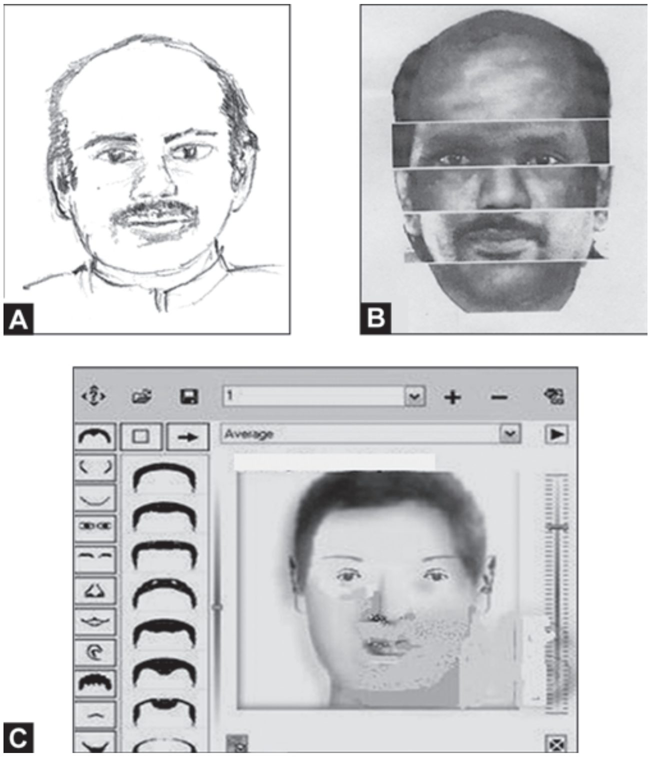
These are also chance prints, may be found at the crime scene on the mirror, corners of the walls, doors and so on. It may also be seen on the body of the victim/assistant. The lines on the nose and shape of the tip etc. (Figs 11.39A to H), may help personal identification.[28,63,71]

Palato Prints (Rugoscopy)

Anterior part of the palate has rugae or grooves and ridges on either side of midline which is considered to be individualistic and help establishing identity. Palate can be high/low; broad/narrow; etc renders them more reliable (Figs 11.40A to D). The palatal rugae has several advantages as identification criteria—they are well protected from trauma being located deep inside the mouth, also they are insulted from heat by tongue and buccal fat; no two palato prints are alike, and that they do not change with growth of the individual.[2,5,29,63]

Box 11.3: Bhowal Sanyasi Case

- ***Kumar Ramendra Narayan Roy****, second son of* ***Raja Rajendra Narayan Roy*** *of Bhowal District, in Dacca, went to Darjeeling in 1909 and he died there and was cremated also. Twelve years later, in 1921 a sadhu came to Dacca and declared that he is Kumar Roy. He said that, in Darjeeling he was attempted to murder by giving arsenic and in a comatose condition he was taken to cremation ground.*
- *There at the time of cremation (night), a heavy storm broke out and the funeral party hurriedly left the crematorium prior to the completion of cremation to his body presumed to be dead.*
- *Sometime later a naga sanyasi troup (a group of wandering saints) came there and found him alive at the crematorium and carried him with them. Later he suffered from total loss of memory (amnesia) for the subsequent 12 years, till he came to Dacca in 1921, where he got his memory back and with that he claimed share in Bhowal Estate by filing a suit at Dacca Court. The case went on for 2 years or more, but finally he was declared to be none other but Kumar Roy, and not an imposter.*
- *The court took cognizance of factors of complexion and features of the contestant in deciding the identity. Court consulted the wife of Kumar Roy,* ***Shrimati Bibhabati Devi****, who was still alive and ultimately declared that this man is none but her own husband Kumar Roy. She revealed also to the court a very personal and private mark of identity of her husband, i.e. presence of a black mole on the dorsum of the penis, which was later examined by the doctors and found to be present in the said person, proving his identity.*



Figs 11.37A to C: *Identikit:* (A) Sketch of the criminal made by an artist based on verbal description of the criminal by the victim. (B) Sketch of the front profile of the criminal created by assembling different strips of a portion of the face bearing the varieties of features as described by the victim such as "big eyes", "pointed nose", "huge lips", "short ears", "wrinkled cheeks", "scalp—full hair", "scalp-bald", "Scalp frontal baldness", etc. Identikit—a likeness of a person's face constructed from descriptions given to police; uses a set of transparencies of various facial features that can be combined to build up a picture of the person sought. (C) Computer generated front profile choosing the specific hairstyle of a criminal as described by victim of assault to the computer graphic expert

Lip Prints (Cheiloscopy)

Lip prints are often used as identification factor in developed countries. However, lip prints as a reliable tool in establishing identity of an individual is yet to be reported. In practice, at the scene of crime, lip prints may be recovered on the wine bottles, glasses, on love letters/ notes, windows, doors, crockery items, cigarette buts etc. Suzuki and Tsuchihastu coined the terminology *figure linearum labiorum rubrorum* for the grooves on the lip prints and classified them in to six types (Table 11.22 and Fig. 11.41).[5,29,30,72]

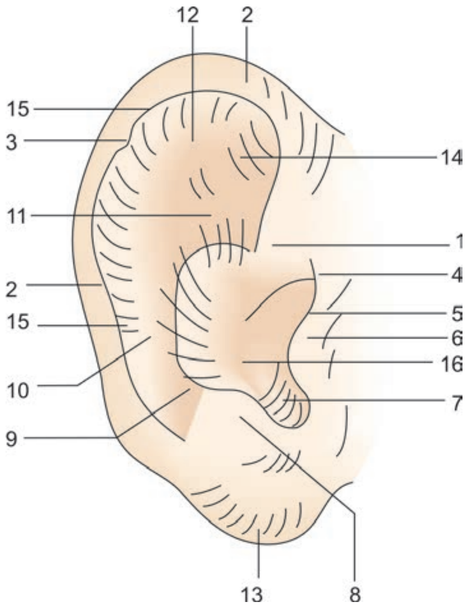


Fig. 11.38A: The external features of the ear. 1. Crus of helix; 2. Helix; 3. Auricular tubercle; 4. Anterior notch; 5. Anterior tubercle; 6. Tragus; 7. Intertragic notch; 8. Antitragus; 9. Posterior auricular furrow; 10. Aanthelix; 11. Lower crus of anthelix; 12. Upper crus of anthelix; 13. Lobule; 14. Triangular fossa; 15. Scaphoid fossa; 16. Concha

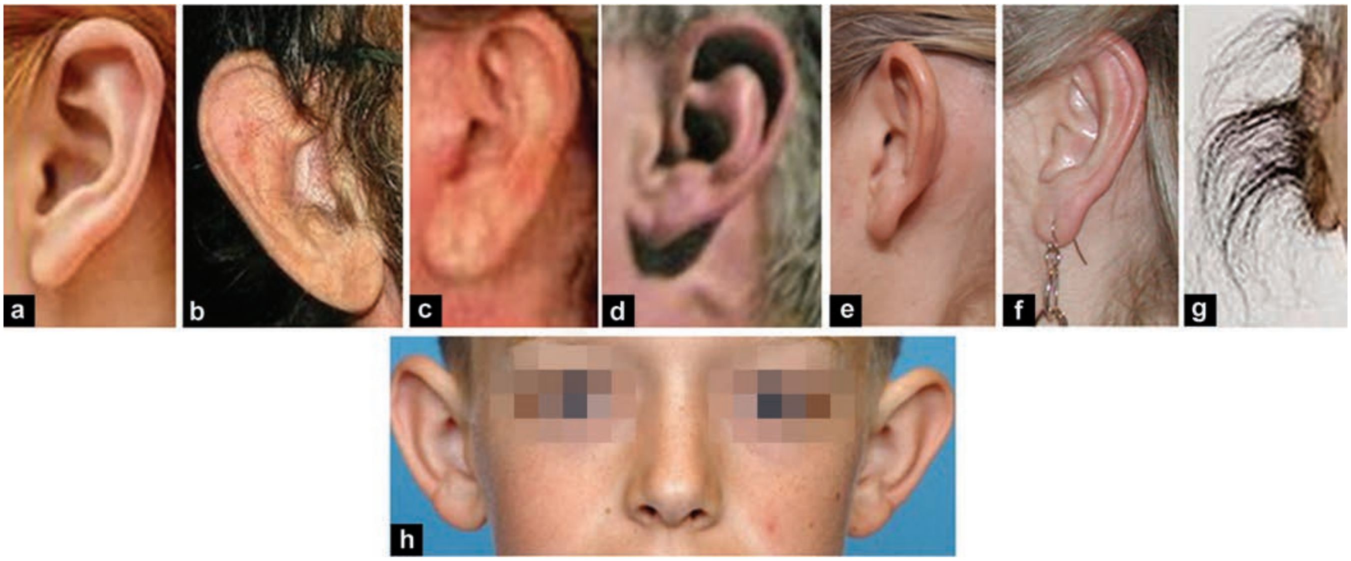


Fig. 11.38B: *Types of ears:* (a) Normal; (b) Broad wing like ear; (c) Ear with wide lobule freely hanging type; (d) Ear with wide lobule fixed to the face; (e and h) Whole ear tending to come forward; (f) Whole ear tending to approach backward; (g) Ear with hairs on the helix and tragus

Electrocardiogram (ECG)

It is been reported that ECG pattern has unique, specific, individualistic characteristics which can help in establishing human identity.[6,63] It has been demonstrated that human identification via the electrocardiogram is feasible and highly effective.[73-75]

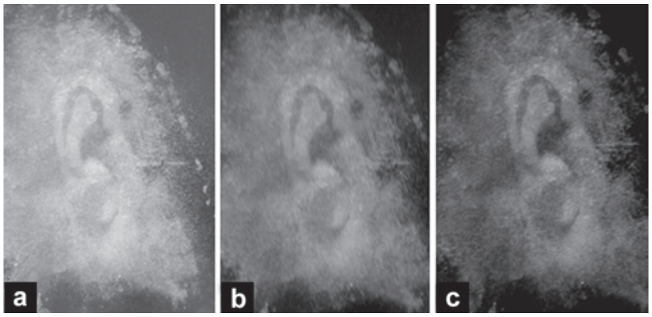


Fig. 11.38C: Experimental latent ear print by a consenting subject, on a glass surface made visible by finger-print powder, showing the effect of gradients of pressure with which the ear was applied to the surfaces: (a) Light pressure, (b) Medium pressure, (c) Heavy pressure. Note the differences in depth of the ear prints

Nail Prints

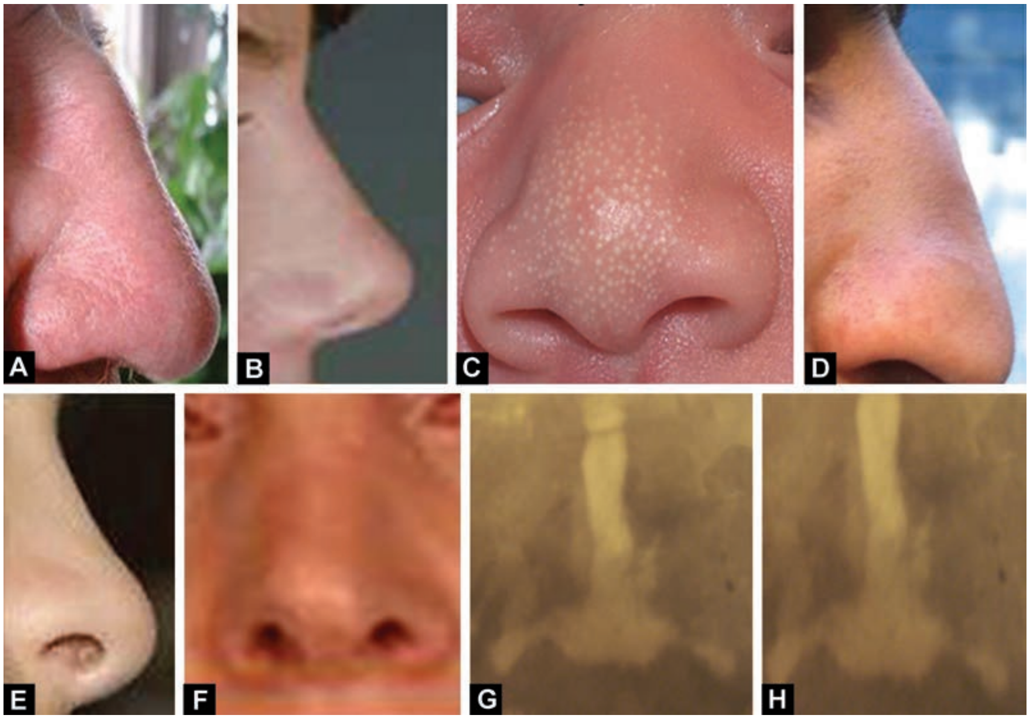
Ridges on the surface of the nails, their number, dimension of each ridge, their depressions and elevations, all in all are of help as unique factors in establishing human identity.[5]

Hairs

Examination of hair is of considerable help in criminal investigation. When a material, alleged to be a hair is given for forensic examination, one may have to establish the following:[1-6,24,25,28]

- Confirm it as a hair
- Confirm it as a human hair
- Site of origin of hair
- Injury to hair
- Singed hair
- Hair and stains on hair
- Hair and identity
- Hair and poisoning
- Hair and miscellaneous information.

Each of these factors is discussed individually below gives projects an idea on how hair can give clues in establishing the human identity.

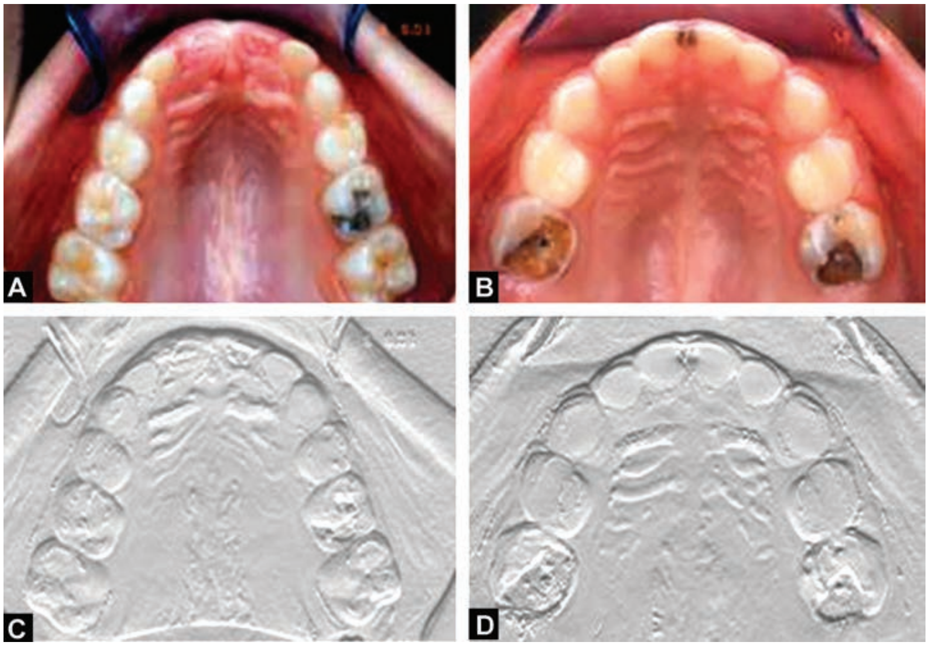


Figs 11.39A to H: *Types of noses:* (A) Aquiline nose; (B) Greek nose; (C) Nubian nose; (D) Jewish or hawk nose; (E) Snub nose; (F) Turn up nose; (G and H) Both are nose prints of types in C and F respectively

Types of nose:

1. *The Roman nose or Hooknose or Aquiline nose:* This type of nose is convex in shape, like a hook. The word aquiline is derived from the Latin word ‘aquilinus’ which means ‘eagle like’.
2. *The Greek nose:* This type of nose is perfectly straight with no curves or hooked like shape. It is known as Greek nose because the great majority of humans, males and females, represented in ancient Greek art, have this type of nose.
3. *The Nubian nose:* This type of nose has wide nostrils. It is generally a little narrow at the top, thick and broad at the middle and wide at the end. The term ‘Nubian’ comes from the ethnic group ‘Nubians’ who belong to northern Sudan.
4. *The Jewish or hawk nose:* The hawk nose is so called because it is very convex, to the extent that it almost looks like a bow. It is very thin and sharp as well. Since it resembles the beak of a Hawk, it is known as the hawk nose.
5. *Snub nose:* This type of nose is quite short in length and is neither sharp, nor hook like nor wide. It is almost as short as a nose possibly can be. Hence, it is known as snub nose.
6. *The turn up nose:* This type of nose is also called as the celestial nose. It is so called because it runs continuously from the eyes towards the tip.

(***Source***: http://www.answers.com/topic/human-nose, **Retrieved on**: 03.08.09)



Figs 11.40A to D: Palatal rugae (Rugoscopy) in two children. Note the high and narrow palate (A) and low and broad palate (B) with comparatively individualistic rugae pattern in each one. Impression of each of these have been taken on POP cast constitutes Palato print (C and D).

Table 11.22: Classification of lip print pattern by Suzuki and Tsuchihastu

Types of lip print	*Description (Fig. 11.41)*
Type I	Clear-cut grooves running vertically over the lips
Type I^1	Partial length grooves of type of variety
Type II	Branched grooves
Type III	Intersected grooves
Type IV	Reticular
Type V	Other patterns (irregular and non-classified pattern)

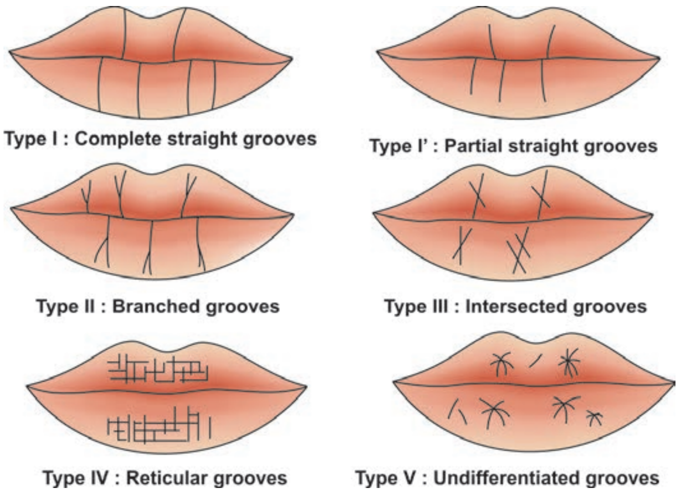

Type I : Complete straight grooves
Type I' : Partial straight grooves
Type II : Branched grooves
Type III : Intersected grooves
Type IV : Reticular grooves
Type V : Undifferentiated grooves

Fig. 11.41: The pattern of lip prints classified by Suzuki and Tsuchihashi[30]

Confirmation of Hair

Confirm first that the material is hair and not any other fiber. To know this, one should know the morphology of hair.

Morphology of hair—Figures 11.42 and 11.43A to E, present the morphology of hair and it comprises of three parts, a bulb/ root, shaft and a tip. Root/bulb is always subcutaneous. The shaft grows from the root through the dermis to skin surface. It consists of three layers: outermost *cuticle*, innermost *medulla*, and *cortex* in between, which is pigmented. These three zones are also seen in the root or bulb. Tip of the hair is the tapering part of the hair and is generally non-medullated.

Confirmation as Human Hair

Usually cotton, nylon, silk, etc. fibers are often mistaken for hair. Hair could also be of animal origin. Hence, it is better to confirm

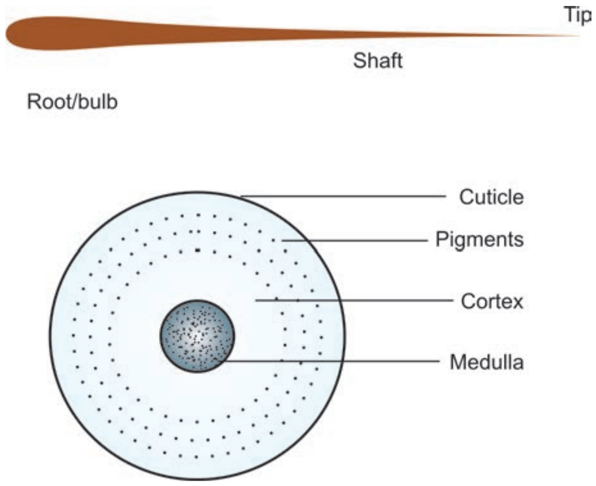

Tip
Shaft
Root/bulb
Cuticle
Pigments
Cortex
Medulla

Fig. 11.42: Line drawing illustrating morphology and cross-section of human hair

the material alleged to be hair, as nothing but hair. This is preferably done by proper gross and microscopic examination, precipitin test and cuticular scale pattern (Fig. 11.44). Table 11.23 enumerates the differences between human and animal hairs.

Site of Origin of Hair

Site of origin of hair is ascertained by comparing the hairs from known site of the body. For example, the scalp hairs are long and slender with a tapering tip in females, while short and cut or chopped in males. Scalp hairs usually emit the smell of the oil used. They may show trace evidence of the dye, if used for colouring. Usually the scalp hair exhibit *splits [fraying]* at the tip due to constant combing or brushing. Hair of the eyebrows are short, thick and sharp. Pubic and axillary hairs are thick and curly. Shape of the cross-section of hair visualized microscopically can give idea about site of hair. Thus, scalp hair has an oval/round shape, while the moustache hair is triangular.

Injury to Hair

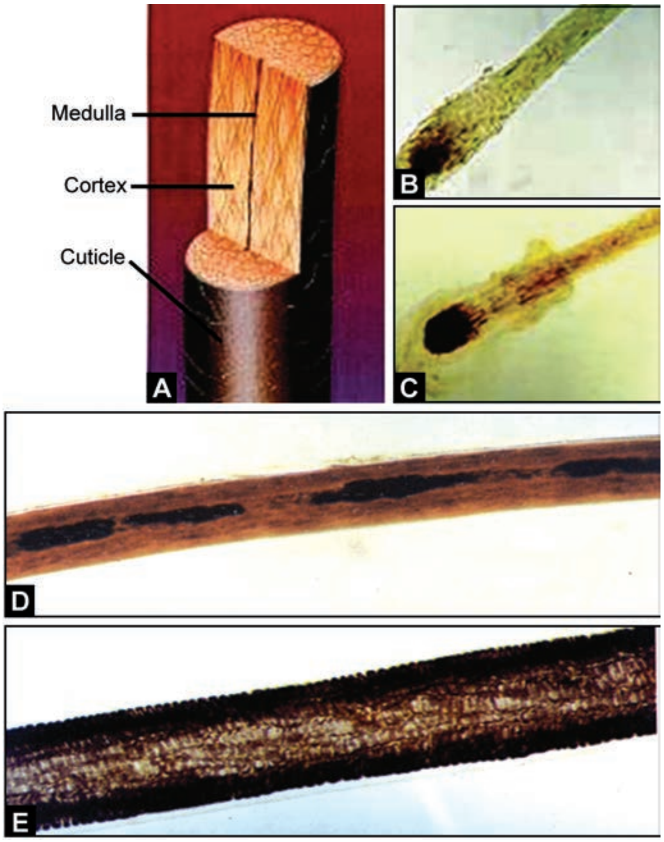
Look for injury on hair by examining under a microscope or by a magnifying lens, from one end to other end. A close examination can yield clues regarding the type of weapon used to produce the injury. Table 11.24 enumerates the type of weapon and the possible injuries sustained in the hair examined.

Singed Hair

A singed hair is peculiar hair showing changes in its morphology due to flame burns or fire arm injury. The singed hairs are grossly — swollen, blackish in colour, fragile, twisted and curly, with carbon particle deposited on its outer surface. It gives a peculiar odour due to burning of keratin. Tip of hair is swollen and resembles the root of hair. *Microscopically* singed hair is wider than normal and with multiple vacuolisation within.

Hair and Stains on it

Hair is often involved in various crimes and can act as good trace evidence in solving most of the crimes. This infers that



Figs 11.43A to E: *Hair:* (A) Human hair structure: Medulla, cortex with pigments and cuticle; (B) Naturally shed hair; (C) Forcibly plucked hair (Note the differences in root / bulbs of both hairs); (D) Longitudinal section of human hair and (E) Animal hairs. (Note the tin and noncontinuous medulla in human hair v/s thick uniform medulla throught the hair)

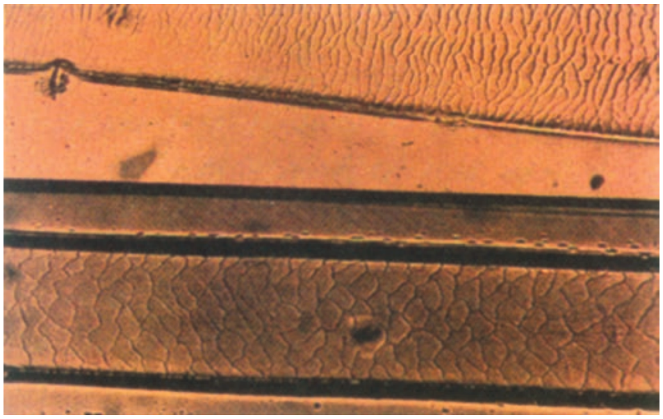


Fig. 11.44: Cuticular scale pattern of hair—small, flat, serrated pattern—human (upper portion) large, polyhedral, wavy pattern—animal (lower portion)

Table 11.23: Differences between human and animal hair

Characteristics	*Human hair*	*Animal hair*
Texture	Fine, thin	Coarse, Thick
Cuticular scale pattern	Small, flat, serrated	Large, polyhedral, wavy
Medulla	Narrow	Wider
Pigments are distributed towards	Cuticle	Medulla
Precipitin test	+ve	–ve

Table 11.24: Type of weapon and possible injuries sustained in the hair examined

Injury on the hair	*Type of Weapon/Injury*
Cut end is clean if seen fresh, but turns round in few days	Sharp weapon
Cut end is crushed/ragged out	Blunt weapon
Bulb is distorted, irregular and swollen if seen fresh	Forcibly plucked hair
Bulb atrophic small and shrunken off with smooth surface	Naturally shed hair

Table 11.25: Various stains on hair and medicolegal information derived

Types of stain	*Medicolegal information derived*
Mud stain	Struggle
Seminal stain	Rape/sexual offences
Blood stains	Trauma/injury
Salivary stains	Asphyxial death
Carbon particles in the stains	Burns/firearm injury
Presence of dyes	Concealing the natural colour or age identity

hair must be carefully looked for the presence of various stains, which provide various information (Table 11.25) of medicolegal significance.

Hair and Poisoning

In organic metallic irritant poisons such as arsenic hair may give some clue. In cases of chronic arsenical poisoning — arsenic may be deposited in the hair and chemical examination may prove the presence of it in the hair.

Hair and Identity

Hair can resist putrefaction and hence forms an important means of establishing identity even in putrefied bodies. Hair also lasts longer and thus constitutes good trace evidence in criminal investigations. Various information in favour of establishing personal identity can be derived from a single hair or hair tuft in the subject when analysed properly by a trained person. Information on deriving *race, age* and *sex* from hair examination is discussed below:

- *Race* — Hair can help in determination of race by its appearance, such as straight hairs of Indian and short curly hairs of a Negroid.
- *Age* — Age of a person can be determined by proper observation for its site of origin and texture. Lanugo hairs are soft, fine, and downy and nonpigmented hairs which cover the body in the prenatal period of the foetus[3,5,28] and thus suggest that they are from a newborn. Pubic hairs grow at 13 years age in female, 14 years in male. Axillary hair grows at 14 years in female, 15 years in male. Greying of hair starts after 40 years. Dyeing of hair is often done for the concealment of graying of hair with aging. Beard hairs are usually first to turn gray and body hairs later.[28]
- *Sex* — Distribution of hair growth on the body is highly significant in establishing the sexual identity of the person.
  - *Distribution of body hairs in males*: Hair in male are often found growing on the pinna of the ears, lips and chin, around the nipples, etc. Pubic hairs have a tendency to grow upwards and merge with abdominal hairs in the body midline above and also grow into perineal and peri-anal regions. Microscopically, rarely/no 'Barr bodies' are seen in the cells of the hair bulb.
  - *Distribution of body hairs in females*: Hair in females are unlikely to be seen growing on body parts other than axilla and pubic region. While the axillary hair show no specific differences than in male, pubic hair present a marked differentiation in its distribution characteristic to females, allowing sex predilection by hairs. They present a specific upper convex border and do not grow further over the abdomen above, but will extend below – on the *perineal* and *peri-anal* regions. Scalp hair in a female are fine and long and gently tapers to an end. Microscopically cells from the hair bulbs show the presence of '*Barr bodies*'.

Miscellaneous Information from Hair

Apart from the facts determined by hair as described above, certain miscellaneous information can also be derived from a proper hair examination.

- *Identifying the weapon/vehicle of crime*: Hair found on suspected weapon or vehicle must be always compared with sample hair (from the victim) to identify and confirm the weapon producing the injury or the vehicle causing accident.
- *In rape/other sexual offenses*: Pubic hair of the victim may be matted with blood and/semen. Proper examination may reveal the presence of pubic hair of the assailant on the victim and vice versa.
- *In bestiality*: Animal hair may be found on the human being and vice versa.
- *Rate of growth of hair* in living is on an average 0.4 mm/day: 0.33-0.35 mm/day for scalp hairs and 0.38 mm/day for beared.[3,5,28] Determination of the time since death by growth of hair after death is a myth. The apparent growth of hair in a dead body is now proven to be mainly due to the rigor of the erector pilorum muscles attached to hair, which causes dimpling/goose skin/orange peal like changes of skin with elevation of cutaneous hair. An additional exploration to this is that the postmortem desiccation or shrinkage of the skin allowing hair to appear more raised and prominent.[25,27-29]

STATURE

Stature is defined as height of a person. The discussion below present certain facts on stature concerning human identity, without which the identity of a human being would be incomplete.[1-6,11,24,25,27-29]

Stature and Diurnal variation of Stature

Stature is proved to be varying at different times of the day, ranging from 1.5 to 5 cm. Stature of person is said to be minimum in the afternoon and in the evening, while maximum in the morning, after sleep.

Factors Affecting Stature Variation

Several factors are explained, but the most convincing and accepted factors are elasticity of intervertebral disks, and muscle tone. Death can bring about body lengthening by about 1.25 cm for a male and 2 cm for a female on an average.

Determination of Stature

When a dead body has been dismembered, the stature from the dismembered parts of the body may be determined approximately by certain mathematical calculations or formulae. Some of these formulae are stated below:

- The length between outstretched fingertips of 2 middle finger is equal to the height (stature)
- Length of one arm × 2 + 30 cm for 2 clavicles + 4 cm for the sternum is equal to the height (stature)
- Vertex to symphysis pubic length = ½ of the height (stature)
- Sternal notch to symphysis pubic length × 3.3 is equal to height (stature)
- Length of entire skeleton +2.5–4 cm (For the soft parts thickness) is equal to the height (stature).

Stature from Skeletal Remains

Stature can also be determined from skeletal bones by mathematical calculations or formula. Usually long bones are ideal for determining stature. There are few formulae, which are often used in routine medicolegal practice and they are:

- Karl Pearson's formula
- Trotter and Glaister's formula

In each of these formulae a *multiplication factor* is derived for various long bones based (Tables 11.26 and 11.27) on the results obtained by dividing average height (stature) of the body by average length of a long bone [measured by a special equipment named *Hepburn Osteometric Board* (Fig. 11.45)] in question, and is presented below:

$$\text{Multiplication factor (MF)} = \frac{\text{Average height (stature) of the body (S)}}{\text{Average length of long bone (l)}}$$

Therefore,

Stature (S) = Average length of long bone (l) × Multiplication factor (MF)

Factors Affecting Multiplication Factor (MF)

The multiplication factor (MF) varies and depends on various factors, which are as follows:

- *Sex*: Varies in male and female
- *Age*: Varies in adults and children
- *Bones*: Varies from one long bone to another long bone
- *Type of bone*: Varies for wet and dry bones
- *Race*: Varies from race to race.

DEFORMITIES

Deformities are excellent means of establishing identification (Figs 11.46A to D) when properly recorded and compared with pre-existing data. Deformities could be:[1-6,23,24,25,27-29]

Table 11.26: Multiplication factor for population in Mysore (Karnataka) in estimating the stature

Bone	*Multiplication factor*	
	Male	*Female*
Humerus	7.08	5.31
Radius	6.01	6.27
Ulna	6.40	6.85
Femur	3.60	3.75
Tibia	4.20	4.39
Fibula	4.44	4.55

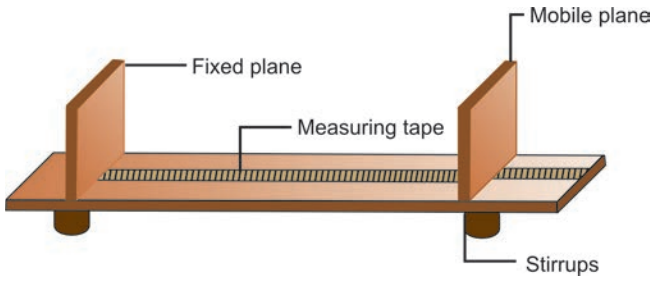
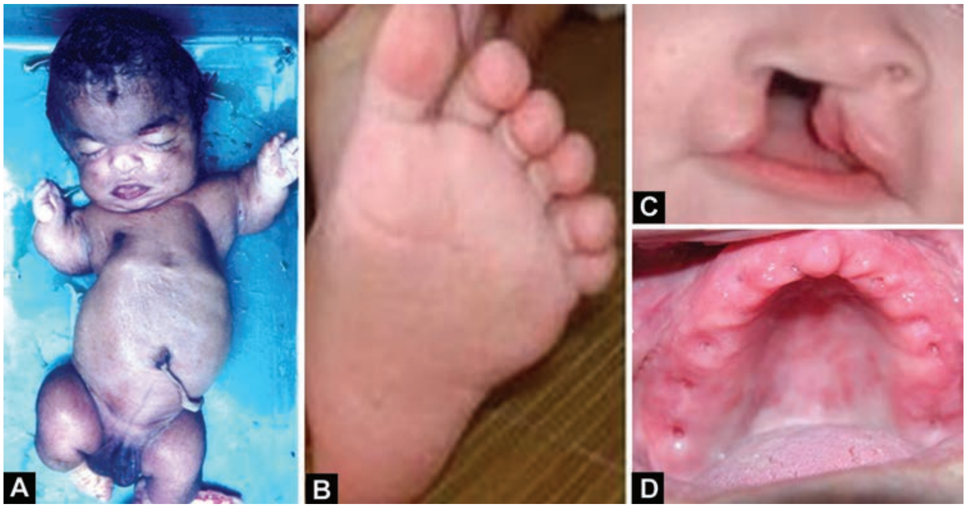


Fig. 11.45: Hepburn osteometric board

Table 11.27: Karl Pearsons formula for adult long bones in dry state for European used in estimating the stature in cms

Bone	*Karl Pearson's Formula [Constant × Length of the bone]*	
	Male	*Female*
Femur	81.306+1.880 × Length of femur	72.884+1.945 × Length of femur
Tibia	78.664+2.376 × Length of tibia	74.774+2.352 × Length of tibia
Humerus	70.641+2.894 × Length of humerus	71.475+2.754 × Length of humerus
Radius	89.925+2.271 × Length of radius	81.224+3.343 × Length of radius

Note—Measures bone length by osteometric board (Fig. 11.45)



Figs 11.46A to D: *Deformities:* Congenital: (A) Congenital deformities in newborn (*Courtesy:* Capt. Dr B Santhakumar, Professor and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai); (B) Congenital polydactylia (6 toes); (C) Congenital cleft lip and palate; Acquired: (D) Edentulous jaw

- *Congenital*, e.g. cleft lip, hare lip, cleft palate, overriding teeth, improper teeth, protruding teeth, moles, birth marks, supernumerary finger/toes (polydactylia), flat feet, webbed feet, etc. Moles and birth marks being often used in routine medical practice as identification marks of an individual (e.g. medical certificates), both are dealt with in detail.

Moles and Naevi

A mole is a small dark spot on the skin (Figs 11.47A to C) can appear at birth or later, anywhere on the body and can be of a variety of shapes and colouration (pink, red, violet, bluish, brown, grey, black, or whitish or otherwise pale). Moles could be raised (papule, plaque), or depressed. Moles are part of the larger group of lesions known as naevi. Technically mole is a timourous growth in the skin known as pigmented naevus or a melanocytic naevus.

Birthmark

A constant mark on the skin anywhere, visible at birth or shortly after birth in a week's time constitutes birthmark (Figs 11.47D and E). It represents accumulation of skin pigment locally, with overgrowth of vessels, or other confined abnormality of the skin. Few birthmarks become pale or fade away partially or completely, in few years, while others persist for life and may transform in to more distinct gradually.

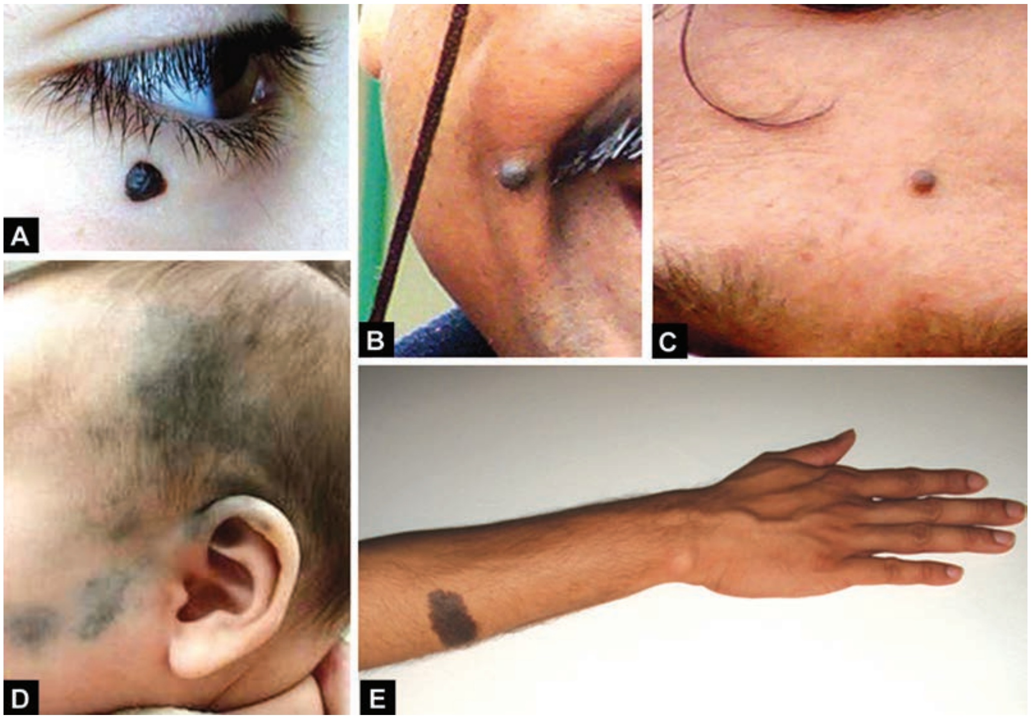
Medicolegal Importance

1. Moles and birthmarks are of great importance in establishing identity of a person. For identification purpose they are described in relation to a nearby anatomical land mark. Usually to be considered as ID mark one has to describe two moles from two different body parts, preferable exposed parts of the body, in one individual for the simple reason that there may be several individuals with similar moles at same spot but rarely there could be two individuals with two moles in similar location.
2. *Removal of the mole*—Techniques of removing the moles include laser treatment, electrocautery technique and surgery. Among the three surgery is the most efficient and effective method of removing the mole completely, irrespective of whether it is superficial or deep rooted in the skin. Mole removal surgery is a very simple method as it allows the surgeon to be sure to remove it as an out patient procedure, where an anesthetic and a freezing solution is applied to the mole, and then shaved it away with a scalpel. Laser treatment and electrocautery technique, are effective only on very flat moles which are on the surface of the skin are both effective only with superficial moles in the surface layers of the skin and not in the deeper parts of skin.
3. Birthmarks are usually painless and harmless, but some may cause complications, such as pressure on other organs, they may be linked with congenital diseases, or, rarely, may transform into a malignancy, which means it is better a birthmark be examined by a doctor. All pigmented skin marks are not birthmarks and may be one has to rule out trauma in such cases.

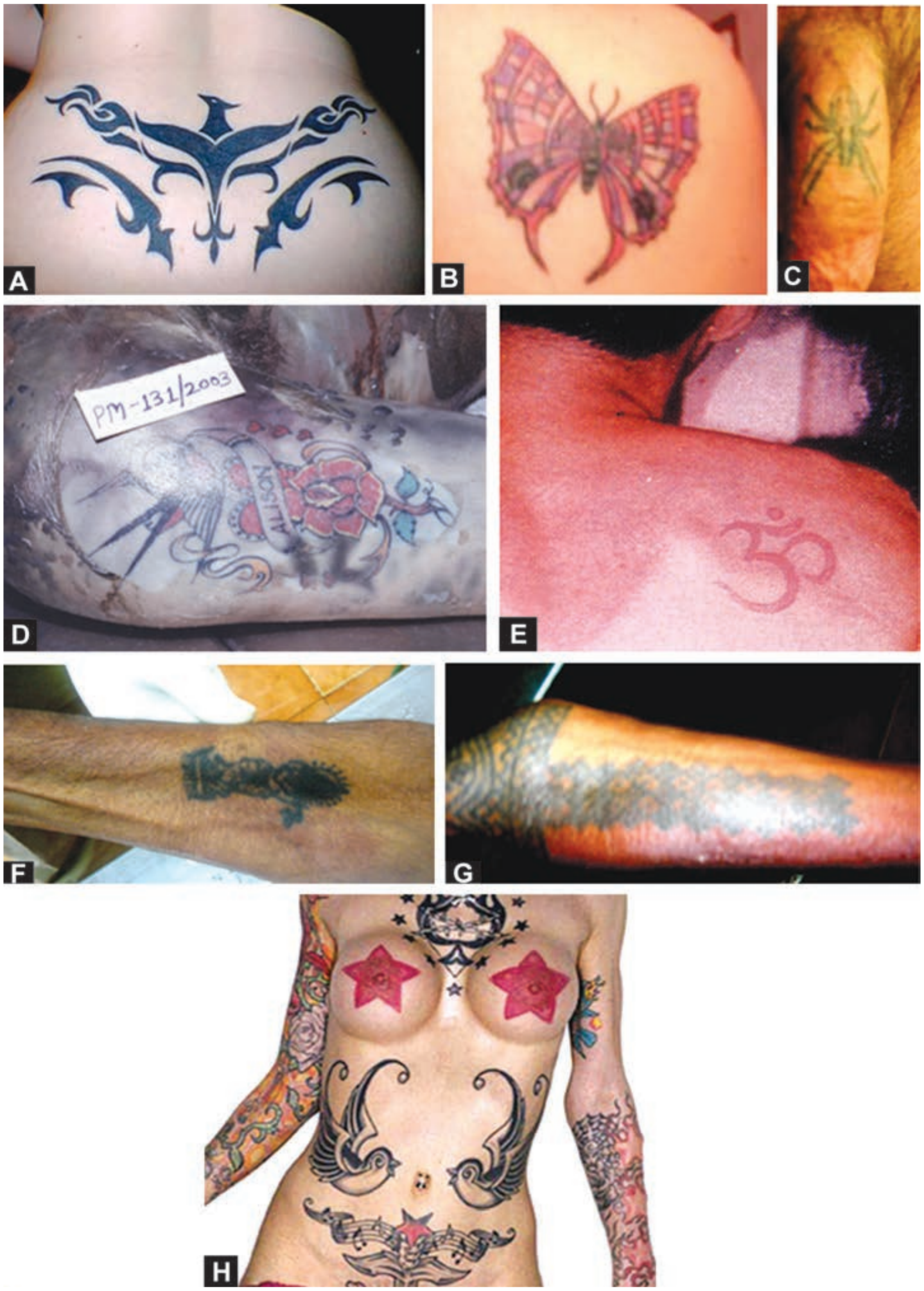
- *Acquired*: For example surgical wounds, amputations, multiple fracture, dental extraction, dental cavities and fillings, circumcised penis, etc.

TATTOO MARK

Tattoo marks (Figs 11.48A to H) are fairly good identification marks, both for living and dead subjects.[1-6,23-24,31-38] These may give wide range of information about the person possessing them. Imprinting pigments of different colours in the dermis by multiple puncture method produces tattoo marks. This may be done mechanically or by electrical device (battery operated). This is practised in all parts of the world.



Figs 11.47:A to E: *Congenital moles and birthmarks:* (A) Black raised mole 1 cm below the right eye; (B) A black mole 2.5 cm away on the right side of the angle of the mouth; (C) A black mole on the mid forehead; (D) Blackish birth mark (naevus) of left side of the face; (E) Brownish black birthmark on right forearm 12 cm above the wrist



Figs 11.48A to H: *Tattoo marks:* (A) On low waist; (B) On the abdomen (*Source:* http://www.tao-of-tattoos.com/butterfly-tattoos.html); (C) Penile tattooing (Source: http://www.tao-of-tattoos.com/images/spider-tattoo-on-penis-21105572.jpg); (D) On the upper arm (*Courtesy:* Prof. SCAD Sapeco, Head, Dept of Forensic Medicine, Goa Medical College, Bambolim, Goa); (E) "Ohm" Tattoo on the back; (F) Tattoo of a Hindu God on forearm (*Courtesy:* Dr Tanuj Kanchan, Asst. professor Dept. of Forensic Medicine, KMC, Mangalore); (G) Design tattoo on forearm; (H) Whole body tattooing (*Source:* http://cajunboyinthecity.blogspot.com/2007_09_01_archive.html)

Pigments Used

Carbon dust, Indian ink, indigo, Chinese black, Prussian blue, cinnabar, cobalt, vermilion, etc. Some of the chemicals contents of different colour pigments used for tattoing are:

- Red pigment – Mercury
- Green pigment – Chromium
- Yellow pigment – Cadmium
- Blue pigment – Cobalt.

Diversity, Alterations and Elimination of Tattoo Marks

Tattoo marks have a great diversity. Tattoo marks mostly represent personal details, details of the favourites/beloved/lover/friend/relatives/pets, place of living, religious beliefs, sexual fantasies, etc. *Alteration* of a tattoo mark is possible. It can be done by over tattooing with titanium oxide or with white pigments. Tattoo marks are fairly permanent, and stay life long if the pigments are placed in dermis and on the covered parts of the body. However tattoo marks may be removed/eliminated. Certain methods by which the tattoos can be erased are enumerated below:

- Surgical skin grafting (*plastic surgery*)
- Local use of *corrosive*
- *Electrolysis* which releases and dissolves the pigments to be washed out;
- Applying *carbon dioxide snow*

- Inflicting injury over the area or by *dermabrasion*
- Application of *caustic substances*
- Exposure to *laser beams*
- *Cryosurgery*, etc.

Making a Faint Tattoo Visible

A faint tatoo may be made visible by either exposure to *ultraviolet* rays or by *rubbing* the suspected area and using magnifying hand lens.

Confirming Tattooing in Attempted Erasure Cases

Biopsy and histological examination of draining lymph nodes, can detect presence of the pigments in them, may help to opine about the erased or removed tattoo, as the pigments used get permanently deposited in the lymph nodes draining the area tattooed, e.g. axillary lymph nodes in a case tattooing over inner aspect of forearm.

Medicolegal Importance

Could give clue about the partial or complete identity of a person by:

- *Designs drawn* can help establishing the identification of race, nationality, occupation, religion, language, name of the person or his/her beloved ones, relatives, friends, etc.
- The designs could be of an idol, obscence figure, a flower, etc. often represent the mental make up, desire, inclination, etc. of an individual
- Identification by tattoos is possible even in a highly decomposed cadaver, as long as the dermis is intact.
- Attempts for concealment of tattoo marks artificially is strongly suggestive of concealment of identity with a positive criminal background.
- A tattooing at times may cause infection (even AIDS), sepsis, ulcer, keloid formation, etc.
- *Medical complications of tattooing:* Tattooing can lead to:
  - Pyogenic infection
    - Superficial infections – impetigo/erythema
    - Deep infections – erysipelas/cellulites
  - Non-pyogenic infections – viral (type B) hepatitis, tetanus, HIV infections
  - Aquired hypersensitivity to pigments
  - Miscellaneous reactions such as keloids, localised sclerodermas, lymphadenitis, etc.
- Drug addicts, especially intravenous drug users, may conceal the site of injection by a tattoo design.
- Those who practice abnormal sex like homosexuals may tattoo a specific design to recognize each other. Usually a '*dove*' on the dorsal aspect of the first web space of the hand is suggestive of such homosexual identity.
- *Tattooing and firearm wounds*: The unburnt gunpowder may also cause tattooing or semi burnt gunpowder particles from a firearm discharge, around the wound of entry, and can give clue regarding the firearm injury and also the range of firing. These are called *involuntary tattoo marks.*
- *Medical and practical applications of tattoing*: Tattoos have a number of legitimate medical applications. Nevi flemmei (port wine stain) have been tattooed for camouflaging it, vitilego patients can be tattooed to almost normal skin colour. Facial tattooing is the latest trend in cosmetic surgery. Correctional tattooing is promoted to get permanent eye lashes and eyebrows etc tattooed in a required style. It is been also used to create nipple and areola after breast surgery. Colour defects in the lips after facial surgery can also be observed by tattooing.

SCARS

Scar is a product of healing of a wound (Fig. 11.49) by fibrosis and cicatrisation. A scar in reality is permanent and may change in its size during the growing ages, but its shape remains unchanged all throughout the life, if there is no keloid formation or any other interference, renders it an important means of establishing human identity.[1-6,24,25,28,29,74]

Medicolegal Importance

- Scars are permanent and they relatively do not change in their shape, position in relation to anatomical landmark of the body; can constitute an ideal factor for establishing identity of a person.

  A scar with *keloid formation* (Fig. 11.50) may not be of any help as an ideal entity for identification purposes. Similarly, scar of vaccination for smallpox, etc. cannot be considered as ideal for identification because of their common appearance in many people.
- Scar can also give clues regarding type of injury, i.e. whether sharp (e.g. Scar of appendicectomy surgery) or blunt force trauma (e.g.: Scar of an unsutured lacerated wound) and thereby the type of weapon used, time since injury, causative weapon, etc.

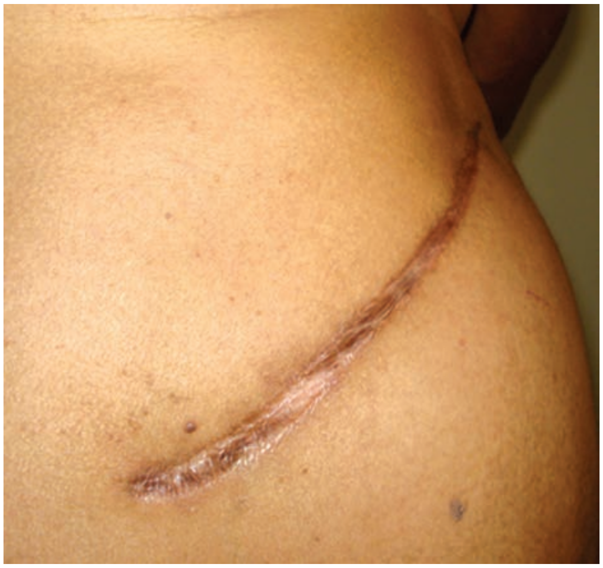


Fig. 11.49: Healed scar of cholecystectomy surgery

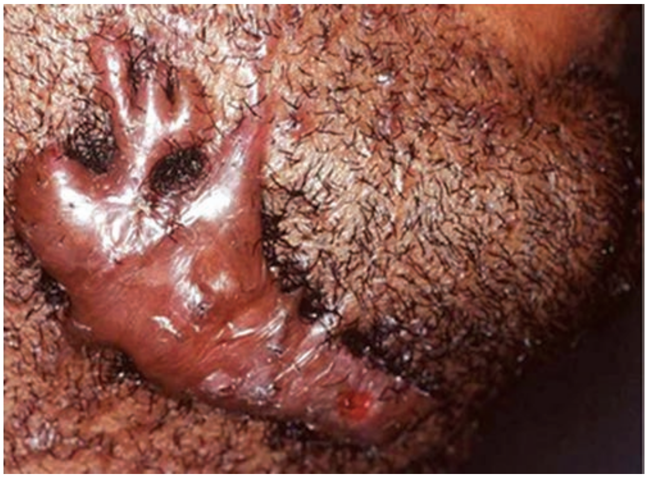


Fig. 11.50: Scar with keloid formation. (*Courtesy:* Dirk M Elston, MD, *Source:* http://emedicine.medscape.com/article/1057599-media)

- A scar on the face (Fig. 11.51A), which is of permanent nature, is a grievous hurt (*Section 320 IPC*).[47]
- *The umbilicus*: It is an iatrogenic/doctor given, permanent that has scar no medicolegal importance.
- Strial albicantes are multiple parallel whitish lines on lower abdomen of a woman who has undergone pregnancy and delivery. These are stretch marks of pregnancy also called strial gravidarum (Fig. 11.51B) and are permanent.
- *Hypertrophic scars and keloids* – Hypertropic scars usually settles or begins to regress in 6 months. However, keloids may extend beyond the wound itself and continue to increase in size by 6 months. Extensor surfaces, strong and burned skin are the commonest site of appearance. Negroes and young people are more often affected.[61]
- A scar over antecubital region or dorsum of hand may be a clue about drug addiction (Figs 11.52 and 11.53).
- *Striae gravidarum* are scars of past pregnancy or tumor in the abdomen.
- Scar on the victim may be claimed as that of an infected wound by the accused.
- Scar may be changed surgically into different shape or completely erased by plastic surgery to avoid implication with a criminal offense.

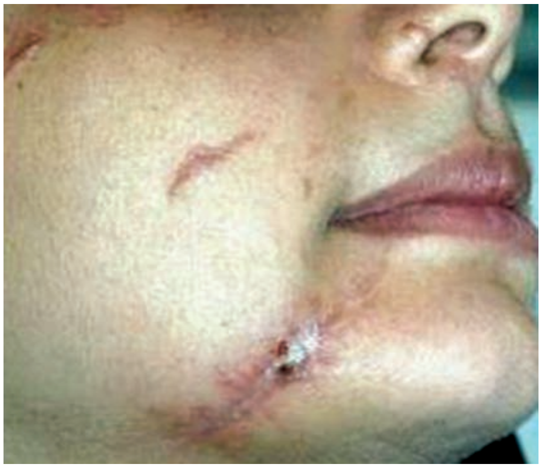


Fig. 11.51A: *Blunt force trauma:* Laceration wound scars on right side of the face

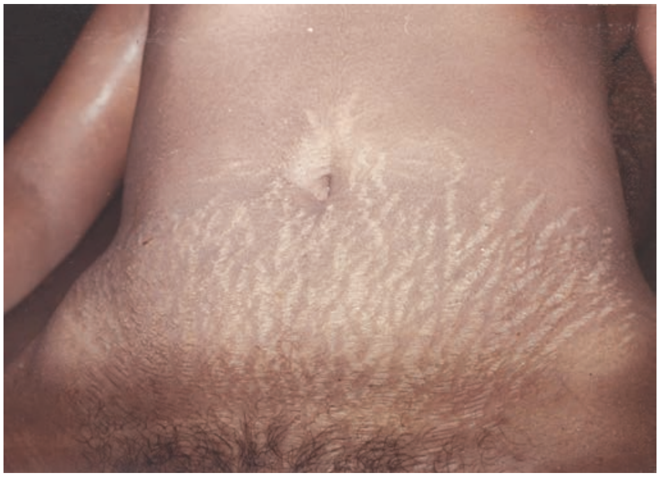


Fig. 11.51B: Striae albicantes—stretch marks seen in pregnancy striae gravidarum (*Courtesy*: Capt. Dr B Santhakumar, Professor and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu, India)

- An apparently invisible scar may be made visible by ultraviolet rays, by application of heat or rubbing.
- A scar may be developed after the removal or erasure of a tattoo or destroy finger-prints to avoid identification.
- Scars being less vascular and fibrous in nature can resist putrefaction and thus help in partial identification of the deceased.
- Scar on the knees should not be considered for identification purpose as they are common in most of the people due to frequent injuring due to falls during the childhood (Fig. 11.54).

ANTHROPOMETRY (BERTILLON'S SYSTEM, BERTILLONAGE)

The science of measuring the body parts constitutes *Bertillon's system or anthropometry*.[1-6,24,28] Historical aspect of anthropometry as a means of establishing human identity is pretty interesting (Fig. 11.55).

Principle

It is assumed that after the age of 21 years the skeleton stops growing and hence the measurements of various parts of the body remain constant. It is also assumed that no two people will have bones of identical dimensions.

Disadvantages

- Only applicable in adults
- Errors in taking measurements by instruments

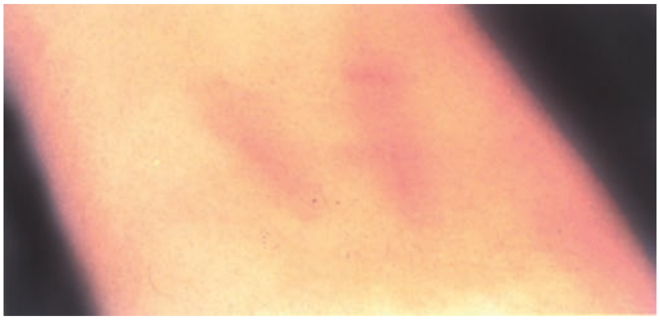


Fig. 11.52: Scar in intravenous drug addict (antecubital region)

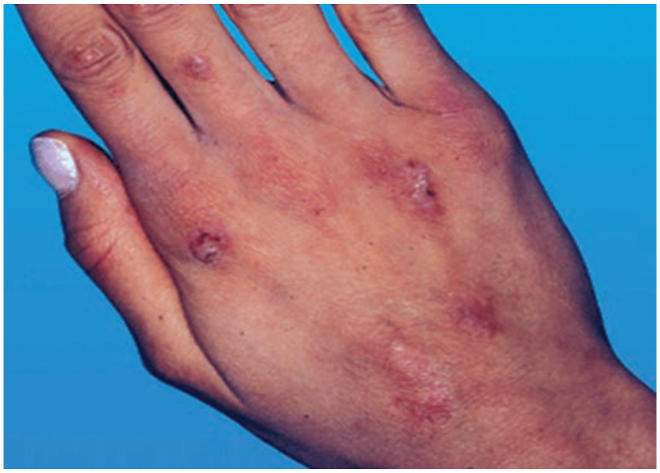


Fig. 11.53: Scars of cocaine shots on dorsum of the right hand in cocaine addict

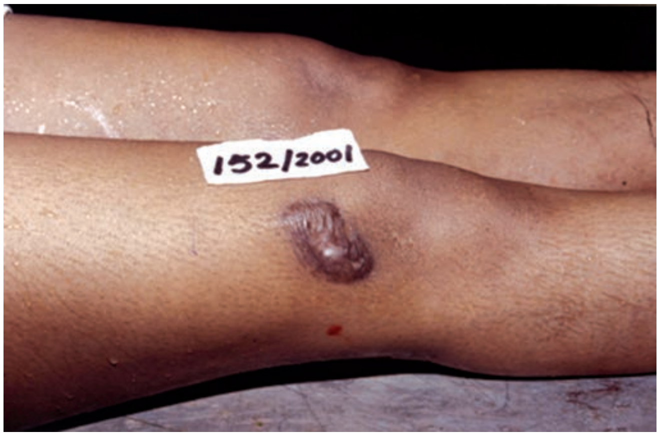

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Fig. 11.54: Scar on the knee joint. (*Courtesy:* Prof SCAD Sapeco, Head, Dept of Forensic Medicine, Goa Medical College, Bambolim, Goa)

- Needs measurements of various parts of the body
- This has been replaced by dactylography technique.

OCCUPATIONAL STIGMA

Large number of occupational stigmas can be of help broadly in determining the job of the deceased, and thereby be of great help to investigating agencies in narrowing down their investigation for identifying the unknown individual, have been described in literature, e.g. cycle rickshaw drivers, tonga/horse cart drivers, sculptors, wind musicians, etc. Presence of grease, paint, dust, etc. on the body and garments worn can give clues regarding the occupation of a person and thereby establish partial identity of a person alive or dead.[1,3,5,6,68]

For example:

- Presence of grease — suggestive of mechanic
- Presence of linear callosities on the fingers of the left hand — violinist, guitarist, etc.

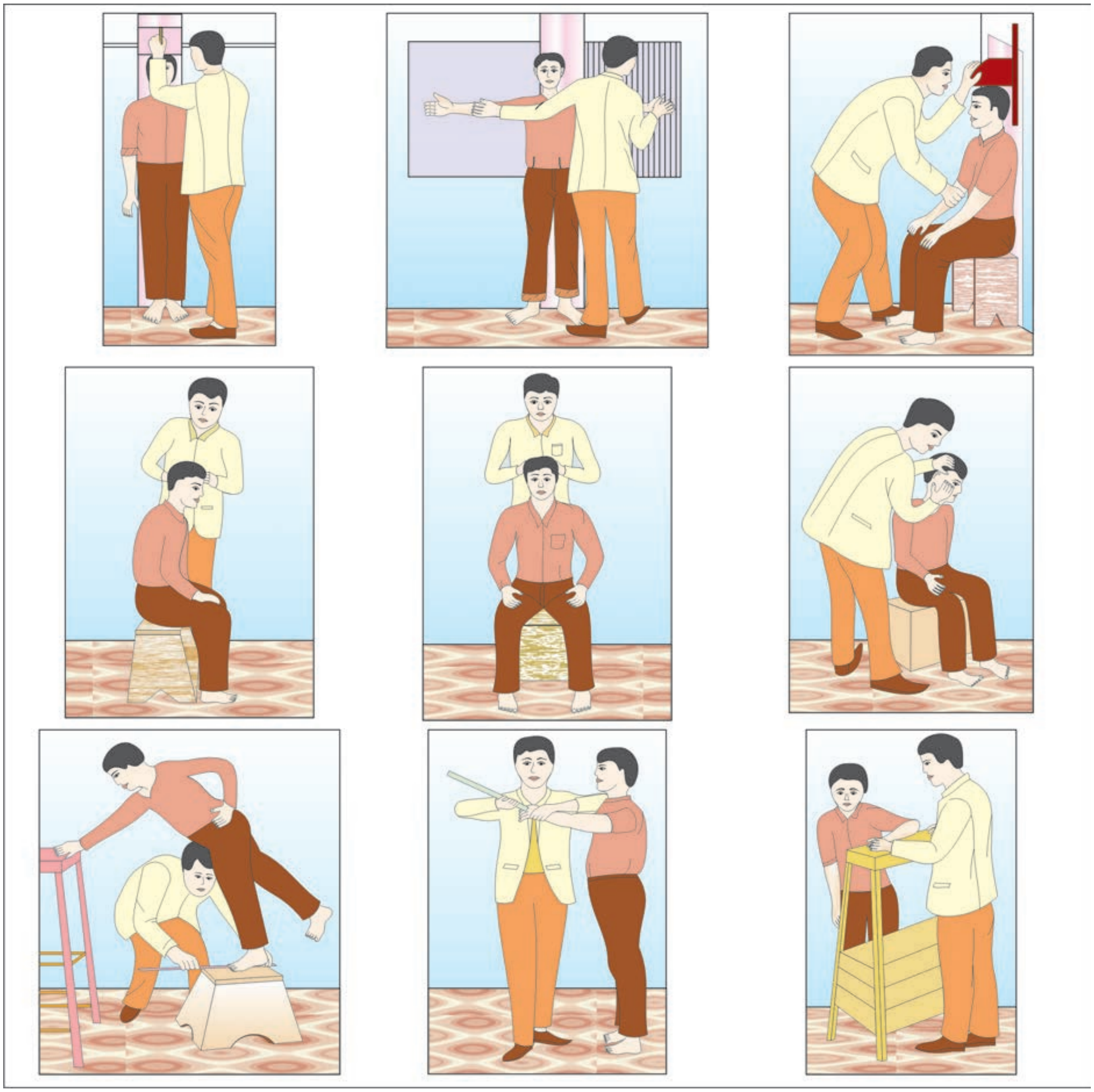


Fig. 11.55: Sketches illustrating Bertillon's system of bodily measurements, called anthropometry, as used in United States in early 1900s. Historical aspect of anthropometry: *Alphonse Bertillon*, a French police officer and an expert introduced the system of recording the detailed physical description including exact body measurements of a subject, birthmarks, moles, colour of the iris, hairs, complexion, etc. as data for establishing identity of a person. This was called *portrait parle*

- *Postural Trauma Callosities* (Table 11.28) in Namazis over the bony areas of the body which are pressed against the ground during '*namaz*' (prayer) have been described (Fig. 11.56). However, in the recent times, the degree of thickening or callositing may not be well appreciated in Namazis who use soft carpet flooring.
- Presence of callosities on the toes (Figs 11.57 and 11.58).

SUPERIMPOSITION

If a photograph of a missing person (front profile) is available and the skull with mandible is both recovered, this technique may be used in ruling out that the skull belongs to the person in the photograph. In a number of instances a skull is produced by the police and the forensic experts are posed with tricky query of whether it could belong to the dead person. There are two methods of identification from skull, the reconstructive and comparative methods. The former aims at determining age, race, sex and as many other criteria of individuality as possible. The comparative method makes use of antemortem records like photographs and X-rays to which the skull may be compared. A refinement of this is the superimposition technique where the life-size photograph of the head of missing person is superimposed on that of the skull under scrutiny. In a number of cases in India and abroad the method has been successfully used to aid in identification of a deceased.[1-3,5,6,10-11,39,41,61,63]

Table 11.28: Distribution of postural trauma callosities in Namazis*

- On the mid-forehead – circular, slightly pigmented and hypoaesthetic
- On the lower knee – elongated and pigmented
- On the lateral malleoli – irregular or circular and pigmented

* *Ordained religious Muslims who offer prayers by kneeling in a particular posture on the floor (see Fig. 11.56).*

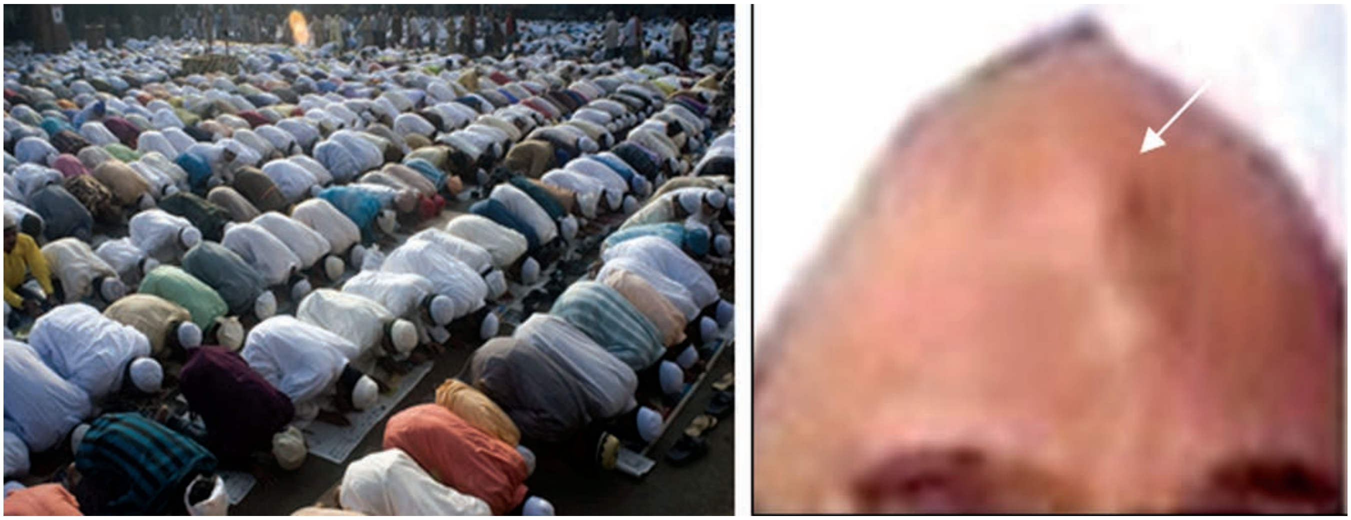


Fig. 11.56: Callosities on the forehead of the Muslims (arrow) due to the special posture opted for prayer (Left side picture represents group prayer by Muslims—Namaz)

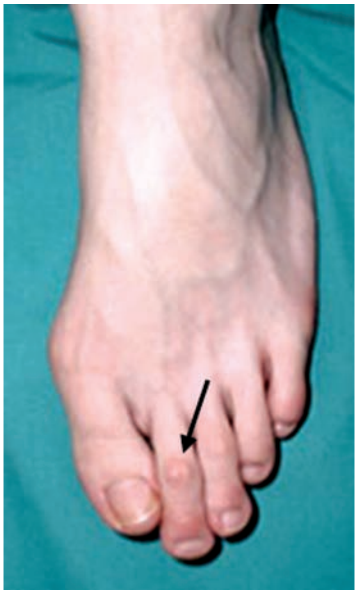


Fig. 11.57: Callosities of the 2nd toe (arrow) by tight shoe

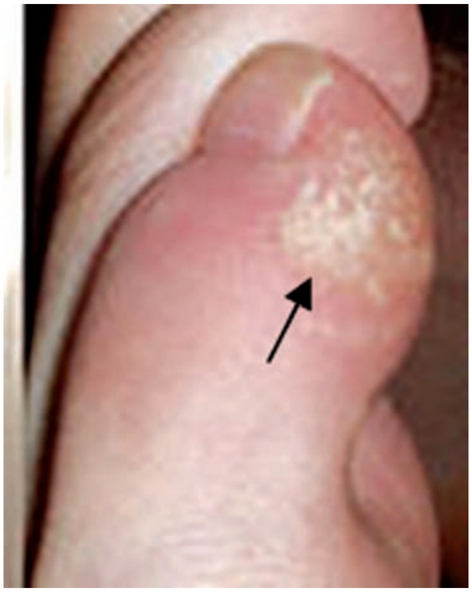


Fig. 11.58: Callosities of the little toes (arrow) by tight shoe

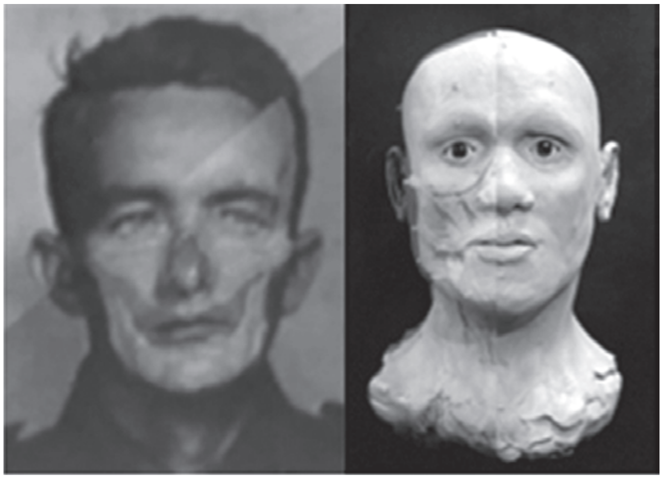
The Technique

The technique comprises of following steps:

- Enlarge the photograph to '*life size*', the enlargement factor being based upon the measurements of the fabric of the deceased, or other measurable items seen in the photograph, or the focal length of camera lenses used, or the dimensions of the anterior teeth, etc. The skull is adjusted in such a way that the inclination and orientation are same as that of the head in the photograph. A life size picture is taken and superimposed on the transparency of life size antemortem photograph, making allowances for soft tissue and hair thickness. The anatomical landmarks are then compared for a positive match. Examine the following characteristics in the superimposed photograph (see the fitting) (Fig. 11.59):
    1. Outlines of skull and mandible fit into the outlines of head and face in portrait.
    2. Vault of the skull to hair line.
    3. Eyes to orbits.
    4. Nasion with origin of nose.
    5. Nasal aperture with nose.
    6. Nasal spine in centre which is little above the nose.
    7. Prosthion in the central line.
    8. Upper border of the upper jaw i.e. below the tip of the nose.
    9. Teeth in relation to lips.
    10. Zygoma over the highlights below the eyes.
    11. Tip of the mandible (Gnathion) with chin.
    12. Position and shape of upper jaw with general contour of cheeks.

Opinion

Pan-American Health Organization (PAHO) and World Health Organisation (WHO) in one of their joint publication on management of the dead bodies of mass disaster victims,[10] highlights the facts on utilizing this technique for establishing personal identity mention that… "*the interpretation of some tests can be misleading. For example, superimposition of skull and ante mortem photographs can only exclude the remains recovered; however sample coincidences in this test do not confirm the identity of the subject…*" Thus, arriving at a positive match does not always mean a positive identification, since photographs of any other person with similar craniometry can produce a successful match with the same skull. As a result, this test can help only in ruling out (exclusion) rather than confirming (conclusion) the identity.



(Left) *Craniofacial video superimposition illustrating an oblique wipe of a transparent skull image on a corresponding facial photograph;* ***(Right)*** *facial approximation, with superimposed right skull half to reveal intermediate steps of muscle construction.*

Left image used with permission from the School of Medical Sciences, The University of Adelaide, Australia. Thanks go to the forensic odontology unit. The University of Adelaide, for the use of equipment to produce this image.

Fig. 11.59: Video superimposition (*Source:* http://www.craniofacialidentification.com/Method_Background.html)

Disadvantages of Superimposition Technique

1. Slight variations in magnification of antemortem photograph or slight change in angulations of the skull from that of photograph can lead to major discrepancies resulting in a mismatch.
2. The method is laborious and time consuming. To avoid these problems use of a *methodical proforma* based approach *(Appendix-A1)* or *video superimposition* technique can greatly improve results minimizing the human errors.

VIDEO SUPERIMPOSITION

When a skull and photograph of deceased are produced for forensic investigation, instead of manual method described above a video graphic technique is adopted here which is discussed below:[39]

Step I

1. The antemortem photograph preferably with smiling face exposing the teeth is now mounted close by.
2. A colour video camera firmly mounted on a tripod is aligned at right angles to the antemortem photograph. The centre of the lens of this camera should be at the same level as the horizontal centre of the photograph.

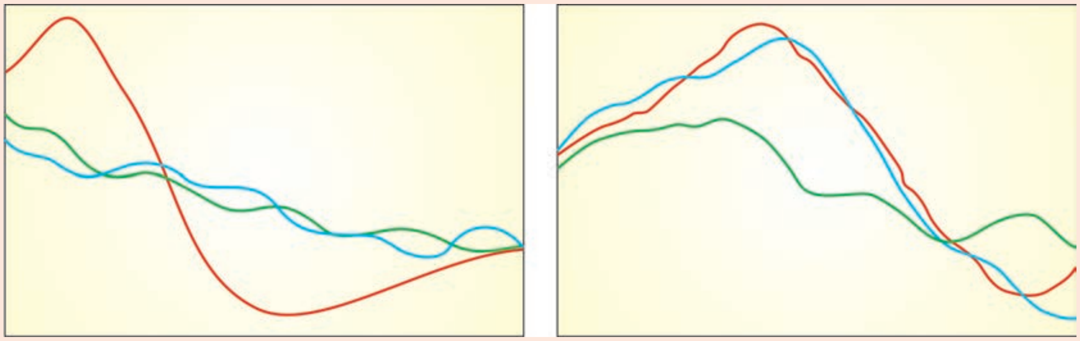
Step II

1. The dry skull without any soft tissue/ any foreign matter on it is mounted on an adjustable support allowing movement in all the three planes.
2. Keep a contrasting background behind the skull.
3. Another colour video camera is firmly mounted on a tripod in a same way as the previous.

Step III

1. The video signals from both cameras are fed into a vision mixer so that a variety of functions like horizontal and vertical wiping, superimposition, and negative simulation can be performed.
2. The skull is oriented on the adjustable mount as closely as possible to the angulations of the head in photograph.
3. The video pictures from both cameras are relayed into the video mixer.
4. By adjusting the mixer, the video picture of photograph can be enlarged. If teeth are visible in skull and photograph, enlargement is carried out till the teeth in antemortem photograph overlap the teeth in the superimposed picture. If teeth are not present/visible in either specimen mounted, the enlargement factor is estimated by adjustment of vertical facial height of photograph to the skull.
5. The video mixer will offer all flexibility here in making the adjustments and provides immediate results without having to process photographs or make tracings.

(A) Dr Lawrence Farwell



(B) Brain finger-printing uses brain waves to test memory. A crime suspect is given words or images in a context that would be known only to police or the person who committed the crime.

How it Works

A suspect is tested by looking at three kinds of information represented by different coloured lines:

- **Red:** Information the suspect is expected to know.
- **Green:** Information not known to suspect.
- **Blue:** Information of the crime that only perpetrator would

Not Guilty

Because the blue and green lines closely correlate, suspect does not have critical knowledge of the crime.

Guilty

Because the blue and red lines closely correlate, suspect has critical knowledge of the crime.

Figs 11.60A and B: Brainwave and brain fingerprinting: (A) Dr Lawrence farwell—founder of brain wave concept, (B) using of brain waves to detect the guilty (*Source:* http://www.brainwavescience.com/SeattlePI.php)

6. After correct enlargement and orientations, superimposition is done, and features are compared with respect to the landmarks.
7. Video mixer can help fading in and out of either pictures, or sweeping over each other in vertical or the horizontal plane.
8. The procedure allows even part of the skull or the photograph to be superimposed in either the vertical or horizontal plane on the video screen so that an accurate match is obtained.
9. The technique can be video taped.
10. Taped material can be used for court purposes also. It may be mentioned here that the postmortem record on *dentition, trabecular pattern and sinus configuration, etc*. in a skull can be compared with antemortem records if available for a comparative study under superimposition technique in establishing human identity.

DNA FINGERPRINTING

Refer to Chapter 12.

BRAIN FINGERPRINTING[69]

Introduction

The truth regarding an incidence that occurred in real life is proved scientifically to be permanently encoded in one's own brain, is been now recorded as a document and made a tool in crime investigation, constitutes *brain fingerprint*.

Dr Lawrence Farwell, who created quite a stir with his patented technique, originally developed brain finger-printing in the early 1990s. Farwell also managed to prove conclusively to the courts, years after a gruesome murder case, that the prime suspect who had been convicted for life since 1977, had in fact not committed the crime. His system has been touted the world over as 100 per cent accurate and is being used by all major investigation services across the globe.

Explanation

The actual experiences of the perpetrator or witness are encoded in a very specific manner in his/her brain. This would be revealed during the course of the EEG (Electroencephalogram) and the carefully formulated probes used to stimulate his brain waves. Probes are the key words or phrases compiled with the help of the investigating officers, act as a trigger for the person under scrutiny. Thus if there was a red shirt on the crime scene that only the investigating policemen know about and the word *'red shirt'* is used as the probe, the subject's brain waves react in a telling manner, reveals the truth.

Medicolegal Significance

- Soon, investigators may ride brain waves to nail the culprits.
- Brain finger-printing v/s Polygraph (Lie Detector) In the polygraph (lie detector) the machine takes note of the subject's suppression or anxiety in divulging information. But with the brain finger-printing we can go beyond that. We can figure out whether a person was actually on the crime scene or not, and what his subjective experience really was. In fact, with all this global terrorism, people are talking of putting gadgets and sensors at all airport doorways, where the brain finger-print with the help of visual stimuli of all passengers would be recorded. So that one can predict if a person with a 'terrorist experience' is boarding the flight and they can at least be called for questioning.
- Ethical dilemma—the scientific research reports needs thinking about its *ethics* of any such a method. This certainly would motivate the intellectuals to think ahead on *the privacy of thoughts*. The Centre for Cognitive Liberty and Ethics has already expressed its suspicions on spurious use of brain finger-printing.
- However, Iowa Supreme Court has already reversed a murder convict Harrington as innocent after 24 years of conviction by brain fingerprinting test reports. Figures 11.60A and B narrate using brainwaves to detect guilt.

TRACE EVIDENCE FACTORS

Biological fluids in medicolegal practice play a vital role, comprising many a times useful scientific evidence in crime investigation. At times it can provide important links in a chain of evidence or support a circumstantial evidence or strengthen weak evidence. It may sometimes furnish conclusive evidence also. It is helpful in cases like murder, traffic accidents, house-breaking, theft, arson, etc. All the branches of science like physics, chemistry, biology, etc. contribute to scientific criminal investigation.

Locard's Principle of Exchange

When two objects collide or come in contact with each other, there is always transfer of materials from one object to the other. Likewise a criminal who commits a crime often leaves some traces at the scene of crime or takes something away from the scene or from the victim. Detection of such evidences constitutes trace evidence, proves the link between the crime and criminal, explains the importance of trace evidence in its medicolegal context.

Basically the investigating officer has to play a vital role in this work, when the trace evidence materials are to be collected from scene of crime. However, it is medicolegal/forensic expert who has to play vital role in collecting trace evidence materials from a living individual or a dead body at mortuary. Examples mentioned below mark the significance of the fact.

Living person: *Seminal stains* in an alleged rape victim, *blood stains* of the victim on the body of the accused in an alleged assault, brought for medical examination.

Dead body: *Salivary dribbling marks* on the front of the dead body in a case of hanging which is suggestive of antemortem hanging.

The facts mentioned above necessitate the need of knowing certain trace evidences of materials of biological origin, which are discussed below.

Blood and bloodstains: Blood is one of the most important body fluid, which easily escapes during any assault, injury, murder, vehicular accident and sexual crimes. Bloodstains are often present on the clothes, weapons, floor, furniture, wheels or part of a vehicle, at the scene of crime, on the victim or the assailant. The examination of bloodstain comprises of three steps:

I. Whether the stain is of blood origin or not.
II. If it is blood, confirm whether human or animal origin.
III. If human, then find out
   - Blood group
   - Sex
   - Age of the stain
   - Arterial or venous origin
   - Whether it is of antemortem or postmortem origin
   - Whether it is of menstrual origin
   - Compare with the victim/assailant's blood,
   - Compare with the blood present on the weapon.

Whether the stain is blood or not: This can be decided *by routine/screening* and *confirmatory tests.*

A. Routine (screening) test includes a biochemical tests namely, *Benzidine test*.

   Benzidine test: Here a negative test is of more importance as it rules out blood, but a positive test is not of significance as vegetable stain, pus, saliva, milk, rust, etc. stains can also give positive reaction with this test needing confirmatory test to prove blood.

B. Confirmatory tests include several tests namely, microchemical tests, microscopic examination, spectroscopic examination, etc.

   i. Microchemical tests—like Haemin crystal (Teichmann's test) or haemochromogen crystal (Takayama test) tests (Figs 11.61 and 11.62). These tests are based on the property of haem (iron) part of haemoglobin to form characteristic coloured crystals with certain reagents, and these crystals can be seen only microscopically.
   ii. Microscopic examination — This is possible only with a fresh stain and one can clearly identify the RBCs and WBCs, which helps in confirming the stain as blood.
   iii. Spectroscopic examination — This is a more reliable test for both old and fresh blood stains. The test needs only a very small quantity of stain. This test depends on property of the translucent coloured fluid to absorb certain rays from the solar spectrum. However, this needs the use of equipment called spectroscope.

Whether human or animal blood: This includes *serological test* namely *precipitin test*—this is a sensitive test based on an antigen-antibody reaction. The human serum contains protein, which forms flocculation with specific antihuman serum. Here a positive reaction is possible with even 1/1000 dilution of sample serum. Gel diffusion, precipitin electrophoresis, antiglobulin consumption test are some of the species-specific tests. A

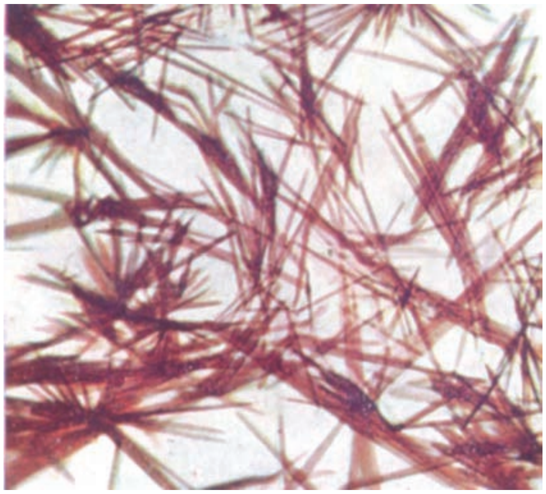


Fig. 11.61: Haemochromogen crystals

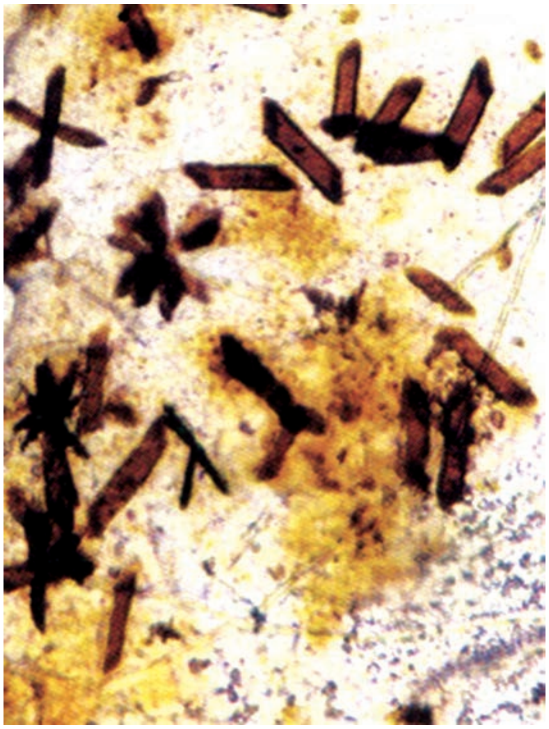


Fig. 11.62: Haemin crystals

peripheral smear of the blood sample may also help, animal RBCs are usually nucleated (Fig. 11.63).

Blood group of the stain — According to the antigens present on the surface of red cell membrane, the blood can be classified into four major groups:

- Group—A
- Group—B
- Group—AB
- Group—O

The plasma contains antibody known as:

- Anti-A
- Anti-B

Red blood cells with antigen A will react with plasma containing *Anti A* antibody and will result in red cells clumping together and this is known as agglutination. Similarly, red blood cells with B group antigen will react with anti B antibody. Both anti A and anti B antibody will agglutinate the red cells containing A and B antigen. The blood in which red cells have neither A or B antigen is called O group. The O group is not agglutinated by either of the antibody. This is the reason for O group blood can be transfused to any person and is known as a *universal donor*.

Similarly AB group is called a *universal recipient*, because the person belonging to this group can receive any group blood, since there are no antibodies in the plasma. If antisera containing *anti A* and *anti B* antibodies are used, with a given sample of blood, the grouping of that sample of blood can be determined by looking for clumping or agglutination (+) under microscope, on adding a drop of antisera to a drop of blood on a glass slide and stirring in the following way.

With this basic knowledge, let us try how blood groups are detected.

Agglutination of RBCs by *anti A serum* reveals that the red cells belong to A group while agglutination with *anti B serum* reveal that the blood is of B group. If agglutination occurs in both antisera, it indicates that the blood is of AB group. If there is no agglutination with anti A and anti B sera, it indicates that the blood is of O group (Table 11.29).

Landsteiner and Wiener in 1940 discovered Rh blood group. If rabbits and guinea pigs are immunized with RBCs of Rhesus monkeys and the resulting antiserum is added to human RBCs, agglutination occurs in a large proportion.

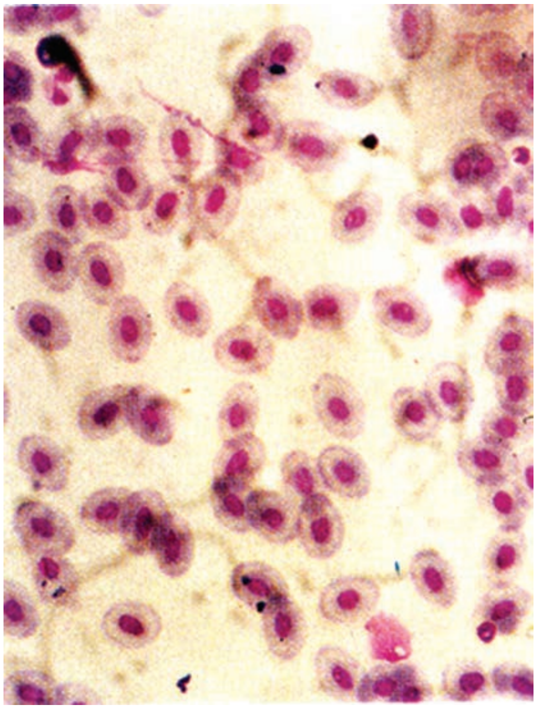


Fig. 11.63: Animal blood smear (Frog blood) with nucleated RBCs

Table 11.29: Showing blood grouping

	A	*B*	*AB*	*O*
Anti A	+	–	+	–
Anti B	–	+	+	–

(+ Agglutination present; – No agglutination)

The red blood cell antigen responsible for this reaction is known as Rh-positive. Those who are not having this antigen in their blood corpuscles are called Rh-negative.

The other blood groups are M, N, S, U, P, K and Duffy.

Sex of the bloodstain: Examination of WBC can help in establishing sex identity.

Sex of the bloodstain can be determined by the examination of leukocytes. The neutrophil contains a drumstick nucleus in females only. This drumstick nucleus is known as *Davidson's body.*

Age of the stain: Colour and nature of the stain can help in establishing age of the stain as follows:

- Blood when fresh it is bright red in colour.
- In 24 hours—it is reddish brown.
- More than 24 hours—it is dark brown and on longer duration it becomes black.
- If the stain is fresh—it is moist and sticky.
- If the stain is old—it is dry and scaly.

Whether arterial or venous blood

- If blood is arterial origin—it is bright red.
- If blood is venous origin—it is dark red.

Shape of the bloodstain and opinions derived (Figs 11.64 and 11.65)

- Circular marks—fall from short height.
- Circular with irregular margins—fall from moderate height.
- Needle-like projections at the margins—fall from a distant range of angles.
- Arterial bleeding is in spurts.
- Venous bleeding is oozing.

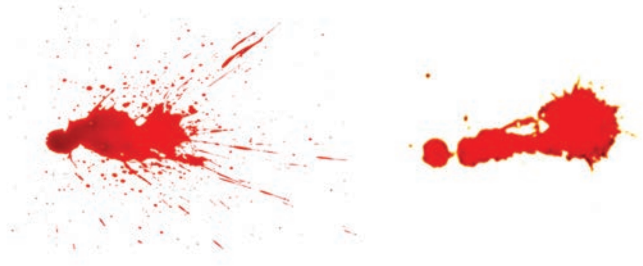


Fig. 11.64: *Blood stain shape and giving opinion:* Spurting arterial blood

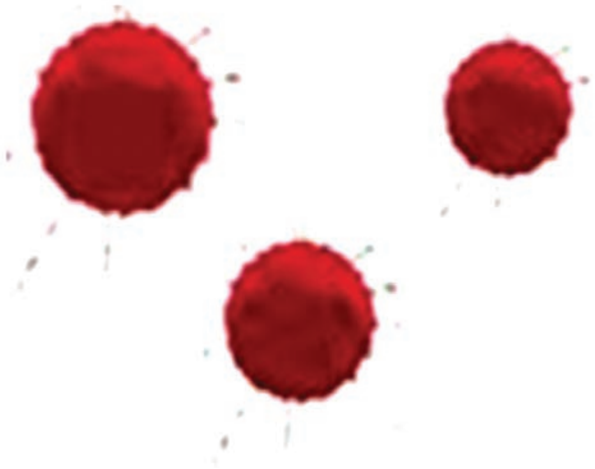


Fig. 11.65: *Blood stain shape and giving opinion:* Venous blood passive bleeding with varies in size and shape and spokes around due to height of fall/dropping

Antemortem or postmortem blood Antemortem bloodstains get broken into scales due to the presence of fibrin, whereas the postmortem bloodstains become powder. The stains, which resemble bloodstains, are paint, iron rust, fruit and vegetable stains and synthetic dyes.

Sources of blood Table 11.30 enumerates the different sources of blood.

Hairs and Fibers

Refer pages 101 to 104.

Semen and seminal stains: Detection of seminal stains is an important aspect in the investigation of cases like rape, sodomy, etc. The male at the climax of sexual intercourse ejaculates semen. About 2 to 5 ml is voided normally each time. Semen consists of sperms and seminal fluid, which is rich in sugars, proteins and enzymes. In *vasectomized* and *sterile persons* semen will not contain sperms.

Appearance: Semen and seminal stains has following characteristics:

- Freshly voided semen will be viscous, dirty white in colour and will have a musty odour.
- In white clothes it produces a light yellow stain with starchy feel on drying.
- In coloured clothes stains are visible only in ultraviolet light. The stains give a blue fluorescence under UV light.

Note: Pus, egg albumin, starch, leucorrhoea discharge, etc. resemble seminal stains.

1. *Microscopic examination:* Semen contains 60 to 150 million sperms per ml. Human spermatozoa will survive (motile) for about 6 to 8 hours at the room temperature. In the vagina sperms may survive up to 24 hours. Dead sperms may be found up to 96 hours in some cases. Detection of spermatozoa in the stain (Fig. 11.66) is an absolute proof of semen. If they are not seen, further tests are to be done.
2. *Chemical tests: Florence test* (Fig. 11.67) and *Berberios test,* (Fig. 11.68) *Acid phosphatase test, etc.* are based on the reaction of certain reagents with various constituents of semen. If they are positive, species-specific test *(precipitin test)* can be done to confirm human origin.
    - *Precipitin test:* Specific antiserum is made to react with the seminal fluid. A positive test will confirm human origin.
    - *Grouping:* Semen can be grouped using the anti A and anti B sera used for blood grouping *(group secretor test).*

Table 11.30: Blood and its sources

Source	*Appearance*
Arterial	Bright red
Venous	Dark red
Nasal bleeding	Blood mixed with nasal mucous and hair.
Stomach or gastric bleeding	Chocolate colour due to the presence of acid, haematin and is acidic in reaction
Menstrual bleeding	Dark coloured fluid blood with foul smell and with endometrial debris, acidic reaction, vaginal epithelial cells and bacteria are present
Abortion	Dark clotted blood. Endometrial and placental debris with foetal remnants sometimes present.

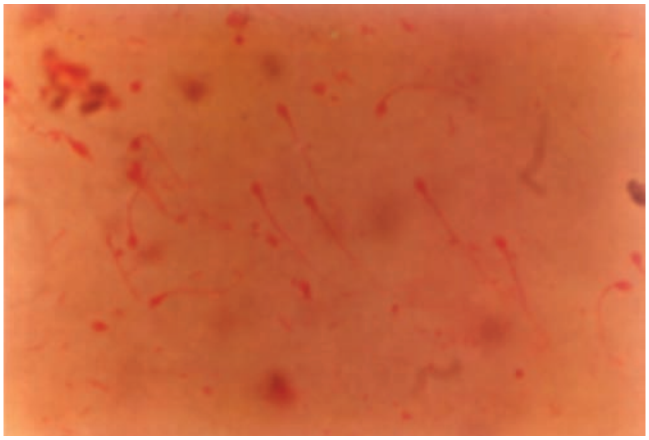


Fig. 11.66: Vaginal smear showing spermatozoa (microphotograph) (*Courtesy:* Dr Uday Pal Singh, KMC, Warangal, AP)

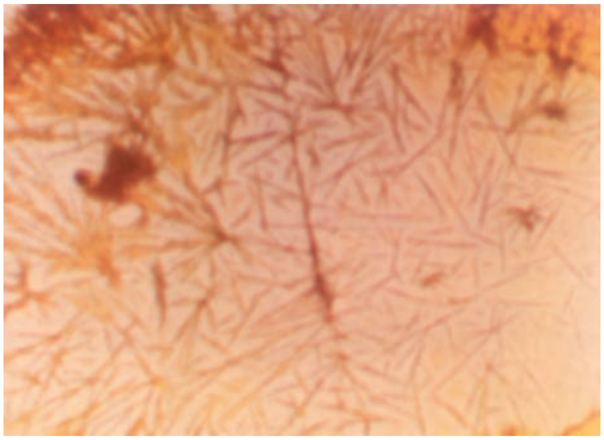


Fig. 11.68: Berberios test—sperminpicrate crystals (microphotograph) (*Courtesy:* Dr Uday Pal Singh, KMC, Warangal, AP)

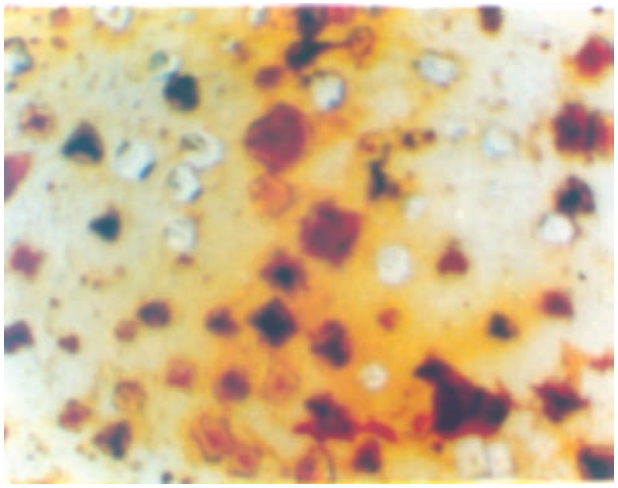


Fig. 11.67: Florence test—choline iodide crystals (microphotograph) (*Courtesy:* Dr Uday Pal Singh, KMC, Warangal, AP)

Saliva and salivary stains: Saliva is the secretion from the salivary glands situated intra- and extraorally. The secretion flows through ducts from the glands opening into the mouth. Detection of salivary stains is important in cases of *strangulation, hanging, smothering, sexual offenses, theft, etc.*

Composition of saliva: Saliva consists of water, salts and various enzymes. Amylase is the important enzyme present in saliva.

1. *Test for salivary stain:* Detection of the enzyme salivary amylase is the basis of tests for salivary stains. Amylase converts starch into dextrose and glucose. Tests based on this principle are employed with suspected stains. Stains are also examined microscopically for the presence of buccal mucosal cells.
2. Species-specific tests *(precipitin test)* and grouping *(group secretor test)* can also be done with salivary stains.

Urine and urinary stains can be detected by chemical tests. Grouping is also possible because urine contains group specific substances. However, *species-specific tests (precipitin test)* cannot be done using the proteins in the urine.

Faeces and faecal stains can be identified by microscopic examination for the presence of undigested matter. Feces contain stercobilin, which can be detected by chemical tests. Grouping can be done using absorption elusion method. However, *species-specific tests (precipitin test)* cannot be done with feces.

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Chapter 12

Forensic DNA Profiling

INTRODUCTION

A fundamental requirement in any criminal prosecution is that a crime be proved beyond reasonable doubt and in trying to meet this stringent requirement forensic practitioners draw upon a variety of scientific disciplines. Forensic science has now entered an era in which increasingly sophisticated technology is not only desirable but also necessary in combating crime and ensuring justice.

In the last two decades advances in molecular biology in particular, have created new means of resolving forensic questions and of identifying offenders with greater speed and certainty. Though the latest technology has helped in identification in the criminal cases, it has also been employed for civil purposes especially in determination of paternity in litigation involving custodial and marital matters. Thus forensic DNA analysis has diverse applications. This chapter is a review of forensic DNA analysis as it is applied today in the forensic laboratories around the world.

FORENSIC GENETICS

The foundations of forensic genetics were laid down when Karl Landsteiner described ABO blood group systems in 1901.[1-4] Following this, the detection of other red cell antigens, serum proteins and erythrocyte enzymes made the serological analyses of blood and other body fluids possible. By 1980 a battery of conventional blood grouping tests were available which considerably improved the forensic utility especially when used in conjunction with the white blood cell antigen system, HLA. However the techniques involved were laborious, technically complicated and utilised expensive sera. Further, the systems were not uniformly distributed in all the tissues and underwent rapid decomposition on drying. This caused impediments in investigations as the forensic specimens are mostly minute in size and undergo weather effects like drying and aging. Further the power of discrimination of these markers was very low and a large number had to be analysed before having some comfortable results.[2-4]

In 1985 a breakthrough came when *Sir Alec Jeffrey's* of United Kingdom described that a set of DNA markers called Variable Number of Tandem Repeats (VNTR) were much more variable among humans and these were immediately applied to forensic cases pertaining to human identification.[1,5]

DEOXYRIBOSE NUCLEIC ACID

DNA is the biological blue print of life. The structure of deoxyribonucleic acid (DNA) was described by James Watson and Francis Crick in 1953.[1,5-8] DNA was determined to be a right-handed double helix. DNA is composed of repeating subunits called nucleotides. Nucleotides are further composed of a phosphate group, a sugar, and a nitrogenous base. Four different bases are found in DNA: adenine (A), guanine (G), cytosine (C), and thymine (T). In their common structural configurations, A and T form two hydrogen bonds while C and G form three hydrogen bonds between them. Because of the specificity of base pairing, the two strands of DNA are said to be complementary. This characteristic makes DNA unique and capable of transmitting genetic information (Fig. 12.1).

The genetic makeup of every individual established at the time of conception is unique.[5] It defines that individual's genetic characteristics contains a large number of polymorphisms that can be used for human identification. Thus a number of new techniques based on DNA polymorphisms have been developed (Fig. 12.2).

Majority of DNA is located in the nucleus organised in the form of chromosomes (nuclear or genomic DNA). Nuclear DNA is inherited equally from both mother and father. With the exception of identical twins, no two people share the same genomic DNA sequence.[1,5]

Another source of DNA is found in the mitochondria of cells. Mitochondrial DNA is inherited only from the mother, and therefore it can be used to match with the maternal lineages.[1]

Advantages of Using DNA for Identification[5-13]

There are important advantages of using DNA for identification purposes:

- DNA is ubiquitous i.e. it is present in all the nucleated cells of the body.
- The DNA makeup of a person is same in all the cells of the body and cannot be altered.
- DNA of every person (except in monozygotic twins) is unique in its profile.
- DNA can be extracted from all body fluids and all the tissues of the body.
- In post-mortem cases DNA can be obtained from body tissues.
- In cases where the body has been buried, DNA can still be obtained from body tissues.
- In burnt and charred remains, DNA can be obtained from hard tissues like bones and teeth.
- DNA can be stored in small quantities easily as compared to other evidentiary material.
- DNA can be stored for very long periods of time without deterioration if stored appropriately.

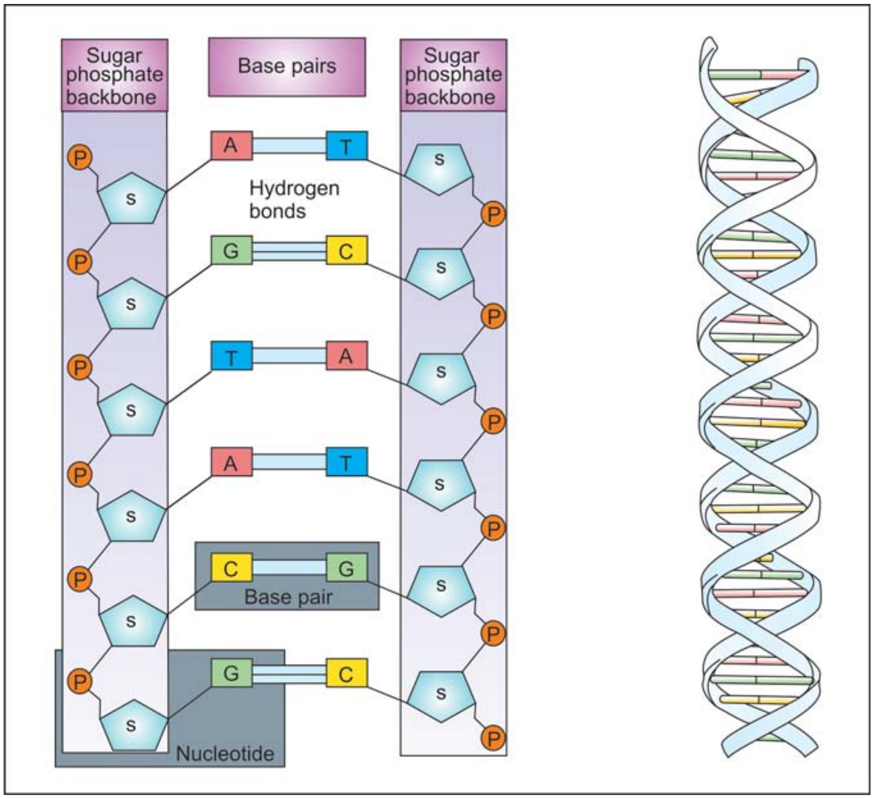


Fig. 12.1: *DNA Structure:* The figure illustrates the double helix and explains the concept of base pairing and nucleotide. The DNA back bone consists of phosphate groups here shown on extremes of the figure as letter P. The hexagons show the sugar deoxyribose. A, T, G and C show the base adenine, thymine, guanine and cytosine. The DNA strands can separate when the temperature is raised or in the presence of chemical like formamide (*Courtesy:* Human Genome Database, USA)

- DNA test detects the genetic makeup whereas blood or protein tests are the genetic products.
- DNA methods avoid any complications of dominance or recessives, e.g., one cannot differentiate between blood groups AA or Aa, however by using DNA analysis this difference can be detected.
- DNA does not combine and thus can detect the number of persons at the crime scene if they have contributed to it.

Disadvantages

DNA analysis depends on the presence of nucleated cells. Therefore it is not possible with biological specimens wherein these cells are absent (or scarce). Such specimens include semen lacking spermatozoa (as in vasectomized males), hair shafts, etc.[1]

THE BASICS OF MOLECULAR BIOLOGY[1-2,12-16]

In order to understand forensic genetics, a basic knowledge in molecular biology is essential and it is suggested to learn about the DNA structure and the organisation of human genome. There are 3 billion base pairs (bp) in a single copy of human genome. These are arranged in compact structures, which we all know as chromosomes. There are 23 pairs of chromosomes in all the cells of humans and so are called diploid cells. Only in the gametes (ova and spermatozoa) one copy of each chromosome is present and these are called haploid cells. Thus in humans (and other animals), one chromosome is derived from each parent at fertilisation (the time of fusion of the spermatozoa and the ovum). The DNA in the chromosomes is arranged as coding and non-coding regions. Only a small part (~5%) of the human genome consists of genes encoding for proteins. This part of human genome is same in all the humans with certain exceptions and is responsible for normality of biological processes in the human body. The genes also have functional portions called *'introns'* and non-functional regions in between the introns called *'exons'*. So a lot of the genome appears to have little functional value (Fig. 12.3).

Polymorphic markers have been detected in these areas of human genome and these are called loci. So the markers exist in different forms of sequences and the alternative forms of the marker at a particular locus are called 'alleles' of that locus. In an individual there are usually two alleles (one at each chromosome) of a locus. If these alleles are same, the person is a 'homozygote', if they are different then the person is a 'heterozygote'. The allelic configuration at a locus is called the genotype of the person at that locus. The genotypes (at different loci) are called the profile of the person at those loci. Considering the amount of genetic variation it is not surprising that no two humans except monozygotic twins are alike at the level of DNA. More over analysing the whole genome is not routinely possible to differentiate between two individuals, various highly polymorphic loci of DNA are used. The study of these markers is the basis of forensic genetics. Out of the whole array of such markers the tandemly repeated (repeating side by side) DNA sequences are the ones mostly applied to forensic genetics. These have been exploited for human identification as they exhibit a high degree of variability in the number of the core repeats among different individuals.

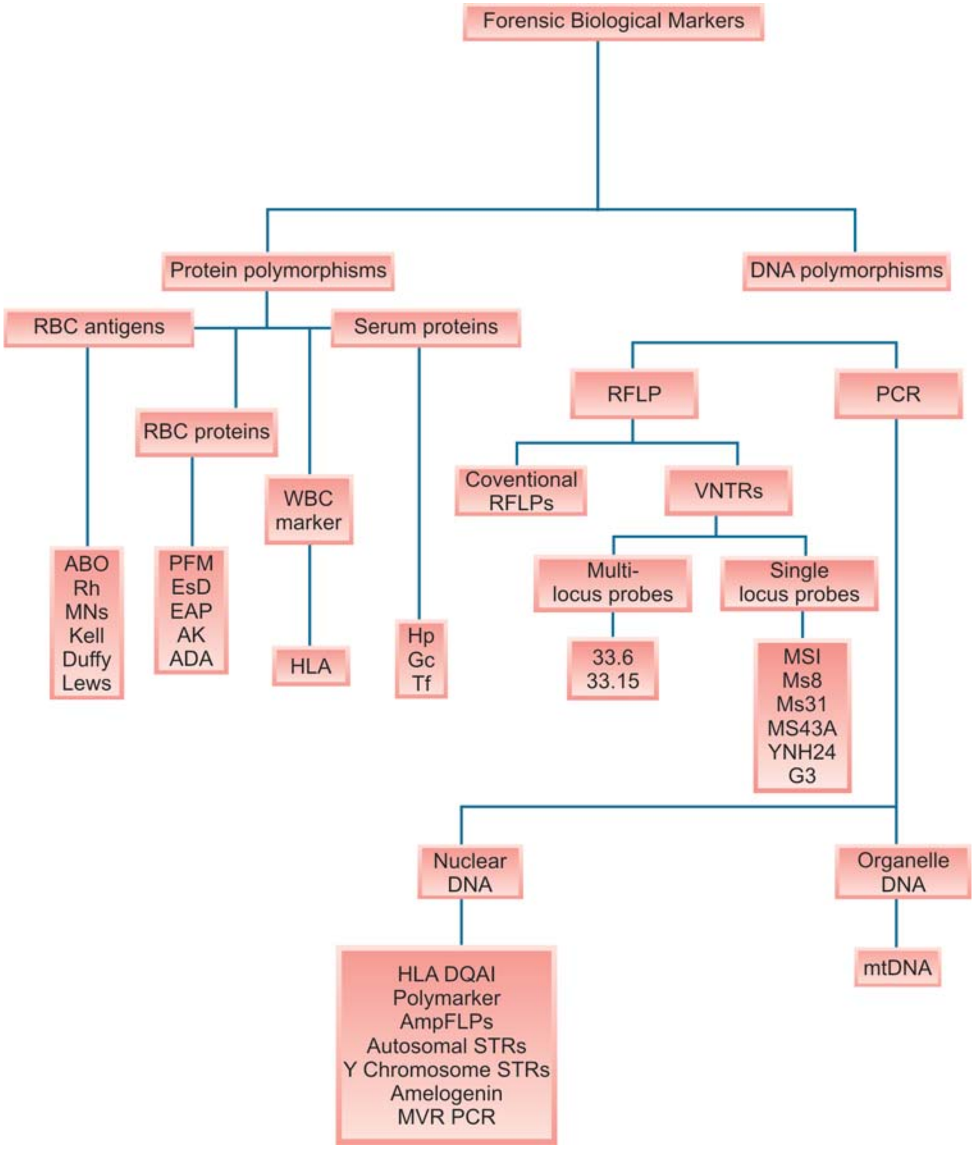

Forensic Biological Markers
Protein polymorphisms
DNA polymorphisms
RBC antigens
Serum proteins
RBC proteins
WBC marker
ABO
Rh
MNs
Kell
Duffy
Lews
PFM
EsD
EAP
AK
ADA
HLA
Hp
Gc
Tf
RFLP
PCR
Coventional RFLPs
VNTRs
Multi-locus probes
Single locus probes
33.6
33.15
MSI
Ms8
Ms31
MS43A
YNH24
G3
Nuclear DNA
Organelle DNA
HLA DQAI
Polymarker
AmpFLPs
Autosomal STRs
Y Chromosome STRs
Amelogenin
MVR PCR
mtDNA

Fig. 12.2: Protein and DNA polymorphisms used for human identification

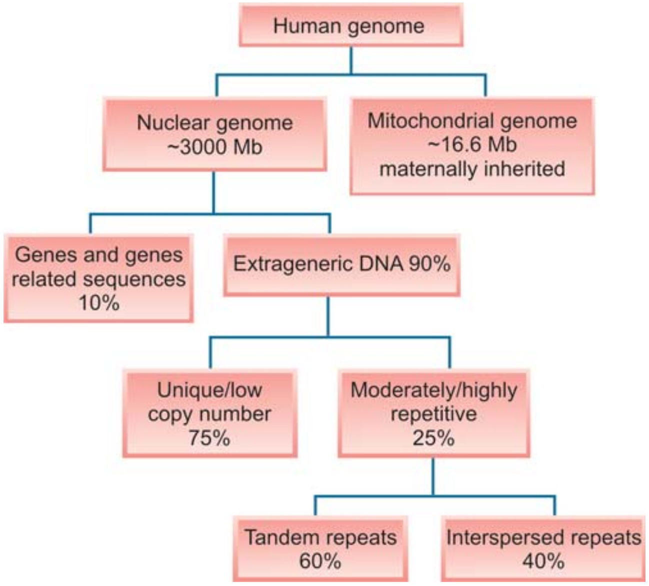

Human genome
Nuclear genome ~3000 Mb
Mitochondrial genome ~16.6 Mb maternally inherited
Genes and genes related sequences 10%
Extrageneric DNA 90%
Unique/low copy number 75%
Moderately/highly repetitive 25%
Tandem repeats 60%
Interspersed repeats 40%

Fig. 12.3: A schematic diagram showing the organisation of the human genome

Extraction, Detection and Visualisation of DNA

The extraction of DNA is now a relatively easy process. Basically the nucleated cells are burst open using osmotic pressure, the cellular proteins are precipitated first and the clear fluid left contains DNA that is precipitated with isopropanolol. Then it is cleaned by washing in ethanol and diluted in a buffer medium or pure water. A number of commercial firms offer good kits for DNA extraction and one can easily follow the manufacturer's instructions. After having extracted DNA from the cells or tissues it has to be visualized. Performing *agarose gel* or *polycrylamide gel* electrophoresis in a buffer system is done traditionally. If the DNA concentration is high it can be easily visualized on an agarose gel using ethidium bromide, which is a florescent dye that intercalates between the bases of DNA. Dye bound to DNA displays increased florescence. However, if the DNA concentration is low then a polyacrylamide gel is required which is thinner and the DNA can be visualized using ethidium bromide staining.

Following ethidium staining many other stains were followed but for very small concentrations of DNA, a more efficient method was required, the breakthrough came with fluorescent dyes which can be attached to the DNA segment to be visualised, but detection requires more sophisticated equipment like charge coupled device (CCD) camera. This camera is incorporated in the automated DNA sequencers and captured the fluorescence of DNA molecules that were labelled with a dye when such molecules were exposed to a laser beam. The images of the camera were then transferred to a software, which converts the light into peaks of different colours, and this is called the electropherogram. This became the standard method in today's automated DNA sequencers. An advancement of these has been in the type of medium used for electrophoresis. In the latest DNA sequencers instead of a slab gel of polyacrylamide, electrophoresis is carried out in a thin capillary which is a very fast method of separation of DNA markers. Further these are user friendly as gels are not required.

TANDEMLY REPETITIVE DNA

Segments of DNA are arranged as a particular sequence being repeated for more than once, thus a sequence GGGCCCTTAA might be repeated many number of times, and such DNA is called tandemly repetitive DNA. Depending on the size, these are divided into three groups and discussed ahead.[16-40]

Minisatellites

Certain minisatellite polymorphisms studied contained a large number of tandemly repeating units of a particular sequence (core repeat) typically 16-80 bp long and were termed *'variable number of tandem repeats'* or *VNTRs*. Several other highly variable regions were subsequently discovered near the human insulin, a-related globin genes, and the c-r-Ha-ras-1 oncogene. In each case the polymorphism resulted from differences in the number of core repeat.

In 1985 Prof. Sir Alec Jeffrey's described a hypervariable minisatellite using multilocus probe technique (MLP) and suggested its utility for human identification.[22-23] The multi locus system of Jeffrey's had a very high power of discrimination due to the number of alleles at a locus but MLP profiles were difficult to interpret and especially difficult to standardize. Therefore several single locus probe systems (SLPs) were developed which detect two alleles in a heterozygous individual; one from each parent, and this became the preferred method for DNA profiling (Fig. 12.4). The process of DNA profiling by RFLP technology required a large amount of DNA and time as it included gel electrophoresis and Southern blotting process followed by probing of the membranes. RFLP was thus not suitable for automation and therefore many laboratories could not utilize this method efficiently. The details of the method, as of now are almost obsolete. RFLP technology needs at least 50 ng of intact high molecular weight DNA, in contrast, PCR based testing which use as little as 500 pg.[1]

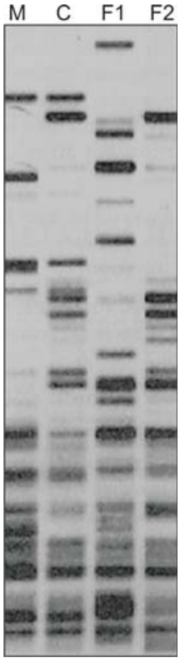


Fig. 12.4: Multilocus probed membrane showing the profile of mother, child and two putative fathers. Note how discriminating the bands in the membrane are and how they exclude one (F1) while include the other (F2) as the true father of the child. At the same time it is difficult to assign the alleles

Polymerase Chain Reaction (PCR)[24]

In 1985 a revolutionary advance in molecular genetics came with the discovery of a simple and powerful technique, the Polymerase Chain Reaction (PCR). DNA replicates itself in the cell and the process has been studied in great detail. Usually the two strands of DNA are wrapped around each other, for replication the DNA has to unwind to have a single strand available for the synthesis of the new strand. This process is called denaturation of DNA and can be achieved by heating the DNA to a high temperature so that the hydrogen bonds between the nucleotides are broken down. The new nucleotides then are inserted at complementary positions by enzymatic action and a new strand is synthesized. Polymerase Chain Reaction is an in vitro molecular photocopying process that generates millions of copies of the target DNA sequence the boundaries of which are defined by synthetic oligonucleotide primers that are complementary to the 3' (called 3 prime) ends of the sequence (Fig. 12.5). The PCR consists of replication of DNA segment with the help of an enzymatic reaction which apart from enzyme contains special buffers, synthetic nucleotides and primers which are small stretches of DNA that anneal to the denatured DNA at complementary positions and the new DNA is synthesized. During this process the DNA is denatured by increasing the temperature to near boiling, then the temperature is decreased till it is optimal for the primers to anneal to the complementary positions on the DNA and again it is raised to allow the Taq enzyme to include the deoxynucleotides on the new strand after the primer. This cycle of denaturation, annealing and extension is repeated a number of times (usually from 30 to 50) to obtain millions of copies of a desired segment of DNA in few hours. The amplicons generated are sufficient in quantity to be analysed by various techniques. PCR thus overcomes the usual problems of a forensic sample, like small quantity, environmental degradation and contamination. The reaction is run in special machines called thermal cyclers (Fig. 12.6). PCR thus was recognized for its application to forensic genetics at a very early stage as it yielded a lot of DNA from minute samples, like saliva on a postal stamp.

However, VNTRs had very long alleles so they were not amplified easily. The scientific community then described smaller minisatellite loci which had alleles from 9-15 bp. These are called AmpFLPs and are composed of short core repeat units. Good examples are DIS80 and ApoB loci. Unlike VNTR loci, most of the AmpFLPs could be successfully amplified.

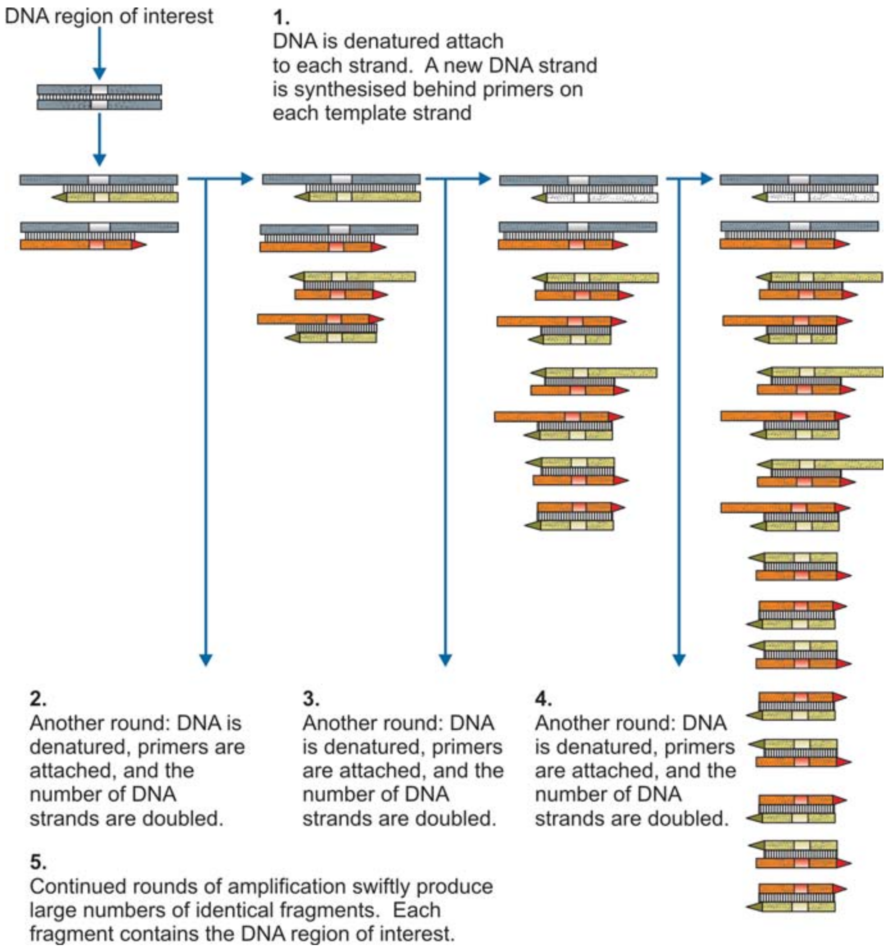

DNA region of interest
1.
DNA is denatured attach to each strand. A new DNA strand is synthesised behind primers on each template strand
2.
Another round: DNA is denatured, primers are attached, and the number of DNA strands are doubled.
3.
Another round: DNA is denatured, primers are attached, and the number of DNA strands are doubled.
4.
Another round: DNA is denatured, primers are attached, and the number of DNA strands are doubled.
5.
Continued rounds of amplification swiftly produce large numbers of identical fragments. Each fragment contains the DNA region of interest.

Fig. 12.5: A schematic diagram showing the process of polymerase chain reaction. Orange and green arrows show the forward and reverse primers respectively (*Courtesy:* Human genome database)

Microsatellites[25-27]

Microsatellites, also known as short tandem repeats (STRs), are an abundant class of DNA polymorphisms. Occurring every 300 to 500 kb in the human genome, these markers have a repeat unit of 1-6 bp in length and are highly polymorphic. These loci could be easily amplified using PCR as the size of the amplification product was 100-500bp and have become the most widely utilized markers for forensic DNA profiling. Currently STRs form the basis of forensic DNA analysis so we would discuss these in some detail followed by Y chromosome specific STRs and mitochondrial DNA analysis.

SHORT TANDEM REPEAT DNA PROFILING[28-32]

As the STRs emerged as important forensic tools for human identification, studies were conducted that showed that the human STR loci selected for forensic use had no non specific reactions from microorganisms or various substrates and offered unprecedented advantages in the context of human identification. It was shown that the STR loci could be sized accurately using fluorescent technology as well as by non fluorescent methods like silver staining of the polyacrylamide gels. Perhaps the greatest advantage of using STRs was the ability to amplify a number of them together in a single PCR. This is achieved by adding more than one primer pair for different target DNA sequences. Each primer pair would amplify a specific sequence or STR. This is called multiplex PCR. This is not an easy reaction to optimize, however once optimized it increases the throughput and one is able to glean an enormous amount of information from very minute DNA samples as is the case in many forensic evidentiary material. For forensic purposes the first multiplex PCR kit was developed by the Forensic Science Services (FSS) of UK and it comprised of four STR loci, THO1,

Fig. 12.6: A modern thermal cycler capable of performing 96 PCRs at a time. The model shown is ABI 9700

Box 12.1: CODIS STR Loci

TPOX, CSFIPO, D5S818, D13S317, D16S539, THO1, D18S51, D7S820, VWA, FGA D3S1358, D8S1179, D21S11 and Amelogenin.

SGM+ Loci Used by FSS UK:
D3S1358, VWA, D16S539, D2S1338, D8S1179, D21S11, D18S51, D19S433, THO1, FGA and Amelogenin (See Fig. 12.7).

vWA, FES/FPS and F13A1 (Box 12.1).[30] This had a match probability (probability of match of a DNA profile i.e. genotype comprising of all the four STR loci to that of a random individual in the population) of 1 in 10,000. The FSS then launched its second generation multiplex (SGM) comprising of six STR loci THO1, FGA, D8S1179, D18S51 and D21S11. SGM provided with a match probability of 1 in 50 million which was satisfactory for the population of UK but for larger populations like US or India the forensic geneticists required a bigger multiplex kit with higher discrimination capacity. Two commercial firms PE Applied Biosystems and Promega came forward with their own multiplex kits with exceedingly high power of discrimination (Fig. 12.7). In USA the core loci used for the CODIS system comprise of 13 loci. All the multiplex STR kits also include a STR amelogenin for gender discrimination. The amelogenin locus is present on the X as well as the Y chromosome at homologous positions. However, on the X chromosome there is a 6 bp deletion of the target sequence. Thus the PCR generates two products (or bands or peaks) for a male and only one product (band or peak) for the female.

CLASSIFICATION OF STRs

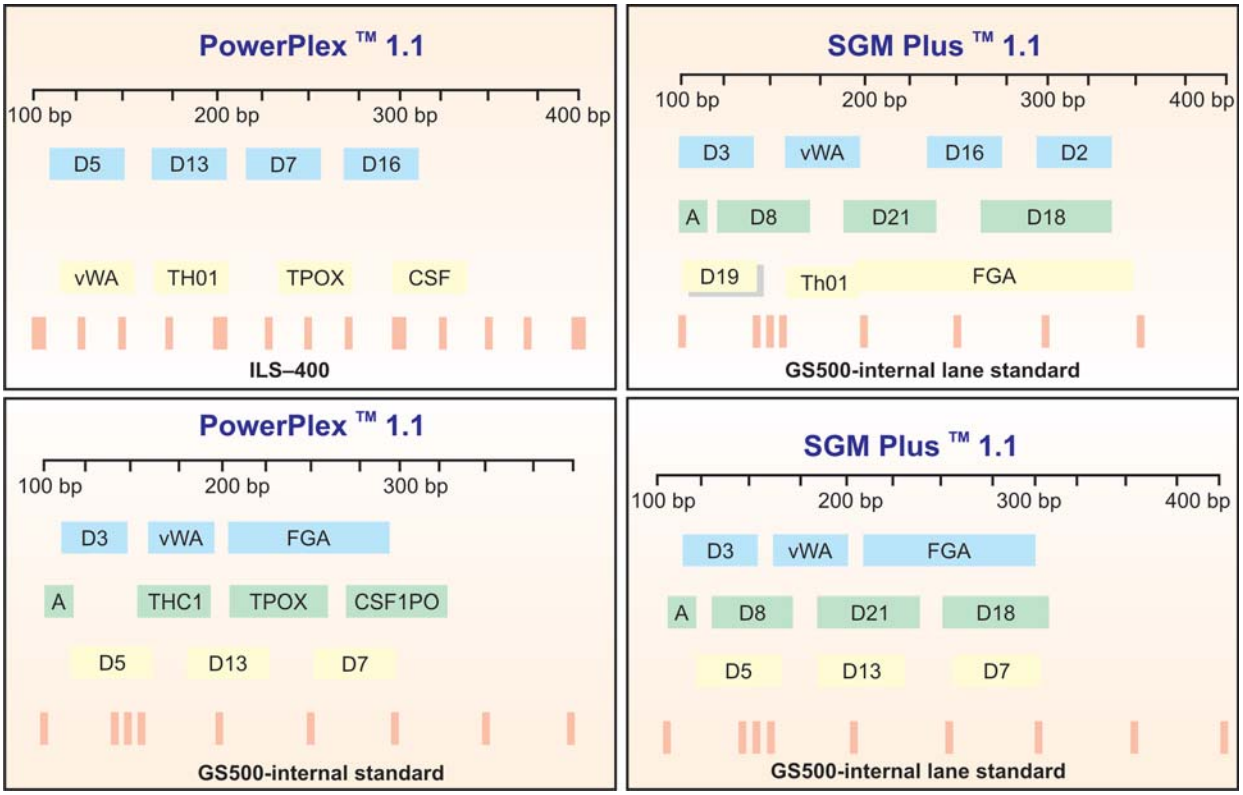
STRs can be classified on the basis of length of the repeat unit so there are di, tri, tetra and penta nucleotide STRs, depending on the repeat unit size of two, three, four or five nucleotides. The forensic community has generally settled for tetranucleotide STRs though some tri and penta nucleotides are also used. However, the important classification of STRs is that where the alleles configuration is taken into consideration and that is important as the world forensic community needs to have universally accepted and followed nomenclature.

There are several types of STRs, and they are classified as shown in Box 12.2.

Mostly the alleles of STRs are easily distinguished from each other and the sizes can be measured in different ways. Here we can pause and look at the strategies which the molecular biologists use to separate fragments of DNA.

STR MUTATIONAL MECHANISMS

The forensic value of STRs is due to their high levels of polymorphisms. These polymorphisms are in part due to the process of mutation occurring in these genomic sequences which generates different alleles at a locus. In most cases the mutations arising during DNA replication are corrected by the repair of enzymatic processes. However, sometimes, *'slipped strand mispairing'* leads to mutation in regions of the genome, having abundant simple repetitive sequences which have been recognised as the major mechanism involved in their generation.



PowerPlex ™ 1.1
100 bp
200 bp
300 bp
400 bp
D5
D13
D7
D16
vWA
TH01
TPOX
CSF
ILS–400
SGM Plus ™ 1.1
100 bp
200 bp
300 bp
400 bp
D3
vWA
D16
D2
A
D8
D21
D18
D19
Th01
FGA
GS500-internal lane standard
PowerPlex ™ 1.1
100 bp
200 bp
300 bp
D3
vWA
FGA
A
THC1
TPOX
CSF1PO
D5
D13
D7
GS500-internal standard
SGM Plus ™ 1.1
100 bp
200 bp
300 bp
400 bp
D3
vWA
FGA
A
D8
D21
D18
D5
D13
D7
GS500-internal lane standard

Fig. 12.7: Few commercial STR multiplex amplification systems for forensic analysis. Coloured boxes show the size range of the alleles of each locus in each multiplex kit. The red lines show the internal ladder

Box 12.2: Types of STRs

1. Simple consisting of 1 repeating sequence e.g. a STR locus has several alleles but each allele differs only in number of repeats but the sequence of the repeats is same.
2. Simple with non-consensus alleles e.g. consider two alleles of locus HUMTHO1
   Allele 3: [AATG] 3
   Allele 4: [AATG] 4
3. Compound with non-consensus alleles, e.g. consider two alleles of the locus vWA.
   Allele 16: TCTA [TCTG] 4[TCTA] 11TCCATCTA
   Allele 16: TCTA [TCTG] 3[TCTA] 12TCCATCTA
4. Complex repeats, e.g. consider two alleles of locus D21S11
   Allele 25: [TCTA] 4 [TCTG] 3 [TCTA] 3 TA [TCTA] 3 TCA [TCTA] 2 TCCA TA [TCTA] 10
   Allele 26: [TCTA] 4 [TCTG] 6 [TCTA] 3 TA [TCTA] 3 TCA [TCTA] 2 TCCA TA [TCTA] 8
5. Hypervariable repeats: SE33. There are few loci which have not only different repeat regions; they also have them arranged in many different ways. SE33 is an example.

During this process the gain or loss of a repeat region occurs depending on the looping out of the newly synthesised strand or the template strand. The theories of mutational mechanics are various and are out of the scope of this chapter. However it is important to understand that the actual mutation rates for the STRs are difficult to determine as the mutations are rare events and generally there is no consensus regarding them. However, pedigree studies have determined the mutational rates to be in the range of 1.2×10^{-4} to 1.5×10^{-2}. The mutation rates differ significantly between di, tri and tetra nucleotide STRs.

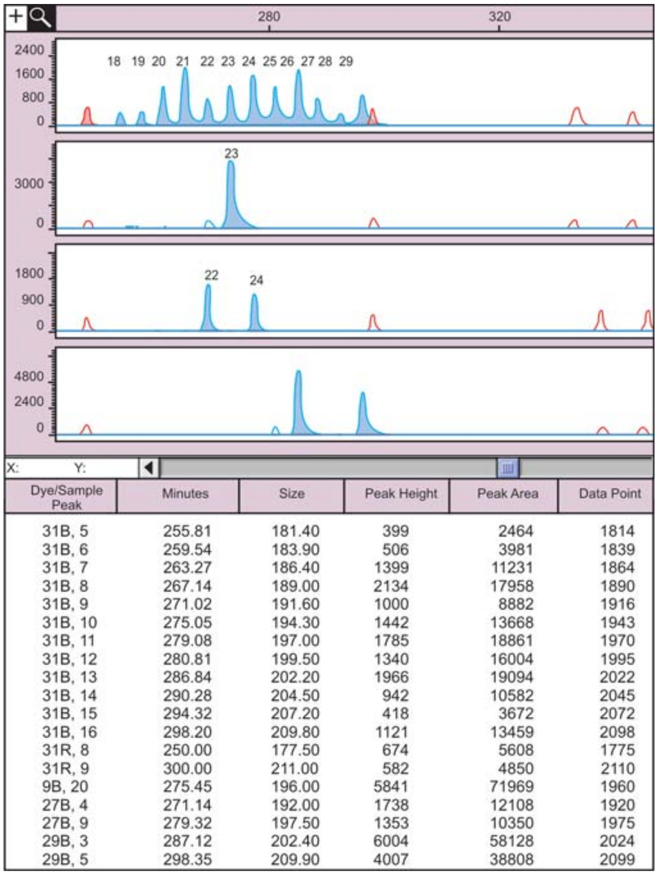
STR RESOLUTION TECHNIQUES

Electrophoresis has remained the cornerstone of STR allele detection, although the STR alleles can be separated by agarose gel electrophoresis with ethidium bromide staining, polyacrylamide gel electrophoresis (PAGE) gives better separation of the alleles.[33] Actually in smaller laboratories the STRs can be detected by silver staining method which allows unambiguous allele discrimination without the need of expensive automated DNA sequencers (Figs 12.8 and 12.9). However forensic laboratories always have a big work load and the samples have to be processed quickly in order to allow criminal or civil cases to proceed. Thus there was a need of automation and easy detection and designation of STR alleles. Figure 12.10 shows alleles of all loci in the SGM+™ kit, where Figure 12.11 shows profile of a person using powerplex™ kit.

STR Analysis and Automation

Instrumentation was developed for the automated analysis of DNA sequence using fluorescent dyes in 1986.[32] The automated systems such as ABI GeneScan® allowed electrophoretic information to be stored and tabulated as the alleles migrated through a gel matrix and pass a laser detection window. The availability of 4 different fluorescent dyes enabled the primers for different loci to be tagged with a distinct fluorescent dye that allowed multiplexing loci even if the size of the products overlapped and also enabled additional levels of controls, including internal size standards (Figs 12.12 and 12.13A to C).

Automatic allele sizing was thus made possible by running an internal size standard with each sample. Thus electrophoretic mobility variations (from lane to lane and gel to gel), that could lead to inconsistent allele sizing were automatically normalised (See Fig. 12.7). These initial studies confirmed the sensitivity of the automated analysis methods over the manual staining methods and also demonstrated that the automated analysis had the requisite, reproducibility, accuracy and precision for use in a forensic setting.



Dye/Sample Peak	Minutes	Size	Peak Height	Peak Area	Data Point
31B, 5	255.81	181.40	399	2464	1814
31B, 6	259.54	183.90	506	3981	1839
31B, 7	263.27	186.40	1399	11231	1864
31B, 8	267.14	189.00	2134	17958	1890
31B, 9	271.02	191.60	1000	8882	1916
31B, 10	275.05	194.30	1442	13668	1943
31B, 11	279.08	197.00	1785	18861	1970
31B, 12	280.81	199.50	1340	16004	1995
31B, 13	286.84	202.20	1966	19094	2022
31B, 14	290.28	204.50	942	10582	2045
31B, 15	294.32	207.20	418	3672	2072
31B, 16	298.20	209.80	1121	13459	2098
31R, 8	250.00	177.50	674	5608	1775
31R, 9	300.00	211.00	582	4850	2110
9B, 20	275.45	196.00	5841	71969	1960
27B, 4	271.14	192.00	1738	12108	1920
27B, 9	279.32	197.50	1353	10350	1975
29B, 3	287.12	202.40	6004	58128	2024
29B, 5	298.35	209.90	4007	38808	2099

Fig. 12.8: Electropherogram showing allelic peaks of different sample generated using an automated DNA sequencer

The casework validation studies showed that mixed samples could be detected and correctly interpreted, STR analysis yielded results where single locus probe analysis had failed. Also the discrimination power of a multiplex STR system was much greater than the systems based on HLA DQA1 and conventional blood grouping systems. Multiplexing is a technique where a number of STR loci are amplified in a single tube PCR. Now kits are available which can amplify 16 STR loci in a single tube, thus increasing throughput and the discrimination power STR analysis immensely.

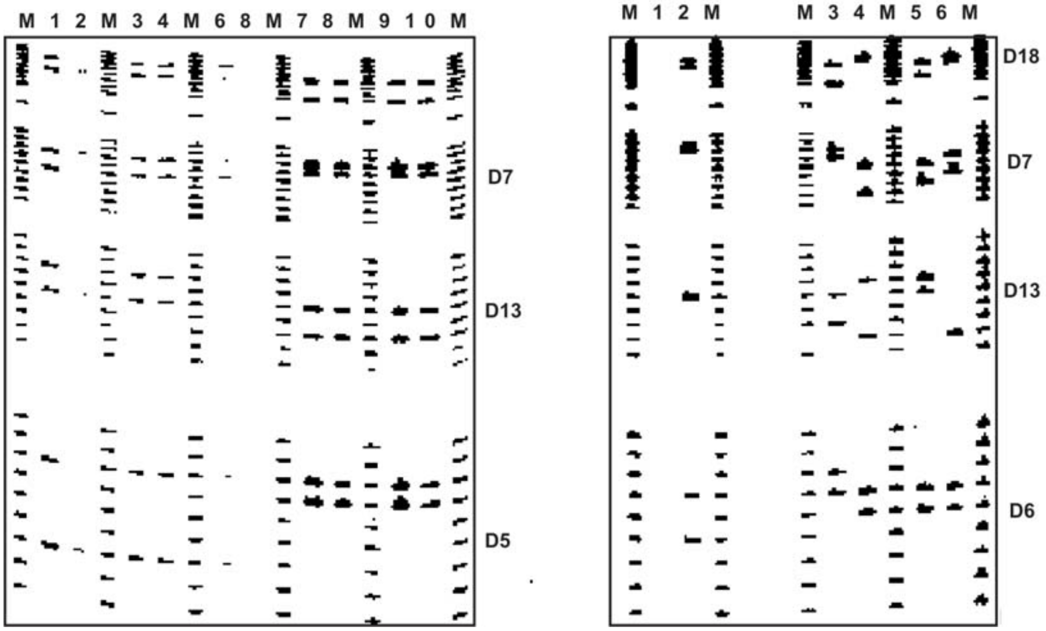


Fig. 12.9: Shows the polyacrylamide gel separation of STR loci and visualisation of bands using the silver staining. Silver staining is cheaper than fluorescent technology and is recommended for laboratories low on budget. The process is labour intensive and it takes longer time. Further it is not compatible with many laboratories which need automation

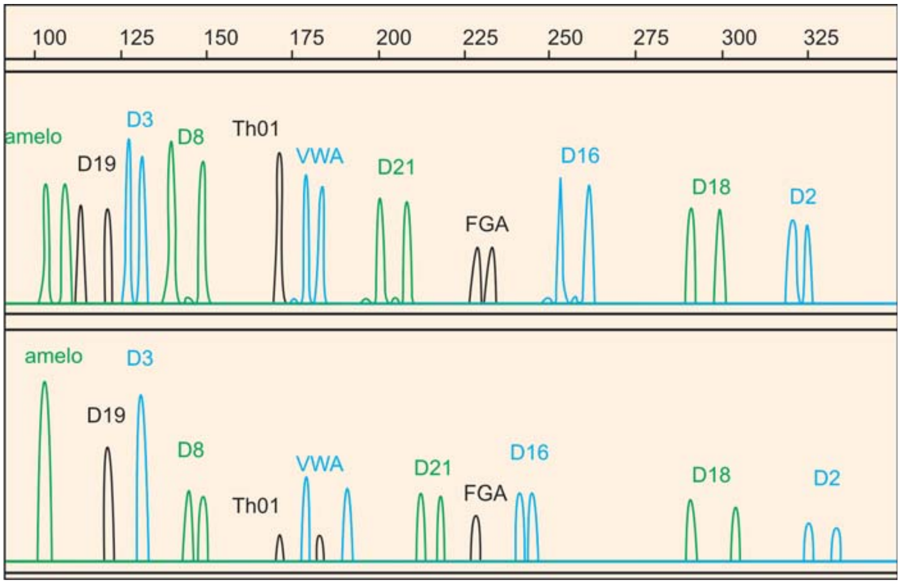


Fig. 12.10: The alleles of all the loci in the SGM+™ kit are illustrated showing the profile of two persons. Note how Loci D19 in the lower panel has only one allele demonstrating that the person is homozygote for this locus. Same is true for the locus THO1 in the upper panel. The locus amelogenin shows two alleles in the upper panel diagnosing the profile of a male and in the lower panel it has only one allele showing a female profile. Also note how the alleles of various loci differ in their spread and position

Use of Allelic Ladders

An allelic ladder consists of a number of alleles of a STR system and is used as a reference to designate the alleles. It can be thought of as a ruler which can measure a segment; allelic ladder measures the size of the allele reference to the allele present in the ladder. The ladder alleles have already been sequenced so their size is known for sure. The use of allelic ladders was shown to size the alleles accurately and it has been a consistent recommendation that sequenced allelic ladders be used in order to designate the alleles (Fig. 12.8). However, forensic standards are tougher than usual laboratories; so internal standards were developed which would be added to the sample before loading on the gel or capillary.

These commercial standards are area tagged with a red dye and contain fragments which are of regular size: the software attached to the sequencer sizes the alleles with reference to this internal ladder and the DNA analyst then compares the data with the allelic ladder which is run with each batch of samples. For this purpose experiments set the windows for the allele size and these windows can be fed in computer programmes like Genotyper™, the programme would call the allele on the basis of the established windows, which is called the '*absolute window*

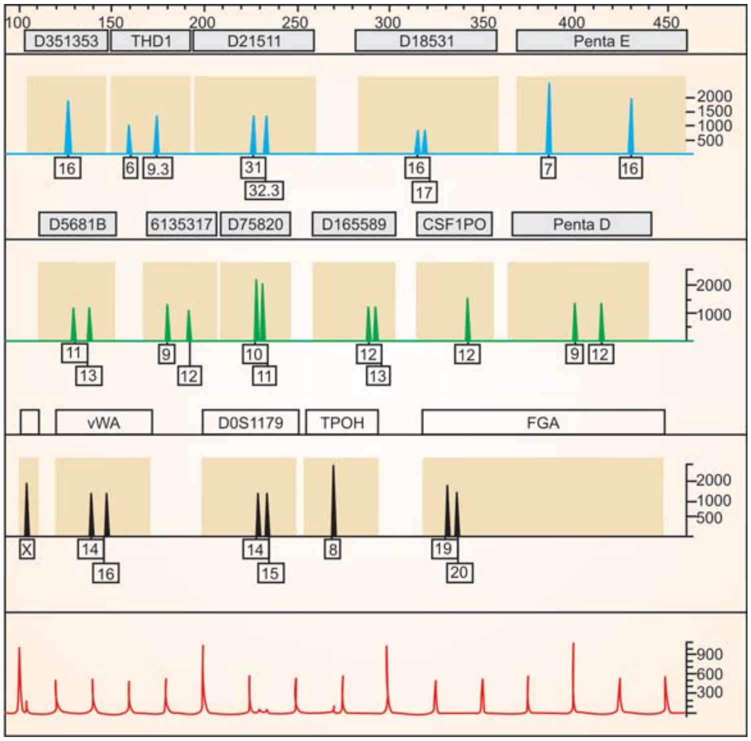


Fig. 12.11: Profile of a person using Powerplex ^{TM} kit. The lowest panel shows the internal ladder. Though the ladder is run mixed with the PCR product but the software allows it to be separated and presented as above for greater ease

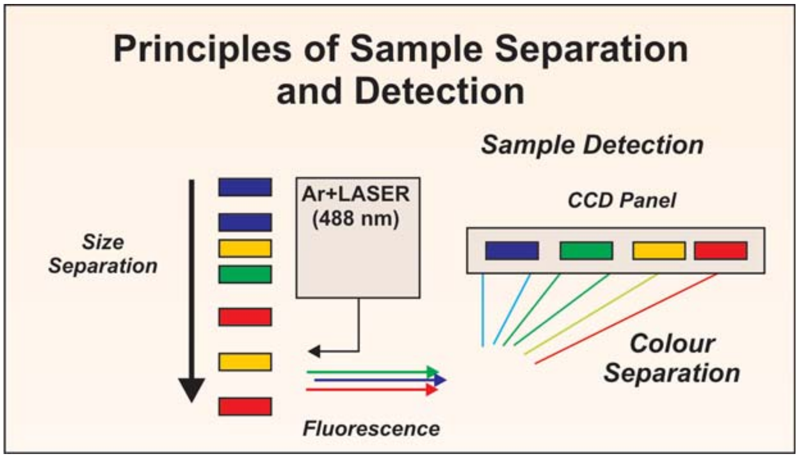


Fig. 12.12: A schematic diagram of the principle of detection of fluorescence by laser excitation, which is employed in all the modern DNA sequencers. Charge Coupled Device camera is an integral part of this device as is computer software, which can convert the fluorescence digitally into numbers

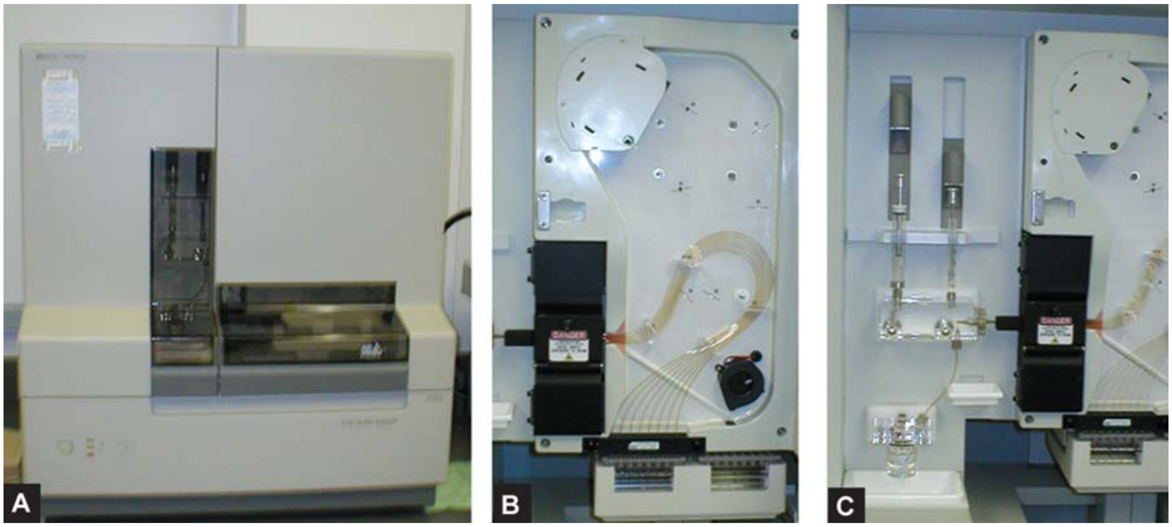
method'. The same method could be used to designate the alleles where the crime scene sample is to be compared to control samples.

Nonspecific Amplification Peaks

The sensitive automated DNA sequencer detects all products generated during a PCR assay and displays them as peaks on the electropherogram; these can include non specific peaks and stutter peaks besides the allelic peaks (Fig. 12.7). The non specific peaks are often less than 5 per cent in size of the allelic peak or have an atypical morphology which makes their recognition easy. The stutters however arise from enzyme slippage during the reaction; they cannot be eliminated completely from the reaction but can be recognised as a smaller (about 15%) peak than the actual allelic peak and are one repeat smaller than the actual peak. In a way these can be beneficial in the recognition of the actual peak. However, in di-nucleotide STR analysis, stutters pose difficulties in interpretation. Also in mixture analysis, stutters might enhance the peaks of the minor component thus causing problems in the identification of the minor peak.

ENVIRONMENTAL CONTAMINATION

The sensitivity PCR renders the microsatellite systems prone to contamination. Possible sources of contamination include, environment, contamination from a previous PCR and between samples during preparation. The latter two sources of contamination could be controlled by appropriate laboratory procedures and designated working areas. The environmental contamination has been shown to be limited.



Figs 12.13A to C: (A) Automated DNA sequencer Model ABI 3100; (B) Inner view of ABI 3100. This model has 16 capillaries. Each capillary can handle one sample and can process a large number of samples in a day; (C) Inner view of ABI 3100 uses capillary electrophoresis. The capillaries contain special polymer for this purpose

ACCEPTANCE OF DNA EVIDENCE AT THE LEGAL FORUM

The use of DNA analysis in forensic investigation offers the potential of identification or exclusion of a suspect with specificity, rarely possible in the past. The tool is especially useful in those difficult cases in which the only evidence recovered is of a biological nature, such as seminal fluid, or blood.

- Maternity identification—is necessary to identify the mother in cases of child abandonment or infanticide or swapping of neonates.
- Paternity identification—is important in cases of sexual assault in which a pregnancy occurs and pregnancy is either terminated or goes to full term.

In 1985 the forensic use of DNA began in the UK and it was applied to a civil immigration case and then to a criminal case which established the ground for the application of DNA analysis to forensic cases. The forensic applications of the technique were investigated and it was found that the technique was suitable for forensic purposes. By 1990 the systems for the DNA profiling were standardised and most laboratories on both the sides of the Atlantic were using them. The methodology was termed as sound in the first report of National Academy of Science's National Research Council (NRC Report I, 1992). UK pioneered the DNA technology, and acceptance of the technique by the legal forum was initially smooth. However, challenges relating to the statistical methods used occurred in UK in cases like R. *v* Andrew Deen (Times Law Report 1994), R. *v* Denis Adams (Cr. App. R. 1996) and R. *v* Alan James Doheny and Gary Adams (Cr. App. R. 1997). These and other cases set the guidelines for the presentation and acceptance of the DNA evidence by the courts in the UK.

As the DNA evidence became acceptable in the UK the FSS quickly moved to PCR based technology and developed a quadraplex system consisting of four STR loci (see Fig. 1.3). The Home Office, UK then commissioned the FSS for the development of a National DNA Database and using a more discriminating Second Generation Multiplex system, the database was started to be developed in 1995. Recently the FSS has started profiling criminal cases using the 11 locus AMPFLSTR® SGM Plus™ kit (Applied Biosystems CA, USA) (see Fig. 12.7).

The US Congress passed the DNA Identification Act of 1994, which helped in the acceptance of the DNA evidence. It also provided the necessary powers to the FBI Director in order to improve the standard of DNA analysis by ensuring quality assurance, appointment of a DNA advisory board and to construct a national DNA databank. Consequently the CODIS database for STRs has been established in the USA which comprises of 13 loci including the gender marker.

STANDARDISATION OF STR NOMECLATURE AND TECHNIQUE

As the use of STR grew for forensic purposes there was a need to standardize each aspect of this new technology so that the laboratories conforming to the standardized methods in different countries/regions could exchange and use the data for legal and population genetic purposes. Firstly there was a need for a common nomenclature for the widespread use of STR systems; guidelines were laid down by the International Society of Forensic Genetics (ISFG) and by one of its working groups, the European DNA Profiling Group (EDNAP). The basic recommendations of the International Society of Forensic Genetics are that the alleles would be designated according to the number of the repeats, and if any incomplete motifs or complex repeats occur they would be expressed by the number of complete repeats followed by a decimal point and then the number of bases of the incomplete motif. Various STR classes were also described such as simple, simple with non consensus alleles, compound, complex and complex hypervariable systems according to the arrangement of the repeat region in the alleles of a particular system as explained earlier. Apart from this, the resolution of STRs using the fluorescent technology, use of sequenced allelic ladders for the designation of alleles and use of standard multiplex sets of STRs set (1.3.4) all were a part of the efforts of the forensic community to standardize the STR technology. This has naturally lead to a boost in the confidence of the scientific and the legal communities in this technology. The International Society of Forensic Genetics as well as the European Network of Forensic Science Institutes has been performing a pivotal role in these standardisation efforts through regular inter- laboratory exercises and publication of its recommendations. Refer to Figure 12.7

presenting few commercial STR multiplex amplification systems for forensic analysis.

POPULATION GENETICS[41-49]

It is important to understand that forensic identification using DNA markers can only be done if a real estimate of the frequencies of those markers in the populations concerned is possible. Since real estimates are not possible due to large numbers a sample of population is examined and population frequencies of the markers estimated for application to that population. These frequencies are kept as a database and it has been recommended that at least 100 profiles should constitute such databases of all regional populations. *The frequencies of the markers are important as these are to be used in calculating the probability of discrimination and/or match by multiplying them together.* Thus population genetics is part and parcel of forensic genetics. In 1989 some leading scientists, who started a debate on the statistical basis of calculating the population frequencies, argued that the use of a general racial database is incorrect and databases from the relevant ethnic population should be used in all cases. It was also stated that the frequencies of the genotypes could be multiplied to generate a profile frequency if the loci were independent. As a result, challenges to the DNA evidence arose though DNA technology and its statistical basis stood a thorough scrutiny by the legal forum. The National Academy of Sciences, USA in its second report (NRC Report II, 1996a), further addressed the population genetic issues and recommended methods to calculate the population substructure and the likelihood of coincidental DNA match and laid down standards for the collection and preservation and presentation of DNA evidence.

Y-CHROMOSOME POLYMORPHISMS[34-40]

Most of the Y-chromosome does not undergo recombination during meiosis thus the paternally inherited chromosome bears the genetic prints from father to the son along the whole paternal lineage. In forensic practice, male specific Y-chromosome markers could be used as an adjunct to other markers on the autosomes and could prove to be a new and useful forensic tool on their own as well. Y-chromosome contains few polymorphic minisatellites like MSY1 and 2 as well. These markers are hypervariable but due to the simplicity of the use and availability of several Y-chromosome, STR systems have not gained widespread use for forensic purposes.

Development of Y-Chromosome Specific STR Systems

It was shown that 60 per cent of the Y-chromosome region on the long arm consists of interspersed tandem repeats. DYS19 was described as the first STR on the human Y-chromosome, which was found to be polymorphic and suitable for sex and paternity determination in deficiency cases. Various Y STRs were subsequently described. Most of these loci were found to be polymorphic and the haplotypes (*profile of an individual at two or more loci*) defined by using all of them could achieve very high levels of individualisation of males.

The terminology to be used for the Y STR haplotypes (a panel of STRs) is important to understand. The haplotypes were designated as Yh1 to Yh5 depending on the markers used to obtain the haplotype. They demonstrated that the power of discrimination of a haplotype consisting of a set of seven such STRs, DYS19, 389I, 389II, 390, 391, 392 and 393 (Yh1) was 74 to 90 per cent in European populations and a ten marker haplotype (Yh4) which includes Yh1 loci and DYS 385, YCAI and II; could be used for human identification as well. Most of these markers have been applied for building up population databases across the world in order to apply them to forensic casework. Various new Y STRs have been described during the last two years in two studies and validated for forensic use.

FORENSIC APPLICATIONS OF Y STRs[9-15, 34-40]

Y STR analysis can be useful for forensic identification in many situations where the autosomal STRs are of limited value. *They can be valuable in mixture interpretation in multiple rape cases or the detection of male specific profile in azoospermic / vasectomized male suspects when spermatozoa are not available.* The Y STRs could determine the male component in the male/ female *mixture specimens in cases where the specimen was small or the differential lysis failed or in other body fluid mixtures where the differential lysis could not be attempted.* The value of *Y-chromosome STR polymorphisms has been determined as excellent for paternity testing in deficiency cases.* The Y linked loci have been proved to be better exclusionary tools in paternity cases than the autosomal markers though their value in positive identification might not have an edge over the autosomal loci. In deficiency cases where the father was not available, other paternal relatives could be tested with Y STRs. Table 12.1 shows some of the Y-chromosome STR markers validated for forensic purposes.

Y STR MULTIPLEXING STRATEGIES[39-49]

Multiplex amplification of several loci allows simultaneous amplification of many STR systems which conserves the usually small samples submitted for forensic analyses. The multiplex systems described by Prinz, M. *et al*, and Kayser, M. *et al*, are mostly used as both are robust and reproducible and together, allow seven Y STRs to be analysed. These systems are therefore favoured at the moment. A new multiplex capable of amplifying seven Y STR loci, the 'Y-PLEX™ 6' has been recently developed by a commercial firm Reliagene, Technologies, Inc. USA.

PHYLOGENETIC VALUE OF Y-CHROMOSOME SPECIFIC STRs

Due to the mode of inheritance, Y-chromosome markers have been utilised as a tool for studying phylogenetic relationships and haplotypes based on Y markers have shown greater differentiation than autosomal or mtDNA markers.[50-52] Population specific Y haplotypes have been described based on such markers, which is of interest for phylogenetic as well as forensic purposes. A world wide sample was studied using biallelic markers along with a Y specific STR (DYS19), tracing early migrations not only from Africa but at least one from Asia as well. In an early study it was shown that with only five Y STRs; 15 distinct populations could be differentiated and phylogenetic trees and networks constructed in line with other data. This has been shown to be true in other populations, however since the mutation rates of Y STRs are high, they do not remain linear for long times therefore Y STRs could be used for population differentiation on a historical time scale rather than an evolutionary one.

Note**:** The calculation of statistical parameters is done using various computer softwares. *Appendix I* shows how after establishing a database of frequencies of particular loci in a population they are used to apply to forensic statistics.[53-54]

Table 12.1: Y-Chromosome STR markers validated for forensic purposes

Locus	*Repeat Motif*	*Allele Size Range*	*Alleles*	*Ref*
1.1.1.1.1.1 DYS19	$(TAGA)_n$	174-210	9	Kayser *et al.,* 1997
DYS389I	$(TCTG)_n$ $(TCTA)_n$	235-263	7	”
DYS389II	$(TCTG)_n$ $(TCTA)_n$	255-383	8	”
DYS390	$(TCTA)_n$ $(TCTG)_n$	191-227	9	”
DYS391	$(GATA)_n$	271-299	8	”
DYS392	$(TAT)_n$	233-263	11	”
DYS393	$(AGAT)_n$	108-136	8	”
DYS385	$(GAAA)_n$	360-414	15	”
DYS388	$(ATA)_n$	126-138	5	
DXYS156Y	$(TAAAA)_n$	160-170	3	
YCAII	$(CA)_n$	144-160	31	Mathias *et al.*, 1994
YCAIII	$(CA)_n$	192-204	7	”
A7 1	$(ATAG)_n$	161-181	6	White *et al.,* 1999
A7 2	$(TAGA)_n$	174-190	5	”

DETECTION OF DNA

Chemicals like the luminol and the UV light both can detect DNA as it fluoresces with both.[55]

Areas to Look for DNA on or off the Crime Scene

DNA can be found in pretty bizarre places and it is recommended that the following be collected from crime scene for detecting DNA:

- Finger nails or nails clippings.
- Tissues, paper towels, cotton swabs or ear swabs.
- Toothpicks, cigarette butts, straws and anything which might have come in contact with the mouth like cellular phones.
- Blankets, pillows, sheets, dirty linen, caps and head gear of any type.
- Eye glasses, contact lenses.
- Used stamps, envelops.
- Ligatures found on the body or the scene.
- Bullets that have passed through a body.

Collection, Storage and Transport of DNA Evidence

1. Wear clean latex gloves while collecting each item of evidence.
2. Each item of evidence must be packed in a separate container or envelope.
3. Blood, semen, saliva, urine and other stains must be air dried before package. Items of evidence having such stains might be dried using a hair dryer or a fan. After drying the samples should be packed in a paper bag or envelope.
4. In case of condoms these should be placed in a sterile tube. If a sterile tube is not available, the condom should be air dried and then packed in several layers of paper and then placed in a paper bag.
5. If stains are to be removed from an unmovable surface:
   a. Photograph surface with a ruler using black and white as well as colour film.
   b. Using a sterile moist swab, rub the swab on the stain till it is transferred to the swab; more than one swab might be required for this purpose. Two additional swabs should be collected from the area adjacent to the stain as substrate controls.

For proper chain of custody, each package should be marked for the case number, name of deceased (if required and known), date of collection, item contained, post-mortem number and any other identification as the case may require. The package should be sealed and the collector must sign across the seal.

APPENDIX I

This appendix shows how two important statistical notations are calculated using the mathematical formulae for interested readers.[53]

Match Probability

Definition: It is the probability that the two randomly selected individuals will have identical genotype.

Formula:

$$pM = \sum_{k=1}^{m} p_k^2$$

where, pM is the match probability, p_k represents the frequency of each distinct genotype, m is the number of the distinctive genotypes.

The combined probability of match over several loci is the product of the value for all the loci.

Example

The frequencies of blood systems, A_1, A_2, B, A_1B, A_2B and O were determined in the British people as 0.34, 0.08, 0.09, 0.024, 0.006 and 0.46 respectively.[53]

If the individual frequencies are squared and then added up.

Putting this value in the formula the probability of match is 0.34 or 34 per cent.

The probability of match of several independent systems is the product of the match probability of individual system.[53]

Power of Discrimination

Definition: It is the probability that two randomly selected individuals will have different genotypes. This is the reciprocal of the probability of match.

Formula:

$P_d = 1 - pM$

For several loci the formula is:

$$P_{docmb} = 1 - \prod_{i=1}^{n}(1 - P_{di})$$

Example

1. If P_M is 0.34, then the ability of the system to discriminate is 0.66.

   The Pd increases with the number of genotypes and is maximum if the genotypes occur at roughly similar frequencies.[53-54]
2. The P_d for loci D3, vWA and FGA for the Punjabi population are 0.913, 0.95 and 0.963. Subtracting each value from 1 the P_M for each locus is 0.087, 0.05 and 0.037.

   Multiplying these values together (0.00016), and then putting the value in the formula the P_{dcomb} is obtained, which is 0.9998.

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Chapter 13

Thanatology

INTRODUCTION

Thanatology is the academic, and often scientific, study of death among human beings.[1] It also describes bodily changes that accompany death and the after-death period. The word is derived from the Greek language. In Greek mythology, *Thanatos* (meaning: "death") is the personification of death. The English suffix — *ology* derived from the Greek suffix — *logia* (meaning: "speaking").[2-5] *Thus, thanatology is a subject that deals with scientific study of death, types of death, the various events, or changes that occur in the cadaver after death and their medicolegal significance.*

Since ancient times, man has pondered the mystery of his own death. It seemed that by knowing meaning of the death, he would be prepared to understand the reason of his life. Over the centuries people were deemed dead when they *stopped breathing* and when their *heart stopped beating*. But the technological advances provided artificial substitution of those functions that were considered responsible for setting the limit between life and death. Neurologists and neurophysiologists documented this at the end of the 1950s. Towards the end of 1960's the *Harvard Medical School* proposed for the first time, a new definition of death on neurological grounds.[6]

Harvard Ad hoc Committee on Brain Death In 1968, this committee of the *Harvard Medical School* published a report describing the following characteristics of a permanently nonfunctioning brain, a condition it referred to as "*irreversible coma*," now known as *brain death*:[7,8]

1. Unreceptively and unresponsitivity—patient shows total unawareness to external stimuli and unresponsiveness to painful stimuli.
2. No movements or breathing—all spontaneous muscular movement, spontaneous respiration are absent.
3. No reflexes—fixed, dilated pupils; lack of eye movement even when hit or turned, or ice water is placed in the ear; lack of response to noxious stimuli; unelicitable tendon reflexes.

In addition to these criteria, *a flat electroencephalogram* (EEG) was recommended. The committee also noted that *drug intoxication* and *hypothermia* which can both cause reversible loss of brain functions should be excluded as causes. The report was used in determining patient care issues and *organ transplants*. The condition of *irreversible coma*, i.e. brain death, needs to be distinguished from the *persistent vegetative state*, in which clinical presentations are similar but in which patients manifest cycles of *sleep* and *wakefulness*.[7,8] The Harvard report appeared few months after *Christian Bernard's* first transplantation of a human heart in December 1967. The year 1981 was highlighted by the report of the US President's Commission for the study of Ethical Problems in Medicine and Behavioural Research to define death.[8] The 1980's and early 1990's were characterised by multi-disciplinary debates. There were still worldwide controversies about a concept of human death on neurological grounds (whole brain, brainstem death and higher brain formulation of death). There was also disagreement on the diagnostic criteria of brain death, whether clinical alone, or clinical and ancillary tests.

The issues the Commission studied included: *definition of death, informed consent, genetic screening and counseling, differences in the availability of health care, life sustaining treatment, confidentiality and privacy, genetic engineering, compensation for injured subjects*, and *whistle-blowing in research*.

DEFINITION OF DEATH

In most of the countries of the world including India, there is no proper definition about death in law. However, *Black's Law Dictionary*[9] defines death as *'the cessation of life'* while the *Chamber's Twentieth Century Dictionary*,[10] defines death as *'the extinction of life'*. Clinically death is defined as the *irreversible cessation of life*. Thus, physician declares death with *cessation of all vital functions such as nervous, circulatory and respiratory systems*.[11-21]

Death is a process not an event and, while the cells of some tissues are still alive and even capable of movement (such as fibroblasts, leucocytes and muscle fibres, etc) others are dying or dead. Almost all doctors, whatever is their specialty, come across death at some time or the other in their profession. The legal and ethical aspects of this inevitable process form an important part of forensic medicine.

For understanding more about death and its mechanism, it is considered under two heads,[11-21] namely:

i. Somatic death
ii. Molecular death.

SOMATIC DEATH (SYNONYMS: SYSTEMIC DEATH, CLINICAL DEATH)

Definition

Somatic death is defined as the *irreversible cessation of functioning of the brain, heart and lungs resulting in complete loss of sensibility and ability to move the body*.[11-21] However, certain parts of the body such as the muscles can be still made to respond to an electrical, thermal or chemical stimulus, suggesting the reality that death has not resulted completely.

Explanation

In somatic death, though life has ceased in the body as a whole, it still persists in its component parts, namely the tissues and cells. Somatic death is diagnosed by establishing the following three facts:

- **Cessations of heart beating** – by careful auscultation of precordium for the heart sounds by a stethoscope continuously, for 5 minutes, and then repeating it thrice, at an interval of 5 minutes. A flat electrocardiogram (ECG) may also confirm it.
- **Cessation of breathing** – by thorough auscultation of the chest for the breath sounds using a stethoscope.
- **Cessation of brain activity** – by the dilated fixed pupil and absence of pupillary and corneal reflexes. A flat Electroencephalogram (EEG) would confirm it.

Thus, somatic death is declared clinically when the three vital organs, namely the heart, lungs and brain (*the tripod of life*)[12,19,21] fail to function and is confirmed by a flat ECG, absence of breath sounds and a flat EEG. Often these criteria are known as the *Harvard's criteria of assessing death*.[6-8] Somatic death shows resemblance to certain conditions such as *apparent death/suspended animation*, *coma* following excess dose of *sedatives* or *hypnotic* and *hypothermia* in old age.

MOLECULAR DEATH (SYNONYM: CELLULAR DEATH)

Definition

Molecular death is defined as ultimate *death of all cellular elements*.[11-13, 16-21]

Explanation

After somatic death, various tissues survive till the *oxygen supply* to them is *adequate*. When the oxygen reserve in the cells gets depleted, *cellular death* or *molecular death* sets in. Generally molecular death is not complete before 2 to 4 hours of somatic death.[16] Molecular death can be confirmed by *absence of any response to an electrical, thermal or chemical stimulus* in the tissues.

Thus, it is a fact that death is a process rather than an event and body dies by *bits and fragments*. It is reported that nervous tissue dies rapidly (i.e. the vital centres of brain die in about 5 minutes), while the muscle tissue lives up to 3 to 4 hours, after the cessation of the circulation.[15-17]

CONCEPT OF DEATH

Reviewing the historical aspects from both medical and legal angles, it is evident that the *concept/moment of death* merely comprises of *cardiac and respiration death*, i.e. cessation of spontaneous heart and lung functions.

Christan Bernard's first transplantation of a human heart was held in December 1967. This advent of human organ transplantation led to the necessity of scrutinising the phenomena of death. The heart transplantation studies also brought into critical focus the medicolegal and ethical questions, most specifically concerning when to remove the heart from the donor. The essential thing to be remembered here is that the needed donor heart has to be removed at the earliest after death is declared, which is difficult to decide, when life is supported by the artificial means of the modern medical technology.

In other words, biomedical innovations such as bypass machines, mechanical respirators, resuscitators, cardiac pacemakers and such other devices of recent invention have helped to maintain the circulation of oxygenated blood to brainstem, thereby prolonging life. Thus, life in such cases can only be *terminated* by withdrawal of these devices. The concept has also become important because the success of organ transplantation mainly depends on *rapidity* of its removal from the donor body after the stoppage of circulation. Organs like kidneys, lungs, liver, etc. need to be removed soon after cessation of circulation, as they deteriorate rapidly. This has established a set of *criteria* by which the moment of death is identified and led not only to the *'concept of death'* or *'moment of death'*,[11-13, 16-21] but also to its evaluation into 'brainstem death'.

BRAINSTEM DEATH AND ORGAN TRANSPLANTATION

For centuries, there has been a fear of being declared dead while still alive. However, only since 1959 there have been conceptual and practical problems with diagnosis of death following the description of *'brain death'*. The ability to *ventilate* 'brain dead' bodies in intensive therapy units preceded the developments of transplant surgery. The concept of brain death is not simply a convenience invented to satisfy the demands of transplant surgeons. According to this concept, depending on the structures involved, the brain death is of three types: *cerebral/cortical death, brain stem death* and *whole brain death*.

Cerebral/Cortical Death

In cortical or cerebral death, brainstem is intact, with continuous heart sounds and respiration, but total loss of sentient (sense of perceiving and feeling things) activity. Thus, severe brain damage, which does not involve the brainstem, may result in a *persistent vegetative state (PVS)*. These patients breathe spontaneously, open and close their eyes, swallow and make facial grimaces. It is in these cases that moral dilemma of allowing some one to die (*euthanasia*) arises.[18]

Causes

Cerebral hypoxia, toxic conditions, widespread brain injury, etc.

Brainstem Death

In brainstem death the cerebrum is intact, but cut off functionally. Brainstem comprises of small area of tissue in the floor of aqueduct between the third and fourth ventricles of brain, containing ascending reticular activating substance, which extends throughout the brainstem from spinal cord to sub thalamus and determines arousal. When this small area is dead, person becomes irreversibly *unconscious* and irreversibly *apnoeic* (i.e. incapable of breathing). The dilemma of euthanasia does not apply to those who are brainstem dead. These patients are dead irreversibly and unequivocally. Switching off the ventilator under such circumstances would not kill the patient, but would certainly discontinue *'ventilating a corpse'*.

Beating Heart Donor

After brainstem death is established, retention of the patient on ventilator, undoubtedly facilitates a fully oxygenated *'cadaver transplants'*, the so called *'beating-heart donor'*. Results of transplant have certainly improved with this. However, this has no legal sanction.[11-13,16-21]

Causes

Cerebral edema, increased intracranial pressure, etc.

Diagnosis of Brainstem Death

Brainstem death comprises of establishing following three things:

1. Patient is *deeply comatose* and *unable to breath spontaneously* and therefore *unconscious* and needs to be maintained on a ventilator.
2. Cause of coma must be known and rule out that it is not due to drugs, hypothermia or profound metabolic disturbance.
3. All the brainstem reflexes (Table 13.1) are absent and the *apnoeic test* is negative.

Brainstem Reflexes

Testing the function of cranial nerves, which pass through the brainstem, elicits the brain stem reflexes. If there is no response to these tests, the brainstem is considered to be irreversibly dead. Diagrammatic illustration of brainstem anatomy is shown in Figures 13.1A and B, and the brainstem reflexes with cranial nerves involved with the test procedure and the results are enumerated in Table 13.1.

Apnoeic test – No respiratory movement occurs when the patient is disconnected from the ventilator for long enough to ensure that the CO_2 concentration in the blood rises above the threshold for stimulating respiration, i.e. after giving the patient 100 per cent oxygen for 5 minutes the ventilator is disconnected for up to 10 minutes. If no spontaneous breathing of any sort occurs within those 10 minutes, the brainstem is incapable of reacting to the presence of the CO_2 and is thus confirmed to be dead.

Time of Death

Time of death is the time at which brainstem death is established. This does not coincide with time when ventilator is switched off; or heartbeat ceases. A physician should emphasize this fact to the relatives. He should clearly tell that ventilator is not withdrawn to let the patient die. Here the patient is already dead.

Whole Brain Death

Whole brain death comprises combination of both cortical and brain stem death. With this newer concept, clinical definition of death has been now modified as irreversible *state of coma* consisting of:

- Deep state of unconsciousness with no response to external stimuli/internal need.
- No movements, no spontaneous breathing.
- Cessation of spontaneous cardiac activity without assistance.
- No reflexes.
- Bilateral dilated fixed pupils.
- A flat isoelectric EEG.
- No profound abnormalities of serum electrolytes, acid-base balance or blood glucose.

This has allowed the feasibility of removal of vital organs such as the heart, kidney, liver, etc. from a donor body for the purpose of organ transplantation successfully without any ethical or legal complications.

THE MODE, MANNER, MECHANISM AND CAUSE OF DEATH

These terminologies are often confused in proper interpretation in practice. These are very essential as a forensic/ medicolegal expert has to pronounce 'cause of death' at the end of a 'medicolegal autopsy' and/or issue a death certificate.[17,18,20,21,24,25]

I. MODES OF DEATH

Depending on the system most obviously affected, irrespective of the remote causes of death, the *modes of death* are classified by *Bichat* into three types: namely *syncope, asphyxia* and *coma*.[15,24] Death could begin in one of the three systems. It may be also noted here that the terminologies *'syncope'* and *'coma'* are not used as cause of death.[17-21]

Syncope (Fainting)

Syncope is a result of sudden stoppage of functioning of the heart, which may prove fatal.[17,18,20,21]

Mechanism

Syncope or fainting is due to vasovagal attacks resulting from reflex parasympathetic stimulation, reflex bradycardia or asystole, or by reflex splanchnic vasodilatation. Due to acute reflex circulatory changes, blood pressure falls suddenly causing cerebral anemia and rapid unconsciousness. Recovery is usually common. However, sudden stoppage of functioning of the heart can also result in ischemia of vital centres of the brain and ultimately death.

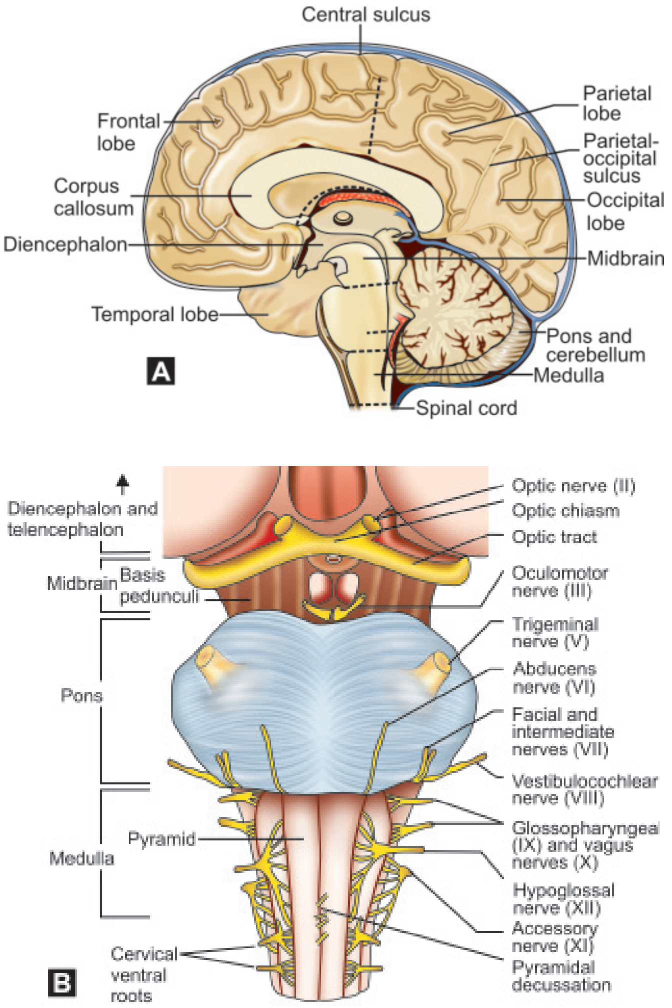
Causes

Anemia due to sudden and excessive hemorrhage, asthenia due to deficient power of heart muscle as in fatty degeneration of heart, myocardial infarction, exhausting diseases, poisoning with cardiac poisons, reflex cardiac inhibition (vagal inhibition of

Table 13.1: Brainstem reflexes and the cranial nerves involved[22,23] — the test procedures and the result

Brainstem reflexes	*Test procedure*	*Test result suggesting brainstem death*	*Cranial nerves*	
			Afferent	*Efferent*
Pupillary reflex* (Light reflex)	By means of a pen torch, instill a beam of light into the eye	Pupils *do not* constrict and remain fixedly dilated	2nd	3rd
Corneal reflex *	Touch the cornea with cotton fibre	Rapid closure of eye lids – *not observed*	5th (1st divsion)	Both 7th
Vestibulo-ocular reflex	Irrigation of ice cold water into external ears (tympanic membrane)	No eye movement (*Doll's eye movement*)	8th	3rd and 6th
Grimace reflex	Pinching over the face (triginimal territory)/to limbs	No grimactic changes	5th	7th
Gag/cough reflex	Tickle soft palate/uvula/throat	*No gag or cough*	9th	10th

* Hutchinson's Clinical Methods, 15th edn, Baillere Tindall and Cassell, London, 1973.


Central sulcus
Frontal lobe
Parietal lobe
Parietal-occipital sulcus
Corpus callosum
Occipital lobe
Diencephalon
Midbrain
Temporal lobe
Pons and cerebellum
Medulla
Spinal cord
A
Diencephalon and telencephalon
Optic nerve (II)
Optic chiasm
Optic tract
Midbrain
Basis pedunculi
Oculomotor nerve (III)
Trigeminal nerve (V)
Abducens nerve (VI)
Pons
Facial and intermediate nerves (VII)
Vestibulocochlear nerve (VIII)
Pyramid
Glossopharyngeal (IX) and vagus nerves (X)
Medulla
Hypoglossal nerve (XII)
Accessory nerve (XI)
Cervical ventral roots
Pyramidal decussation
B

Figs 13.1A and B: *Brainstem Anatomy:* (A) Midline view. The brainstem lies at the centre of the brain, connecting it to the spinal cord and is comprised of the medulla, pons, and midbrain. The cerebellum is attached to pons. The diencephalon and cerebrum are the higher levels above the midbrain. (B) Front view. Showing the three major distributions (medulla, pons, and midbrain) and cranial nerves carry information from the body to brainstem and from the brainstem to body

heart) due to sudden fright or emotion or trigger area injuries (e.g. blow on epigastrium), etc. Death resulting so, is called instantaneous physiologic death, vasovagal shock, vagal inhibition or neurogenic shock.

Clinical Features

Pallor of lips, face, dimness of vision, dilated pupils, skin-cold with perspiration, gasping respiration, nausea-vomiting, weak, slow pulse, fall of BP, delirium, convulsion and death.

Autopsy Findings

During autopsy the heart is contracted and the chambers are empty or show little blood, all the viscera (lungs, brain, abdominal organs) appear pale.

Asphyxia

Asphyxia is a condition resulting from interference with respiration or sudden stoppage of functioning of lung causing unconsciousness or death.[17,18,20,21] Various causes (etiology) and types of asphyxia are discussed in Chapter 15.

Mechanism

Sudden stoppage of or failure of functioning of the lungs can lead to impaired blood oxygenation leading to tissue anoxia. Nervous tissue is affected first by deficiency of oxygen, disturbing their functions. Subnormal blood supply to brain causes rapid unconsciousness. In all forms of asphyxia, the heart may continue to beat for several minutes after stoppage of respiration.

As per the 'rule *of thumb*', breathing stops within 20 seconds of cardiac arrest, and heart stops within 20 minutes of stoppage of breathing. The brain is highly sensitive to oxygen deprivation, which results in failure of vital centres and ultimately death.

Gordon's Hypothesis (1944)[21,24-26]

It emphasises the fact, that tissue anoxia, irrespective of its origin can invariably lead to cessation of vital functions, especially the circulatory failure resulting in death. According to this hypothesis, anoxia is classified into four types, namely *anoxic anoxia, anemic anoxia, histotoxic anoxia and stagnant anoxia.*

Anoxic anoxia: This is mainly due to mechanical asphyxia leading to defective oxygenation in lungs. Here the lungs are normal, blood and blood circulation is normal, but due to the mechanical asphyxia, respiration is difficult and entry of oxygen into the lung is impaired, resulting in defective oxygenation of the tissues. *Hanging, strangulation, suffocation, choking, drowning, high altitudes, etc.* constitute good examples for this type of anoxia.

Anemic anoxia: This is mainly due to the reduced oxygen carrying capacity of blood. Here though the lungs and blood circulation both are normal, the defects are in the circulating blood both in its quantity and in quality which results in defective oxygenation. *Exsanguinations, CO poisoning, etc*. constitute good example for this type of anoxia.

Histotoxic anoxia: This is mainly due to the depression of tissue oxidation. Here the lungs and blood circulation are normal, but due to impairment of cytochrome oxidase enzyme the tissues are unable to utilize oxygen. Cyanide poisoning is a suitable example of this type of anoxia.

Stagnant anoxia: This is mainly due to the inefficient circulation of blood. Here though the lungs are normal and blood in circulation is normal the circulation is inefficient; hence oxygenation is improperly taking place. Congestive cardiac failure, traumatic shock, heat stroke, etc. constitute good examples for this type of anoxia.

Clinical Features of Asphyxia

Clinical features of asphyxia can be described under three stages, which may last for 3 to 5 minutes and they are:[15-17, 19-21]

- Stage of dyspnoea
- Stage of convulsions
- Stage of exhaustion and respiratory failure.

Stage of dyspnoea: This is due to excess accumulation of carbon dioxide, which stimulates the respiratory centre resulting in increased rate and amplitude of respiratory movements.

Stage of convulsions: This is due to lack of oxygen and the victim will show labored respiration, clouding of consciousness, convulsions, sphincteric relaxation, etc. Face and hands are deeply congested and cyanosed. This stage may last for 1 to 2 minutes.

Stage of exhaustion and respiratory failure: It lasts for 2 to 3 minutes. Respiratory and other nervous centres are completely paralyzed. Muscles are flaccid, reflexes are lost, breathing is gasping with long intervals between gasps and then eventually stops, pulse is imperceptible, heart may continue to beat for few minutes and then stop, declaring death.

Postmortem Appearances of Asphyxia

Characteristic findings are cyanosis, deep postmortem hypostasis/lividity, and petechial hemorrhages (Tardieu's spots), visceral congestion and cardiac dilatation.[20-21] Specific findings depend on actual causes of asphyxia and they could include ligature mark in hanging, fine froth around nostrils and mouth in drowning, etc. Important external and internal autopsy findings are highlighted in Tables 13.2 and 13.3, respectively.

Following facts may also be remembered during autopsy of a case of asphyxial death:

- About 50 per cent of cases with constriction of the neck do not show signs of asphyxia and in these cases death occurs rapidly due to cardiac arrest.
- Asphyxial signs are found in several natural diseases also.
- One of the effects of asphyxia is that it causes vomiting due to medullary suboxia, which may therefore be aspirated in the air-passages prior to death. This is more often reported among the infants, which therefore should not be assumed to be the cause of asphyxia
- *Variations in intensity of asphyxial signs:* When the asphyxial process is slight and lasts for a prolonged period, congestion is less. However, when the asphyxial process is of intense nature and lasts for a short period, congestion and lividity is marked. If during asphyxia, heart failure occurs prior to respiratory failure, asphyxial signs will be less. Thus, at times the findings may not be sufficient to use an accurate term, and the cause of death may have to be given in a broad framework such as *'consistent with asphyxia'*.

Coma

Coma results from sudden stoppage of functioning of the brain. It is a state of unarousable unconsciousness determined by absence of any psychologically understandable response to external stimuli or inner need.[20,21,24,26,27]

Table 13.2: Important external autopsy findings of asphyxia

Sites	*Changes noticed*
Face	Pale (with slow asphyxia), or distorted, congested, often cyanosed and purple, at times swollen and edematous.
Ears	Bluish
Fingernails	Bluish
Tongue	Protruded and with frothy and bloody mucous evading from mouth and nostrils.
Eyes	Prominent, conjunctiva congested and pupils dilated.
Skin above the level of obstruction — in the scalp, face, eyebrows, eyelids, fore head, behind the ears, circum-oral region, conjunctivae and sclera.	Petechial hemorrhages (Tardieu's spot) - appear as rash like shower (in hanging and strangulation and traumatic asphyxia) due to acute increase in venous pressure, over distension and rupture of thin walled peripheral vessels and capillaries especially in their lax, unsupported tissues. Differential diagnosis of petechiae are: • Bacterial endocarditis • Meningococcal septicemia • Blood dyscrasias • Purpura/hemophilia • Coronary thrombosis, etc

Table 13.3: Important internal autopsy findings of asphyxia

Sites	*Changes noticed*
Scalp	Tardieu's spots seen in it's under surface.
Brain	Multiple Tardieu's spots, especially in the white matter
Heart	Tardieu's spots seen as discrete spots, reddish patches in the auriculo-ventricular junction and the back.
Large veins/vessels	Full of blood and may burst open, especially in the eardrum and nose resulting in ear- nose bleeding.
Blood	Fluid and dark due to increase in CO_2 and fibrinolysins.
Thymus	*Tardieu's spots* – Round and pin head to 2 mm in size.
Larynx and trachea	Congested unusually with frothy (slightly) oozing.
Lungs	• Colour – Dark purple • Cut section – congested • Lower lobes and inter-lobar fissure Tardieu's spots • Marginal portion – Emphysematous • Posterior and dependent parts – Fluid accumulation (not edema) • Alveoli - edematous with serous/sero – sanguinous fluid exudates.
Abdominal viscera	Marked venous congestion.
Pleura and Peritoneum	Tardieu's spots – rarely seen in the parietal layers

Mechanism

In coma there is a combination of both syncope and asphyxia leading to death. It is due to paralysis or insensibility of central portion or vital centres of the brainstem.

Causes

Various probable causes of coma are:

- Compression of brain due to diseases and injuries of brain or its membranes, e.g. intracranial hemorrhage, inflammation, abscess, or neoplasm of the brain.
- Acute poisonings with opium, cocaine, chloral hydrate, anesthetics, barbiturate, alcohol, etc. *having specific depressor action on brain and nervous system*.
- Metabolic disorders and infections—uremia, eclampsia, diabetes, cholaemia, acetonaemia, pneumonia, infectious fevers, heat stroke, etc.
- Other causes – embolism and thrombosis in cerebral vessels, epilepsy, hysteria, etc.

Autopsy Findings

Postmortem findings could reveal all specific pathological findings depending on the actual cause. The brain and meninges are congested. The right side of the heart is usually full, while the left empty. Lungs are congested.

II. MANNER OF DEATH

It explains how the cause of death came about. Manner of death is generally considered to be natural, homicide, suicide, accident and undetermined. Some people also use the category unclassified. Just as a mechanism of death can have many causes and mechanisms, a cause of death can occur through multiple manners.

Thus manner of death, in India, is determined by the court after examining all facts about the case including the evidence and interpretation by the doctor.[11-13,15-17] However, in some of the other countries, especially in the United States of America where medical examiners system prevails, after visiting and evaluating the scene of crime, incidence, and the victim, doctor has to opine manner of death.

An individual can die of massive hemorrhage (the mechanism of death) due to stab injury of the heart (the cause of death) with manner of death being homicide (when somebody stabbed the individual) or suicide (when he stabbed himself) or accident (when the weapon was upright and he fell on it) or undetermined (when one is not sure which of the above has occurred).

The manner of death, when determined by a forensic pathologist is just an opinion and is based on facts which are known concerning *circumstances leading to death,* in conjunction with autopsy findings and the laboratory tests. The autopsy findings may contradict or agree with the facts provided on how death occurred. Thus, if one has a history (facts) that an individual stabbed himself and the autopsy reveals stab wound on the back, the history specified is obviously incorrect, as the site of stabbing is inaccessible for oneself. However, if it is found on the front of the chest on left side over the precordium, easily accessible for oneself who is a right-hander, then the autopsy findings are *consistent with the history*.

It may also be remembered here that just because the forensic pathologist makes an opinion as to manner of death, does not mean that the family of the deceased or other agencies may accept it. There are several cases where forensic pathologist opined homicide as against police agencies who have written an accident. At times families challenge opinion of forensic pathologist and go to court to have the manner of death overturned. In most instances the court will support forensic pathologists; however he should not get upset if court does not. Manner of death is '*undetermined*', when the facts are insufficient about circumstances surrounding the death, or when the cause of death is *unknown*. Thus, if one finds skeletonised remains of a young adult male without any evidence of trauma, one cannot opine that the manner is – *suicide* / *homicide* / *accident* / *natural* and this has to be opined as '*undetermined*'.

In addition to usual classification of manner of death – suicide, accident, homicide, natural and undetermined, some people use the term '*unclassified*'. This also refers to death in which cause and circumstances are not known. For example, a woman who came to hospital for medical termination of pregnancy (MTP), hypertonic saline solution was injected, and the woman went in to labour, delivered a live 350 gm infant, who died after 90 minutes, without any mechanical influence. Death obviously is not a suicide/ homicide/ accident/ natural, but is simply opined as *undetermined*.

Agonal period: It is the time between a lethal occurrence and death.

III. MECHANISM OF DEATH

It is the physiological or biochemical derangement produced by the cause of death, which is incompatible with life and results in death. For example, mechanism of death could be, haemorrhage, septicemia, cardiac arrhythmias, etc. One has to realize here that a particular mechanism of death can be produced by multiple causes of death and vise-versa. Thus, if an individual dies of massive hemorrhage, it can be produced by a gunshot wound, a stab wound, a malignant tumor of lung eroding into a blood vessel, and so forth. Vice-versa of this is that a cause of death, for example, a gunshot wound of the abdomen, can result in many possible mechanism of deaths, such as hemorrhage and peritonitis.

IV. CAUSE OF DEATH

Cause of death is defined as the disease or injury that produces a physiological derangement in the body that results in death of the individual. Thus, the following are the examples for cause of death: a gunshot wound of head, stab wound of the chest, adenocarcinoma of lung, coronary atherosclerosis, etc. Cause of death could be any one of three types (i) immediate cause, (ii) antecedent cause and (iii) contributory cause.

Immediate Cause

Immediate cause is the actual cause at the time of terminal event, e.g. bronchopneumonia, peritonitis, trauma, etc.

Antecedent Cause

Antecedent cause is the actual pathological process responsible for the death at the time of the terminal event or prior to or leading to the event, e.g. gunshot wound of abdomen complicated by generalized peritonitis.

Contributory Cause

Contributory cause is the pathological process involved in or complications, but not causing the terminal event, e.g. Carcinoma stomach.

Not infrequently, the cause of death is in appropriately listed as '*cardiac arrest*' or '*cardiopulmonary arrest*'. This simply means that '*heart stopped*' or the '*heart and lungs stopped*'. It is a fact that in all individuals who die, heart and lungs stop functioning. Hence these terminologies cannot be considered as cause of death and to a degree are not even mechanisms of death. Yet clinicians wrongly continue to mention so in the death certificate issued and government agencies accept the same.

MEDICOLEGAL IMPORTANCE OF DEATH

Medicolegally, death is an important entity in its own way,[15-17, 19-21, 51] and is enumerated below:

Death and the Indian Penal Code (IPC)

Section 46, IPC, states the fact that the word '*death*' denotes death of a human being unless the contrary appears from this context.

Disposal of the Body

Rarely, during cremation of a dead body immediately after somatic death, spontaneous movements of hand or feet may be observed in the cadaver on the funeral pyre, creating an impression that the person is not actually dead and the disposal is premature.

Tissue and Organ Transplantation

Viability of transplantable tissues and organs falls sharply after somatic death; a liver must be removed within 15 minutes, kidney within 45 minutes and heart within an hour.

Legal Presumption of Death

- In India the Law is:
  a. that if a person is proved to have been alive within 30 years the legal presumption is that he is still alive, unless
  b. it is proved that the person has not been heard of for 7 years by those who would naturally have heard of him if he had been alive, in which case the law presumes that he is dead.

  The law, however, presumes nothing as to the time of death, the period of which, if material (as it often must be in cases of succession and inheritance), must be proved by evidence. In either case the presumption arising may be rebutted by proof (a) of the person's death and (b) of his being still alive.
- If a person is unheard of for seven years, the court may on application by the nearest relatives, presume death to have taken place. *Section 108*, Indian *Evidence Act (IEA) 1872*, deals with burden of proving that the person is alive who has not been heard of for seven years. Accordingly when the question is whether a person is alive or dead and it is proved that those who would naturally have heard of him if he had been alive have not heard him of for 7 years, the burden of proving that he is alive is shifted to the person who affirms it. *Section 107*, *Indian Evidence Act (IEA) 1872*, deals with burden of proving death of a person known to have been alive within thirty years. Accordingly, when the question is whether a man is alive or dead, and it is shown that he was alive within thirty years, the burden of proving that he is dead is on the person who affirms it.

Question of Presumption of Survivorship

When two or more persons die at almost the same time, or by a common accident, the question may arise who survived the longest; and if no direct evidence on this point is available the question becomes one of presumption of survivorship. Study the case example below and see how the question arises.

Suppose a rich father A has left property by executing a valid will to his son B, and that A and B die by a common accident, no direct evidence being available as to whether A or B died first. Here the question of presumption of survivorship may arise, because if A died before B, B may be considered to have succeeded to the property left to him by A. It is generally accepted that normally a younger, stronger and more vigorous will survive longest.[12,20-21]

Issuing of Death Certificate

Before issuing this, doctor must confirm that the person is dead. *Death certificate* or *Certificate of cause of death* has a standard format. The *International Form of Medical Certificate of cause of Death* prescribed by the *World Health Organisation (WHO)* calls for statements on the morbid condition directly leading to death, the conditions antecedent to it, and contributory conditions not related to the direct or the antecedent causes. It consists of *two parts*, designated as I and II. Part I is subdivided into *(a), (b)* and *(c),* and further providing space for entries on the *direct cause of death (a)* and the *antecedent causes (b)* and other contributary conditions *(c)*, and their approximate interval between onset and death. Part II takes care of the significant *contributory conditions*. This WHO format (proforma) is appropriately presented in Figure 13.2.

SUDDEN DEATH (RAPID DEATH, SUDDEN NATURAL DEATH)

The reviews of the extensive literature on sudden death agree unanimously that there is a great inconsistency in definitions of sudden death. The question is "*how sudden is sudden*?" The deaths, which are not preceded or only preceded for a short time by morbid symptoms, are called sudden death. The WHO definition of a sudden death is that it is *someone who dies within 24 hours of appearance of symptoms*.[31] However, in forensic sense, most of deaths are in minutes or even in seconds of appearance of symptoms.[15-17, 19-21] A sudden death is not necessarily unexpected death and an unexpected death is not necessarily sudden, but very often the two are in combination.[19-21,31]

To have a systemic view of differential diagnosis of the cause of death and to make a logical choice of most likely cause will help to improve the state of mortality statistics, assist the legal authorities and satisfy the bereaved relatives, perhaps by helping them to obtain insurance and compensation benefits.

When sudden and unexpected deaths are considered, an added dimension appears, as these deaths are usually reportable to the authorities for medicolegal investigation. The vast majority are due to natural causes, but often deceased either has not seen a doctor recently or the unexpectedness of their death does not allow their medical practitioner to have any idea of the reason why they suddenly died. Figure 13.3, below Illustrates common causes of sudden or rapid death. Medicolegal importance of this type of sudden unexpected natural death is that it usually raises a suspicion of foul play and death certificate must not be issued in such cases till an autopsy is conducted and cause of death is confirmed.

Cause

Where a natural death is very rapid, perhaps virtually instantaneous, the cause is invariably cardiovascular. Indeed, if a person collapses and is clinically dead when someone nearby runs to assist him, this can only be a cardiac arrest, as virtually

DEATH CERTIFICATE

Name of the Deceased:

Sex:

Age:

Religion:

Identification Marks: **1.**
2.

Date and time of death:

CAUSE OF DEATH		Approximate interval between onset and death
Part-I Disease or condition directly leading to death:	a.	
	b.	
	c.	
Antecedent causes:		
Part-II Other significant contributory conditions:		

Place :

Date :

Signature of MO:

Name of the MO:

Reg.No:

Designation:

Official Seal:

Fig. 13.2: Format (Proforma) of a certificate of cause of death as per the WHO regulation

no other mode of death operates so quickly. This type of collapse is the one, which may respond best to cardiopulmonary resuscitation. Extracardiac causes, even still in the cardiovascular system, are rarely so rapidly fatal, though death in minutes is common. Of course, in all such discussion, what is meant by death must be defined, but for our purposes here, irreversible cardiac arrest is taken as the criterion of death. Sudden death may occur from both natural and unnatural causes (such as violence or poisoning) or from a combination of both. However cardiovascular system accounts for the vast majority of sudden deaths. Common causes of sudden deaths are presented diagrammatically in Figure 13.3 and discussed below individually.

Cardiovascular System

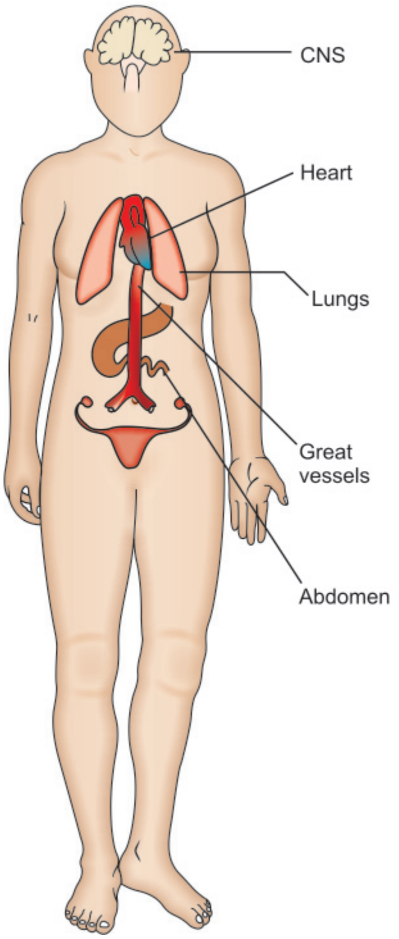
A lesion that causes most of the sudden unexpected deaths (SUD) is usually in the cardiovascular system, even if the vessel concerned lies anatomically in the brain or abdomen.[20-21] Although there is huge geographical variation due to the remarkable variation in the atherosclerosis out of all the prime cause of SUD lies in the heart itself. The following lesions are most obvious.

- Coronary artery diseases (atherosclerosis, thrombosis, syphilis, etc.)
- Congenital heart diseases
- Valvular heart diseases (rheumatic, syphilitic, etc.)
- Hypertensive diseases
- Infections (myocarditis, postinfectious myocardial degeneration)
- Cardiac tamponade — constitutes a lesion wherein ruptured myocardial infarct, trauma, etc. results in collection of blood in pericardial sac. About 250 to 300 ml blood may act fatal, making heart unable to function normally, resulting in cardiac standstill leading to death.
- Obscure conditions such as cardiomyopathies, Fiedler's myocarditis, etc.
- Aortic aneurysms of atherosclerotic or dissecting type.

Respiratory System

Most of the causes of sudden death within the respiratory organs are again vascular.[20-21] Pulmonary embolism is a common and in fact is the most under-diagnosed cause of death. In almost every case, the source of emboli is in the leg veins, as pelvic thrombosis is very rare.

After any tissue trauma, especially where immobility or bed rest occurs, deep vein thrombosis develops. Most remains silent and cause no problems, but a proportion embolises and blocks pulmonary veins of varying sizes. Some produce no lung lesions at all, others produce infarcts, which may or may not lead to clinical signs, and a minority (though an appreciable number), block major vessel and cause death.



		Relative incidence
CNS	Sub-arachnoid haemorrhage	++
	Cerebral haemorrhage	+
	Epilepsy	
Heart	Coronary artery disease	+++++
	Hypertensive heart disease	+++
	Aortic stenosis	++
	Myocardiopathies	
Lungs	Pulmonary embolism	+++
	Haemoptysis (T.B. or tumour)	
	Asthma	
	Viral pnemonia	
Great vessels	Atheromatous aneurysm	+++
	Dissecting aneurysm	++
	Syphilitic aneurysm	
Abdomen	Perforated viscus	+
	Mesenteric embolism	+
	Gastrointestinal haemorrhage	
	Abortions	
	Rupured ectopic pregnancy	

Fig. 13.3: Common causes of sudden death

Majority of cases of pulmonary embolism deaths have predisposing cause such as fractures, tissue trauma, surgical operation, burns trauma, bed rest, forced immobility, etc. but others occur unexpectedly in normal, ambulant people. This makes relationship of death to an injurious event difficult.

Various causes of sudden natural death of respiratory system origin are:[11-13, 15-17]

- Pulmonary embolisms
- Massive hemoptysis (from pulmonary tuberculosis)
- Severe infections such as fulminating viral pneumonia (usually influenza)
- Chronic asthma/status asthmatics
- Anaphylaxis
- Obstruction of respiratory tract.

Intracranial Vascular Lesions

This has been dealt with in details in chapter on Trauma—Regional Injuries, but several types of intracranial vascular lesions are important in sudden or unexpected death. A matter of nomenclature should first be mentioned, as the term '*cerbrovascular accident*' is in common usage, both as a clinical diagnosis and as a form of death certification. Unfortunately, it is sometimes misinterpreted as an unnatural cause, because of the use of the word accident. It is much more satisfactory to avoid the term and either be more specific about the lesion if possible, such as '*cerebral hemorrhage*' or '*cerebral infarction*', or use a less ambiguous description, such as *cerbrovascular lesion*. Commonly encountered intravascular lesions resulting in sudden natural death are:[11-13, 15-17, 20, 21, 26-28]

- Intracranial bleeding due to *cerebral atheroma* and *stroke* or *hypertension*
- *Subarachnoid hemorrhage* from ruptured aneurysm
- Cerebral thrombosis
- Embolisms
- Infections of meninges *(meningitis)*
- Brain tumors, which can result in death due to increased intracranial pressure, sudden hemorrhage from tumor mass, etc.
- Idiopathic epilepsy
- Functional inhibition of the vagus nerve.

Psychiatric Patients

Sudden, unexpected death may also occur in psychiatric patients,[31] has raised the concern that the use of psychotropics, especially antipsychotics, may be associated with an increased risk for sudden death. This concern is maintained even though not all psychiatric patients who have succumbed to sudden death have been on psychotropics.

Gastrointestinal System

The major cause of sudden death within the gastrointestinal system may once again be mentioned to be mostly of vascular origin and they are:[11,13,16,17,20]

- Severe gastrointestinal bleeding due to gastric or duodenal ulcers

- Ulcerative colitis, malignancies, etc. can be fatal in a short-time, even though most are moderate enough to allow medical or surgical treatment
- Mesenteric thrombosis and embolism leading to infarction of the gut are not sudden, but may be rapid and remain undiagnosed
- Perforation of peptic ulcer can be fatal in hours if not treated
- Intestinal gangrene due to strangulated hernias and torsion due to peritoneal adhesions can be a fulminant and fatal condition
- Aortic aneurysmal rupture
- Diseased viscera undergoing rupture
- Fulminant hepatic failure
- Acute haemorrhagic pancreatitis.

Gynaecological Conditions

If a women in her child bearing age is found to be dead unexpectedly and suddenly, following may be considered as cause of death:[11,13,16,17,20]

- Complication of pregnancy must be first thought of, just to make a primary exclusion.
- Haemorrhage in female genital organs could be due to abortion or ruptured ectopic pregnancy, etc. and is a grave emergency that can result in death from intraperitonial bleeding, unless rapidly treated by surgical intervention.

Endocrinal Causes

Sudden natural deaths occur due to any one of the following:[13,16,17,21,27]

- Adrenal insufficiency
- Diabetic coma
- Myxoedemic crisis
- Parathyroid crisis

Iatrogenic Causes[17,21,27]

Following may be suspected and may have to be ruled out:

- Abuse of drugs
- Sudden withdrawal of steroids
- Anesthesia
- Mismatched blood transfusion.

Miscellaneous Causes[17,21,27]

There is a vast list and comprise of cases of anaphylaxis, bacteremic shock, shock from fright or emotions, malaria, sickle cell crisis, alcoholism, etc.

Special Causes in Children[17,21,27,28]

If the victim happens to be a child following may have to be thought and ruled out, namely *SIDS (sudden infant death syndrome)*, or *cot deaths*, Mongol's and other types of congenital or mental disorders, concealed puncture wounds, indeterminate – very rarely. Recently vaccinations have been proved (DTP vaccine especially) to be the single most prevalent but most preventable cause of sudden infant deaths.[32-36]

CHANGES AFTER DEATH (SYNONYM: POSTMORTEM CHANGES, SIGNS OF DEATH)[11-13,16,17,20,21,27]

Changes after death vary with time since death and therefore it is better discussed time orientedly under three heads:

1. Immediate Changes

These are changes seen in the dead body at time of somatic or clinical death and are as follows:

- Insensibility
- Respiratory arrest
- Circulatory arrest.

2. Early Changes

These are changes seen in the dead body in the first 12 to 24 hours of death and include:

- Postmortem cooling
- Eye changes
- Skin changes
- Postmortem lividity
- Muscle changes.

3. Late Changes

These are changes seen in the dead body after 24 hours of death and include a chain of events known as postmortem *decomposition (putrefaction)*. Eventually the body will be reduced to skeleton and the change constitutes '*skeletonisation*'. At times putrefaction may be replaced by two alternative changes, i.e. either *adipocere formation* or *mummification*. Thus, late changes are enumerated as:

- Putrefaction
- Adipocere formation
- Mummification
- Skeletonisation

Medicolegal Importance of Postmortem Changes

They can help in assessing**:**

- Time since death (Postmortem Interval/PMI)
- Probable position of the deceased at the time of death.
- Cause of death
- Manner of death: *suicide, homicide,* or *accident*.

Each of the changes mentioned above are enumerated in Table 13.4, and discussed at this stage briefly.

INSENSIBILITY

Insensibility comprise of complete loss of response to sensation, viz touch, pain, temperature, etc. and loss of voluntary power to move. Though these are considered as the earliest signs of death, they can also lead to error, as they are found in other conditions such as: prolonged fainting attacks, epilepsy, vagal inhibitory phenomena, catalepsy, trance, narcosis, electrocution, etc.

Test for confirming insensibility—A flat EEG or loss of EEG rhythm.

Respiratory Arrest

Complete stoppage of respiration for more than 3 minutes. However, it may also be observed in conditions such as voluntary acts of breath holding, Cheyne-Stokes, breathing pattern breathing in apparently drowned, newborn infants, etc.

Table 13.4: Changes after death

Immediate changes	*Early changes*	*Late changes*
Insensibility	Postmortem cooling	Putrefaction
Respiratory arrest	Eye changes	Adipocere formation
Circulatory arrest	Skin changes	Mummification
	Postmortem Lividity	Skeletonisation
	Muscle changes	

Box 13.1: Historical aspect on death and fear of premature burial when alive

The macabre tales of the eighteenth century, on "corpses" reviving during funerals and exhumed skeletons found to have clawed at coffin lids led to widespread fear of premature burial, Coffins were developed with elaborate escape mechanisms and speaking tubes to the world above (Fig. 13.4), mortuaries employed guards to monitor the newly dead for signs of life, and legislatures passed laws requiring a delay before burial [37]

The medical press also paid a great deal of attention to the matter. In The Uncertainty of the Signs of Death and the Danger of Precipitate Interments in 1740, Jean-Jacques Winslow advanced the thesis that putrefaction was the only sure sign of death. In the years following, many physicians published articles agreeing with him. This position had, however, notable logistic and public health disadvantages. It also disparaged, sometimes with unfair vigor, the skills of physicians as diagnosticians of death. In reply, the French surgeon Louis published in 1752 his influential Letters on the Certainty of the Signs of Death. The debate dissipated in the nineteenth century because of the gradual improvement in the competence of physicians, invention of diagnostic clinical equipments such as stethoscope, etc which lead to the concomitant increase in the public's confidence in them.

Tests for confirming cessation of breathing—The recommended method is thorough auscultation for breath sounds by a stethoscope. No audible *breath sounds* on continuous *auscultation* of upper part of chest and in *front* of or on the *larynx* for minimum of 5 minutes, confirms cessation of breathing.

Other tests of historical interest which are never practiced now are:

- **Feather test** – no movement of a feather held in front of the nose.
- **Mirror test** – mirror held in front of the nose does not turn dim due to any moisture of breath.
- **Winslow's test** – no movement of surface of water in bowl kept on the chest.

Circulatory Arrest

Complete absence of heart sounds for more than 3 to 5 minutes. **Tests for confirming cessation of circulation**—The recommended method is again thorough auscultation for heart sounds by a stethoscope. No audible heart sounds on continuous auscultation on precordial area of chest for a minimum of 5 minutes, is suggestive of cessation of circulation and a flat ECG recording for 5 minutes confirms this.

Other tests of historical interest which are never practiced now are:

- **Magnus's test (Ligature test)**—fingers fail to show congestion distal to a ligature applied at their base.
- **Diaphanous test (Transillumination test)**—Failure to show redness in the web space between the fingers on transillumination from behind.
- **Icard's test**—fluorescent dye on being injected at a given site in a dead body fails to produces any discolouration.
- **Fingernail test**—no blanching and filling of blood in the fingernail on application of pressure and release.

SUSPENDED ANIMATION (SYNONYM: APPARENT DEATH)

Suspended animation is a condition, wherein the vital functions of body (heartbeat and respiration) are maintained at a low pitch reduced to a minimum for sometime, that they cannot be detected by routine methods of clinical examination.

Classification

Suspended animation is of two types, voluntary and involuntary.

Causes

- Voluntary—yoga practicing
- Involuntary—in newborn infants, insanity, drowning, electrocution, cholera, frozen coma, after anaesthesia, typhoid state, shock, sun-stroke, etc.

Medicolegal Importance

Prior to certifying death it is better to rule out that the patient is not in suspended animation state and then declare death and issue death certificate (Box 13.1).

POSTMORTEM COOLING (SYNONYM: ALGOR MORTIS)

(Algor = Coldness/Chill, Mortis = Of Death)

A brief discussion on postmortem cooling is presented below:[11-13,16,17,20,21,27,28]

Definition

Postmortem cooling is defined as the cooling of the dead body.

Mechanism of Cooling

Normal human body temperature is 37.2°C (98.4°F). In life a balance between heat production and heat loss maintains this. After death, the dead body behaves like an inert object and looses heat by the conduction, convection and radiation, till it reaches equilibrium with the temperature of its surroundings. However, during the first 2 to 3 hours after somatic death, *'living tissues'* and *'bacterial action'* continues to produce heat.

Measuring the Cadaveric Temperature

Usually three methods are in practice using *cadaveric/rectal thermometer*, which is an ordinary *chemical thermometer* calibrated in degree centigrade:

1. By measuring the *rectal temperature* by introducing the bulb, 8 to 10 cm deep into the *rectum*.
2. By measuring the *inner core body temperature* by placing the bulb in contact with the *inferior surface of liver* through a midline incision.
3. By measuring *vaginal temperature* by inserting the bulb 8 to 10 cm deep into the *vagina*.

Rate of Cooling

It is not uniform. In temperate climate, the cooling rate is:

- In first 2 to 3 hours there is no cooling
- In the next 6 hours it is about 1.5°C/hour
- In later 6 to 12 hours it is about 0.9°C to 1.2°C/hour.

Thus, the whole body surface gets cooled by 10 to 12 hours of death. However, it is well established that the internal organs cool slowly (cools by 18-24 hours of death). Graphically the way in which a dead body cools is represented in Figure 13.5, which shows a sigmoid curve. An approximate idea of number of hours after death (postmortem interval—PMI) may be calculated by using formula presented below:

$$\text{PMI} = \frac{\text{Normal body temperature (37.2°C)} - \text{Rectal temperature of the cadaver}}{\text{Rate of temperature fall per hour}}$$

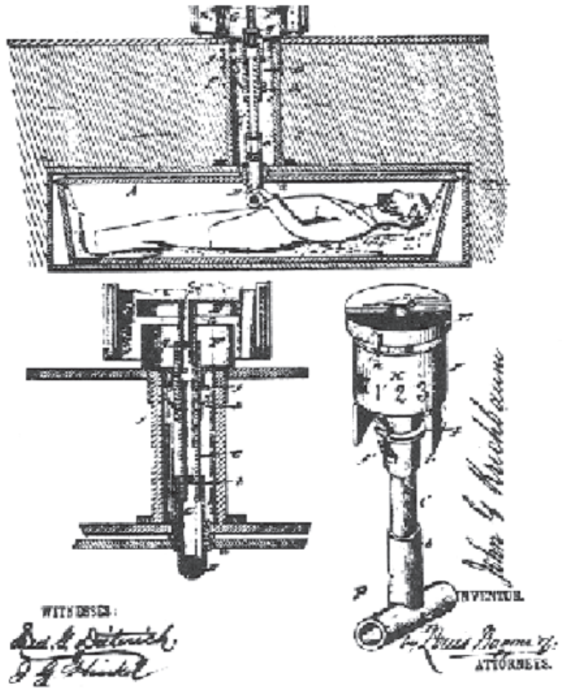


Fig. 13.4: Kirchbaum's device[37] for indicating life in buried persons, patent sketch, 1882

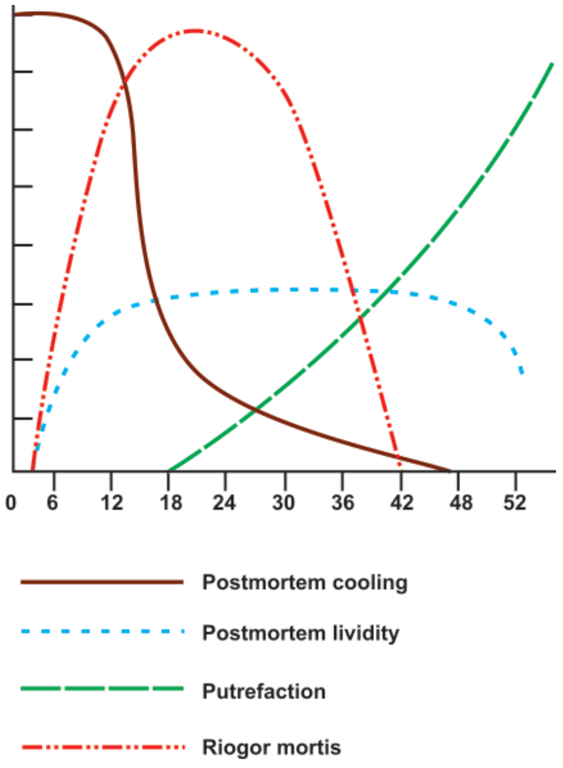


Fig. 13.5: Graphical representation of time related postmortem changes in a dead body

Graphical method of calculation of PMI: Record at least 3-4 temperature readings at 0.5 to 1 hour intervals and plot a graph with temperature v/s time. The interval at which it cuts 37.2°C is the postmortem interval (PMI).

Factors Controlling Rate of Cooling

Age: The bodies of young and middle-aged person that are relatively bigger, cool more slowly than bodies of children and old-aged people.

Condition of the body: Obese and well-nourished large bodies retains heat for long time.

Initial body temperature[28]: Higher temperature take long time in cooling.

Position of the body: Bodies in supine/prone position cool rapidly than bodies in crouched position.

Manner of death: Cooling of the body is more rapid in deaths due to chronic, wasting diseases, than in deaths due to accident or acute disease or apoplexy. However in deaths due to asphyxia, lightening, CO_2 poisoning, etc. cooling is slower.

Clothing: Bodies covered with thick garments cool slowly.

Atmospheric temperature: Bodies in warm/hot atmosphere, cooling is delayed.

Size of the room: Bodies in a small, ill-ventilated room, cooling is delayed.

Medium in which the body lies: Bodies immersed in running water cools rapidly than when exposed to air.

POSTMORTEM CALORICITY

(Meaning: Post = After; Mortem = Death, Calor = Heat)

Definition

Postmortem caloricity is defined as an initial rise of temperature of the body after death, followed by cooling as usual.

Causes

Sunstroke, Pontine hemorrhage, tetanus, strychnine poisoning, acute bacterial/ viral infections, intense asphyxia, muscular activity, etc.

EYE CHANGES

Soon after death, eyes loose their luster. At death eyes look staring and vacant, pupils assuming nearly mid-position. Various changes in the eye after death are as follows:[11-13, 16-17, 20-21, 27-28]

- **Loss of corneal reflex:** is not only observed in death, but also in all cases of coma. Hence is not a reliable sign of death.
- **Cornea turning opaque:** Cornea looses its glistening appearance, becomes dull and opaque and looks like dimmed glass. This is due to lack of lachrymal moistening. The finding is hastened when eyes are open after death. This is due to drying and is retarded by closure of eyelids after death. However, in closed eyes by 24 hours usually the cornea turns cloudy. The glistening appearance of cornea may get dimmed even before death in conditions like-uremia, cholera, narcotic poisoning; whereas the cornea may retain its glistening appearance for sometime after death in cases of death due to carbon monoxide (CO), cyanide (HCN) poisoning, death from apoplexy, etc.
- **Flaccidity of the eyeball** – due to fall of intraocular tension, this is dependent on the arterial pressure for its maintenance. This causes the eyeballs to sink into orbits.
- **Pupillary changes** – usually dilated at death, which constricts later due to development of rigor mortis of iris (in narcotic poisoning it remains constricted).

- **Vitreous biochemical changes** – though a steady rise in *potassium* is noticed in vitreous humor after death it is found to be not reliable.
- **Retinal vascular changes** – When viewed with an ophthalmoscope, the retina provides one of the earliest positive signs of death, the '*trucking*' of blood in the retinal vessels. This is due to loss of blood pressure after death, which allows the blood to break up into *segments*. This phenomenon occurs all over the body, but only in retina it is accessible for direct viewing. Fundoscopy reveals that the blood stream/column in these vessels get rapidly *segmented* or *fragmented* within 10 seconds to one minute of death, followed by *pallor* of the *optic disc* later. Segmentation of blood column is called, as '*trucking*' or '*rail-roading*' movement of red cells as it resembles to *unbraked goods-train wagons* on stoppage. This is also an indication of *cerebral death*.
- **Tachenoire (*Taches Noire De La sclerotica*)** – is an artifact of drying consisting of brownish discolouration of sclera in exposed parts, when eyelids are not closed at death and left open for 2 to 3 hours. Cause of this change is mainly the formation of cellular debris and dust settling thereon. It is yellowish to start with, turns reddish brown and lastly black in course of time.

SKIN CHANGES

Various postmortem changes of medicolegal significance in the skin are enumerated and discussed as below:[11-13,16-17,20-21,27-28]

- **Loss of elasticity** – prevents gaping of the incised wound if caused after death.
- **Colour changes** – ashy white, pallor, due to draining blood from blood vessels of skin. Thus, after death, skin turns pale, lusterless and ashy white; more evident in fair skinned individuals. However, if death is associated with agonal spasm, the face remains congested, bluish black for sometime after death. Skin may have yellowish/reddish discolouration due to jaundice or poisoning due to carbon monoxide (CO) and cyanide (HCN).
- **Changes in the lips** – lips turn brownish, and hard due to drying.
- **Postmortem lividity** – This change in the skin is of great medicolegal importance.

POSTMORTEM LIVIDITY

(*Synonyms: Postmortem Stains, Postmortem Hypostasis, Postmortem Suggilation, Postmortem Vibices, Livor Mortis*).

Definition

Postmortem lividity is the purplish or reddish purple areas of discolouration of skin and organs after death due to accumulation of blood in dependent parts of the body and seen through the skin (Figs 13.6 and 13.7).

Mechanism

After death, blood in its fluid state gravitates into the toneless capillaries and venules of the 'rete mucosum' in the dependent parts of body and causes capillovenous distension, which through the skin imparts a discolouration to the area involved.

Site

It can involve skin, all tissues and viscera. In body which was supine at death, it is seen on the back, ears and posterior aspect of the body except the pressure areas (contact areas) like bony prominences, buttocks, (See Fig. 13.7) the neck area with a tight collar, neck tie, etc.

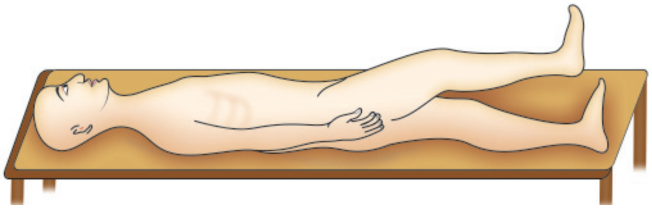


Fig. 13.6: Diagrammatic representation of postmortem lividity

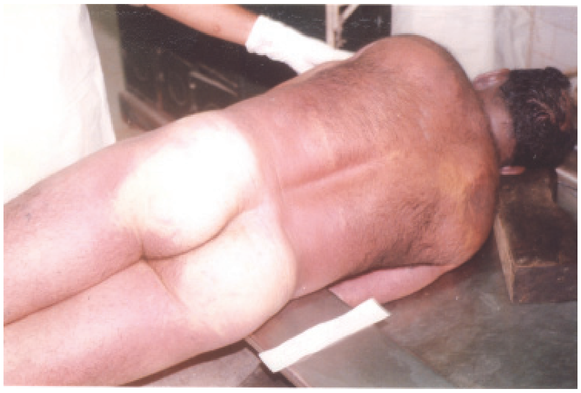


Fig. 13.7: Postmortem lividity in the back with contact flattening in the buttocks. (*Courtesy:* Dr NG Revi, Professor and Head, Dept. Forensic Medicine and Police Surgeon, Medical College, Trichur, Kerala)

Time of Onset

The lividity appears by 1 to 3 hours of death. Initially it will be in the form of a series of mottled patches, which gradually increases in size and then coalesce by 3 to 6 hours, and becomes fully developed and fixed in 6 to 8 hours of death.

Fixation

Generally, it is said that if pressure applied by a thumb blanches the area, the lividity is not fixed and time since death is less than 8 hours. If the area does not blanch, lividity is fixed and the time since death is more than 8 hours. Fixation is due to the diffusion of haemoglobin through capillary walls and staining of the tissues, permanently.

According to Knight B, fixation of lividity is untrue as there can be '*secondary gravitation*' and if left in new position for further few hours, some or all hypostasis may slip down to the most dependent areas then.[28]

Colour of Postmortem Lividity

Usually the colour of lividity is purplish. However, it will change depending on the cause of death, and Table 13.5 enumerates the various colour changes along with its causes.

Hypostasis of the Visceral Organs/Internal Organs

Postmortem hypostasis is also seen in the dependent parts of visceral organs, depending upon position of the body. In a dead body lying on the back, it will be evident in the posterior portion of the cerebral lobes, cerebellum, in pial vessels in the posterior

Table 13.5: Postmortem stains—changes in colour relating to its causes

Cause of death	*Colour*
Hemorrhage	Pale purple
Anemia	Pale purple
Asphyxia	Dark purple
Poisoning by:	
• Carbon monoxide	Cherry red
• Cyanides	Bright pink
• Potassium chlorate	Chocolate brown
• Phosphorus	Dark brown
• Nitrites	Red brown
• Hydrogen sulphide	Bluish green
• Opium	Grayish/black
Death due to burning/cold	Cherry red
Death due to septic abortion (Cl. Welchii infection)	Brown/pale bronze mottling
Death due to hypothermia	Bright pink (Low Tissue metabolism fails to take up O_2)
Methemoglobinemia	Brownish red

fossa, posterior surface of heart, lungs, liver, kidneys, spleen, larynx, stomach and intestines. Hypostasis in heart may be *mistaken* for myocardial infarction, hypostasis in lung may resemble pneumonic consolidation, and hypostasis of the dependent portion of intestinal coils may resemble *strangulated* coils. Hypostatic engorgement of gastric mucosa may simulate *irritant* poisoning like changes. Table 13.6 differentiates *visceral hypostasis* from *inflammatory congestion*.

Medicolegal Importance of Postmortem Lividity

- Can assess time since death.
- Can decide the position of the deceased at the time of death.
- Can establish the cause of death (See Table 13.5).
- It may be mistaken for a contusion. Table 13.7 enumerates the differences between a contusion and postmortem lividity.

MUSCLE CHANGES

Muscles in a dead body usually pass through *three stages*:[11-13, 16, 17, 20, 21, 27, 28]

- Primary flaccidity
- Rigor mortis
- Secondary flaccidity.

PRIMARY FLACCIDITY

Immediately after death, as the control from higher centres (brain) on each of the muscle is lost and every muscle of the body shows complete relaxation, e.g. pupillary dilatation, sphincteric relaxation, urinary/stool incontinence. However, these muscles can be still made to react to an electrical stimulus.

RIGOR MORTIS (SYNONYM: CADAVERIC RIGIDITY)

The literary meaning of the words are *rigor* meaning *rigidity* and *mortis* meaning *death*). Thus rigor mortis is the *postmortem stiffening/rigidity* of the muscles in a dead body (Figs 13.8 and 13.9).

Mechanism

The physico-chemical basis of rigor mortis is complex. The changes occurring under these phenomena are mainly due to the irreversible fusion of two contractile elements, the essential proteins, namely *actin* and *myosin* filament of muscle fibres into a *dehydrated stiff gel*, making them remain in a rigid inextensible (Fig. 13.10) state. This will persist till the actin and myosin filaments undergo *autolysis*. Rigor mortis is basically due to the *depletion* of adenosine triphosphate (ATP) reserve from the muscle. It is reported that when the ATP level falls to 85 per cent of normal level, rigor mortis is initiated and it will be maximum when this level is 15-20 per cent of normal.[28]

ATP is the main source of energy for muscle contraction. Muscle needs a *continuous* supply of ATP to contract because the amount of ATP present in muscles is sufficient to sustain muscle contractions for only a *few seconds*. After death, generation of ATP *stops*, though consumption *continues*. With fall of ATP levels, actin and myosin filaments become permanently complexed (fused) *into a dehydrated gel* and with this rigor mortis *sets in*.

Any violent muscular *exertion* prior to death will produce a *decrease* in ATP levels. This will *speed* up the *onset* of rigor mortis, since *no* ATP is produced after death. Some of the *factors* that can cause depletion of ATP prior to death are violent or heavy exercise, severe convulsions and high body temperatures. All these factors in turn can bring about rapid onset of rigor mortis within minutes in some cases, and in rare instances, instantaneously, which is thus called as '*instantaneous rigor*' or '*cadaveric spasm*'.

Rigor mortis *disappears* with onset of *decomposition*. Cold and/or freezing will delay onset of rigor mortis as well as prolong its presence. Rigor mortis, when it develops, it involves all the muscles at the *same time* and at *same rate*. However, it becomes most evident in the *smaller muscles*. Thus rigor mortis is said

Table 13.6: Comparison of internal hypostasis and inflammatory congestion of viscera

Features	*Internal hypostasis*	*Inflammatory congestion*
Position	Only in dependent parts of organ	Generalized
Redness	Discrete and irregular	Uniform all over the organ
Mucosa	Dull and lusterless	Angry looking
Inflammatory reaction	Negative	Positive

Table 13.7: Differentiating the contusion from postmortem lividity

Features	*Contusion*	*PM Lividity*
Situation	Seen anywhere depending on site of injury	Occurs only in the dependent parts of the body
Distribution	Lies under the epidermis, in between layers of dermis/still deeper	Always superficial confined to the epidermis
Swelling	Positive	Negative
Margins	Not well defined, due to tissue reaction and age of the wound	Always sharply defined
Contact flattening	Negative	Positive
Gravity shifting	Negative	Positive (prior to fixing)
Abrasion	Positive	Negative
Colour changes due to healing	Positive	Negative
Blanching on pressure	Negative	Positive (prior to fixing)
On incision	Extravasation of blood seen in the tissues around the bruise and this cannot be washed away with stream of water.	Blood is within the blood vessels, which oozes out of cut ends of capillaries and venules and this can be easily washed with gentle stream of water.
Histopathology examination	Presents vital reaction and is confirmatory	Not so

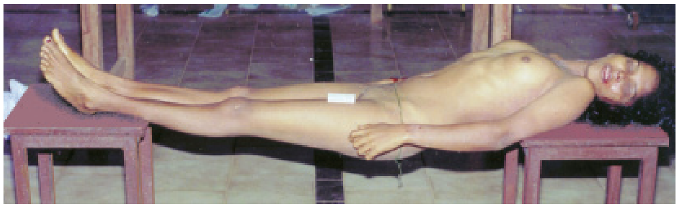


Fig. 13.8: Rigor mortis—body is stiff and can be made to remain so on two supports at either ends of the body (heels and head)

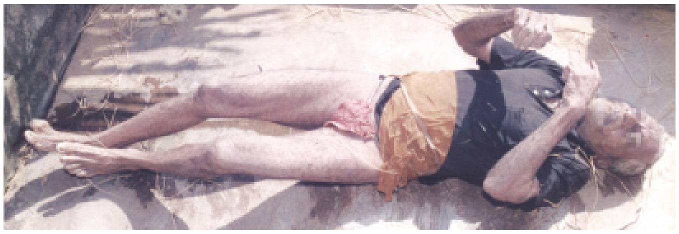


Fig. 13.9: Dead body of drowning recovered from a drinking water well. *Note:* Well established rigor mortis and so also instantaneous rigors in both hands

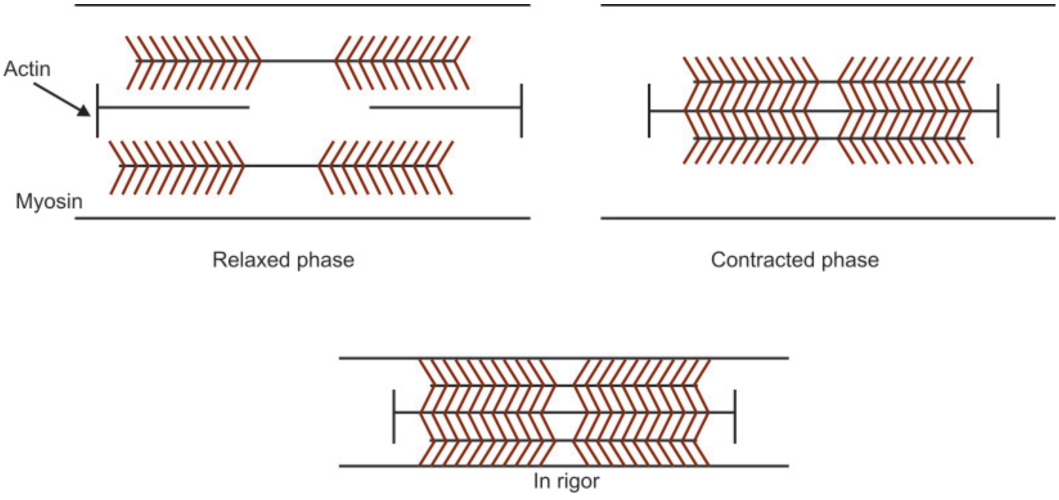


Fig. 13.10: Diagrammatic representation of arrangement of actin and myosin filaments in muscle fibres in relaxed phase, contracted phase and in rigor

to appear first in the smaller muscles, such as the muscles of eyelids, face, jaw and then gradually spread to larger muscle groups (Table 13.8). The classical presentation of rigor mortis in order of appearance is – jaw, upper extremities, and lower extremities in a proximo-distal distribution. It passes off also in the same order.

Testing for Rigor in Cadaver

Testing for rigor in cadaver is done by trying to open eyelids, depressing the jaw, gently bending the neck and various joints of the body and noting the degree (complete, partial or absent) and distribution.

Onset of Rigor

Gradually muscles begin to stiffen. It first occurs in the involuntary muscles and then in the voluntary muscles, which may be outlined as summarized in Table 13.8.

Time Taken for Onset of Rigor Mortis

Upper part of body—stiffens by about 7 to 9 hours of death. Whole body—stiffens by 11 to 12 hours of death.

Secondary Flaccidity

When the molecular death has occurred in each of the actin and myosin filaments, the muscular stiffness that had developed earlier in the previous stage will begin to disappear slowly and gradually, relaxing the body back to the original state [however, now it shows no response to any stimulus]. Sequence of this relaxation commences in the same order as that of commencement of the stiffening, starting from eyelids downwards. Just as for developing complete rigor mortis 12 hours time is required, passing off of rigor also require another 12 hours.

Position Opted on Complete Development of Rigor Mortis

Rigor mortis does not cause any significant contraction of muscles.[27] However, if a person opts a particular position at death and remains like that, rigor mortis will be established in same position. This can provide a clue on position opted by the deceased at the time of death.[20,27,28]

Postmortem Interval and Rigor Mortis

In general, rigor mortis sets on within 1 to 2 hours after death, and is well developed from head to toes in about 12 hours. It is then maintained so far about 12 hours and passes off in about another 12 hours subsequently with onset of putrefaction. This is also known as *'March of rigor'* or *'Rule of 12'*. However, when rigor mortis sets in *early*, it will tend to pass off *quickly* (*vice versa* of this is also *true*).

Conditions Resembling Rigor Mortis

- Cadaveric spasm
- Heat stiffening (pugilistic attitude)
- Cold stiffening
- Gas stiffening.

Cadaveric Spasm (Synonym: Instantaneous Rigor, Death Clutch, and Cataleptic Rigidity)

Cadaveric spasm is a condition wherein stiffening occurs in certain groups of muscles, which were already in a state of contraction at the time of death. It is a rare form of virtually instantaneous rigor that develops at the time of death with no prior phase of primary flaccidity.

Table 13.8: Various muscles of the body and the time interval of developing rigor mortis

Muscle sites	*Time interval after death*
Eyelids	3 - 4 hours
Face and jaw	4 - 5 hours
Neck and trunk	5 - 7 hours
Upper extremities	7 - 9 hours
Legs	9 -11 hours
Finger and toes	11-12 hours

Mechanism: Mechanisms of cadaveric spasm though not clear; explanation given below is accepted:

- In cases wherein somatic death occurs rapidly, followed by early muscle changes, e.g. in violent deaths such as drowning.
- Great emotional tension, at the time of death, e.g. a soldier in battlefield with fear, a person committing suicide by cut-throat injury.

Findings: Drowning victims hands are tightly clenched gripping seaweeds/plants (Figs 13.11 and 13.12). A cutthroat suicide victim will tightly hold the blade/knife used to cut in a firmly gripping hand. Findings thus suggest antemortem drowning/cut throat injury.

Medicolegal importance: It can help not only to assess cause and motive of death but also the attitude of the deceased at the time of death.

Heat Stiffening (Synonym: Pugilistic Attitude)

The attitude of a boxer in self-defense - taken up by the body in death, due to heat coagulation of muscle proteins (Fig. 13.13).

Cold Stiffening

Here muscles are stiff due to solidification of fat and freezing of body fluid due to the extreme cold. Noise of cracking of the ice is heard on attempting the flexed elbow to open.

Gas Stiffening

This is due to accumulation of *putrefaction gases* in the tissues so as to cause a false rigidity resulting in stiff limbs (Fig. 13.14).

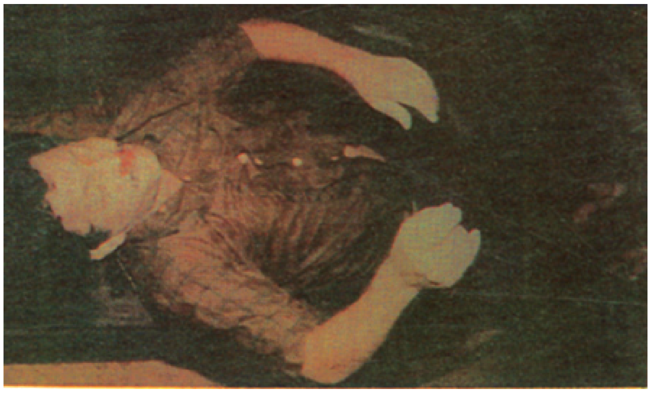


Fig. 13.11: Cadaveric spasm of right hand (in a drowning victim)

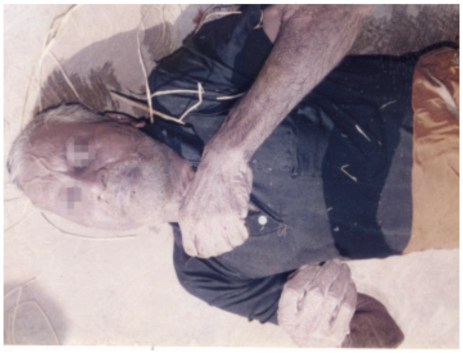


Fig. 13.12: Cadaveric spam of both hands (in a drowning victim)

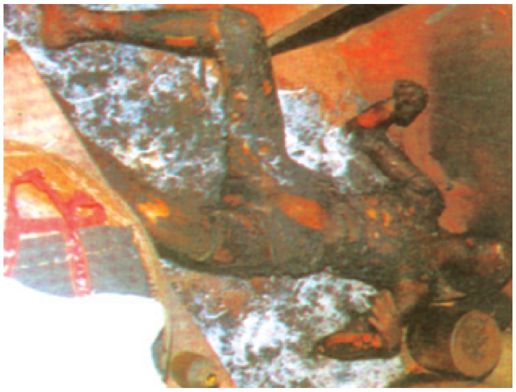


Fig. 13.13: Pugilistic attitude (in a burns victim)

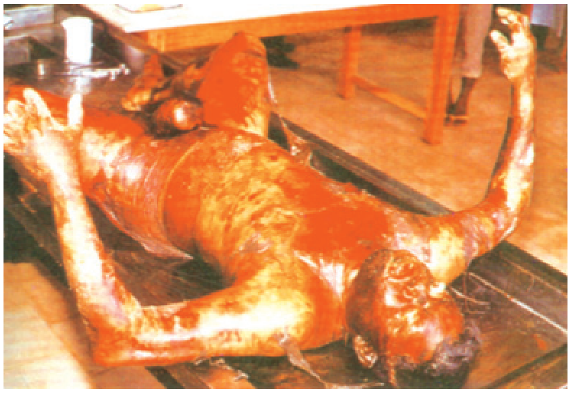


Fig. 13.14: Postmortem gas stiffening

Factors Affecting Rigor Mortis

- **Diseases** – Wasting disease or any condition leading to extreme exhaustion—rapid onset of rigor mortis, followed by rapid passing off of the same, lasting for a short duration.
- **Health** – In a healthy person, onset of rigor mortis is slow. It also passes off slowly, lasting for a longer duration.
- **Age** – In old age, starts early, lasts for a short duration, and passes off quickly.
- **Temperature** – In warmer temperature, onset and disappearance are both hastened. In colder temperature onset and disappearance are both retarded.

Rigor mortis in other tissues with medicolegal significance[28]

- **Eye** – Iris is affected so that ante-mortem constriction or dilatation is modified.
- **Heart** – Rigor of heart causes the ventricles to contract, which may mimic and look like left ventricular hypertrophy.
- **Skin** – Rigor in the erector pile muscles in the skin, can cause a dimpling effect or 'goose–flesh' appearance with elevation of the extraneous hairs.

POSTMORTEM DECOMPOSITION

Postmortem lividity and rigor mortis occur relativity soon after death when somatic death has occurred, but cellular death is incomplete. Death as discussed earlier is a process and not an event, and while the cells of some tissues are still *alive* and even capable of movement, others are *dying* or *dead*.

Process of decomposition begins in some cells while the others are still alive, and this overlap continues for several days in temperate climate. Decomposition involves *two* processes (i) *autolysis* and (ii) *putrefaction.*[27] Each of these is discussed in detail.[11-13,15-17,20-21,26-30] However, when we discuss about decomposition, we *usually mean* putrefaction. Decomposition differs from body to body, from environment to environment and even from one part of the body to another in a corpse.

Autolysis

The actual literal meaning of the words is *Auto* meaning self and *lysis* meaning destruction. In molecular death, cell death is ultimate and with this, each cell release certain enzymes, which are responsible for lysis of the tissues. Autolysis is therefore a completely aseptic chemical process and organs rich in enzymes undergo autolysis earlier than organs with less amounts of the enzymes. Thus pancreas autolyses much earlier than the heart.

Time required for autolysis Usually it commences within 3 to 4 hours of death and completes in about 2 to 3 days or few more days.

PUTREFACTION

Putrefaction is the final process observed in the cadaver, leading to the gradual dissolution and liquefaction of the tissues. This to most of the forensic experts is though synonymous with *decomposition*, is due to *bacterial fermentation*.

Mechanism

Putrefaction is brought about in a dead body by bacterial action.

Bacterial Action

Certain bacteria can produce proteolytic and other enzymes, mainly lecithinase which are capable of bringing lysis of tissues, e.g. *Cl. welchii, E. coli, Staphylococcus, Streptococcus, B. proteus, etc.* These organisms, which are responsible for putrefaction, are both *anaerobic* and *aerobic*. Factors favouring bacterial action are:

- Marked *increase* in tissue *hydrogen ions*
- Rapid *decrease* in *tissue oxygen* levels
- Presence of adequate *moisture* in the body
- Conveniently *warm* atmospheric temperature (70°-100°F).

Body tissues gradually dissolve mainly into *gases, liquids* and *salts*. Eventually all the organic matter will disappear leaving only small quantity of inorganic matter especially in the *bones* until they also crumble into inorganic dust over a period of time.

Source of these bacteria: Normally they originate mainly from the large intestine, and partly from respiratory tract (*intrinsic*) and open skin wounds (*extrinsic*). After death the *protective mechanisms* being absent, within a short time they enter the blood vessels and spread rapidly throughout the body. Thus highly vascular organs, situated more proximally to the source of bacteria, are likely to putrefy.

Onset of Putrefaction

Tissue undergoing putrefaction mainly shows three changes:

- External changes
- Colliquative putrefactive changes
- Internal changes.

External Changes

External changes include following:

- Colour changes of skin
- Marbling of skin
- Evolution of foul smelling gases
- Appearance of maggots.

Colour Changes of Skin

The first external evidence of putrefaction in a dead body exposed to air is the formation of greenish blue/greenish black discolouration of the skin, (due to the formation of *sulphmeth-haemoglobin*) in localized areas, which later on coalesce and spread all over the body.

Mechanism–Usually *sulphmeth-haemoglobin* is formed in the intestines. Lyses of RBCs release the *haemoglobin (Hb)* which, in combination with the *hydrogen sulphides* available in the intestinal sources, form *sulph-meth-haemoglobin*. This imparts a greenish blue/greenish black colouration. As such haemoglobin can also stain the tissues around directly producing a red/reddish brown colouration initially, which turns greenish yellow later. Haemoglobin released in to the blood from lysed RBCs can also successively discolour other parts of the body such as neck, face, shoulders, arms and legs, etc. which soon coalesce to discolour the whole body. Figure 13.15A presents a schematic representation of various events responsible for the colour changes of putrefaction.

Site–In a dead body undergoing decomposition in air, the earliest site showing *discolouration* of decomposition is on the abdomen in the right iliac fossa (Fig. 13.15B), over *caecum*. Anatomically caecum lies very close to the anterior abdominal wall and is also laden heavily with bacteria, rendering the colour changes of putrefaction to right iliac fossa be visible first in the cadaver undergoing decomposition. Order of appearance of colour change in body:

- In case of decomposition in air—the colour changes appear in an order of the abdomen, chest, face, legs, shoulders, arms.[16]
- In case of decomposition inside water—the colour changes appear in an order of face, neck, thorax, shoulder, arms, abdomen and legs.[27]

Colour Changes of Skin and Postmortem Interval

- Over right iliac fossa and flanks—12 to 24 hours.
- Whole body—48 hours.

Marbling of Skin

This is a colour change seen in the form of mosaic-like pattern on the skin on certain parts of a cadaver where the veins converge[27] (Fig. 13.16).

Mechanism–Blood, which is decomposing releases, haemoglobin, reacts with hydrogen sulphide (H_2S) gas with development of greenish black discolouration of blood vessels, which may become visible through the skin (especially in fair skinned individuals).

Site–Commonly seen over the right iliac fossa, roots of the limbs (Fig. 13.16) and neck, etc.

Marbling and postmortem interval–In summer, it just commences in 24 hours of death and becomes prominent in about 36 to 48 hours after death.

Evolution of Foul Smelling Gases

As the colour changes commences over the abdomen, the body gradually starts emitting *unpleasant* and *offensive* smell due to formation and collection of *decomposition gases* by the breaking down of body *proteins* and *carbohydrates* with in 12-18 hours of death in summer. These gases include:

- Hydrogen sulphide
- Ammonia
- Phosphoretted hydrogen
- Methane
- Marsh gas
- Carbon dioxide, etc.

These gases though non-inflammable in early stages; with advanced putrefaction and more of H_2S production, if ignited, can burn with blue flame. However, these gases are medicolegally important because of their *pressure effects*.

Site–Usually gases begin to accumulate inside the *hollow viscera*, e.g. inside the intestines; under the *skin*, e.g. postmortem blisters (Figs 13.17 and 13.18) and inside the *solid viscera* eventually, e.g. foamy liver (Fig. 13.19).

Effects–various pressure effects of the gases formed are enumerated below:

1. *False rigidity (Gas Stiffening)* – due to gas under the skin and mimic rigor mortis (see Fig. 13.14).
2. *Bloating of the body* – is often first noticed in the face, wherein features such as eyelids, nose, cheeks becomes swollen, eye balls bulge out (Fig. 13.20), tongue turns black and protrudes out between teeth and lips (Fig. 13.21A). Breasts in female are swollen enormously (Fig. 13.21B). Distention of abdomen may occur, which may at times, burst open with bursting noise (Fig. 13.21C).

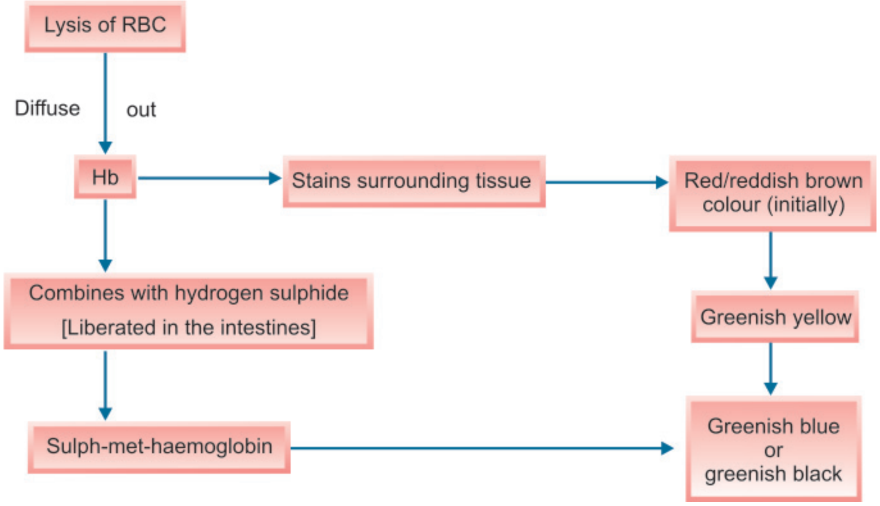

Lysis of RBC
Diffuse
out
Hb
Stains surrounding tissue
Red/reddish brown colour (initially)
Combines with hydrogen sulphide [Liberated in the intestines]
Greenish yellow
Sulph-met-haemoglobin
Greenish blue or greenish black

Fig. 13.15A: Schematic representations of various events of colour changes during putrefaction

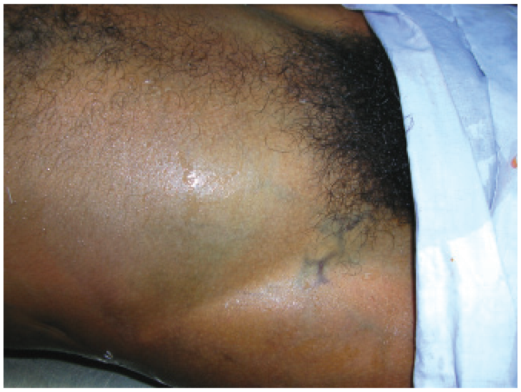


Fig. 13.15B: Earliest site of discolouration of decomposition is in the right Iliac fossa (*Courtesy:* Dr Ritesh G Minorges, Assoc Prof. of Forensic Medicine, KMC, Mangalore, Karnataka)

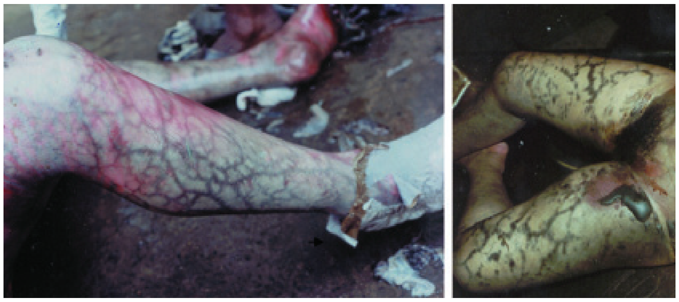
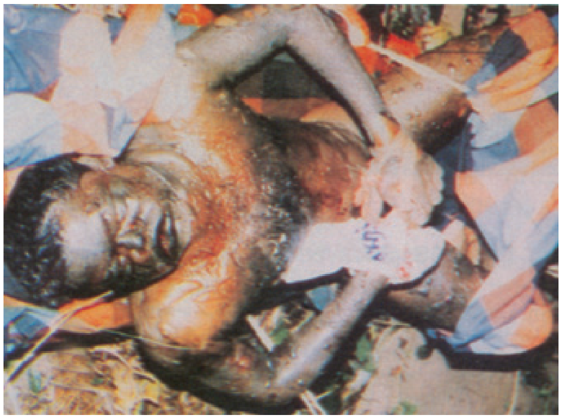


Fig. 13.16: Marbling on left leg and thighs (*Courtesy:* Capt. Dr B Santhakumar, Prof and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai)



Figs 13.17: *Putrefactive changes:* Multiple postmortem blisters of varying sizes on the entire body

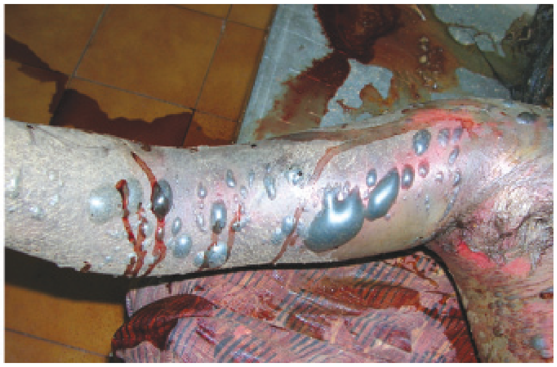


Fig. 13.18: Postmortem blisters of varying sizes on the right arm

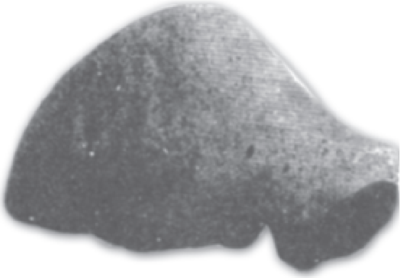


Fig. 13.19: Effects of postmortem gas accumulation in liver (foamy liver)

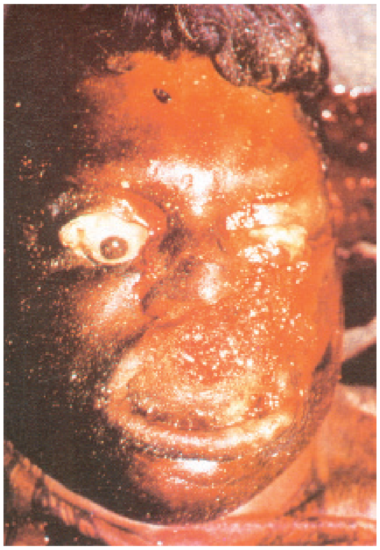


Fig. 13.20: *Putrefactive changes:* Effects of postmortem gas accumulation—Bloating of face (Note—There is a sutured scar on the midchin)

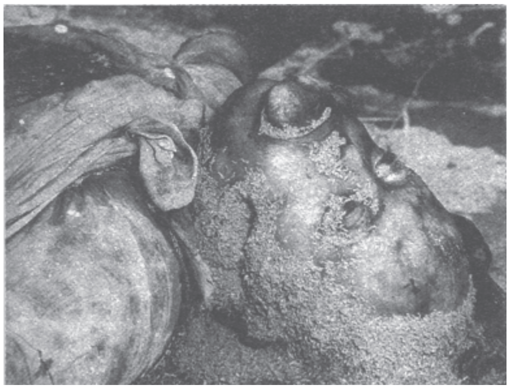


Fig. 13.21A: Cadaver in a state of putrefaction infested with maggots (*Courtesy:* Dr PG Paul, Former Professor and HOD Forensic Medicine, KMC, Mangalore, Karnataka)

3. *Shifting of postmortem hypostasis* – as blood undergoes postmortem liquefaction – may shift from one area to other depending on direction of gas pressure.
4. *Changes in the skin, hair and wounds*
   - *Putrefactive blisters* – Several blisters are formed over different parts of the body surface, due to accumulation of putrefactive gases under the skin. These are small

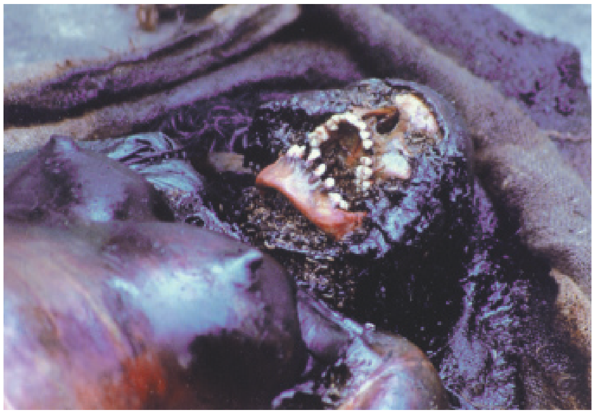


Fig. 13.21B: Decomposition bloating of the body—Note enormously swollin breasts, partial skeletanisation and maggots infestation in the neck (*Courtesy:* Capt. Dr B Santhakumar, Prof and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

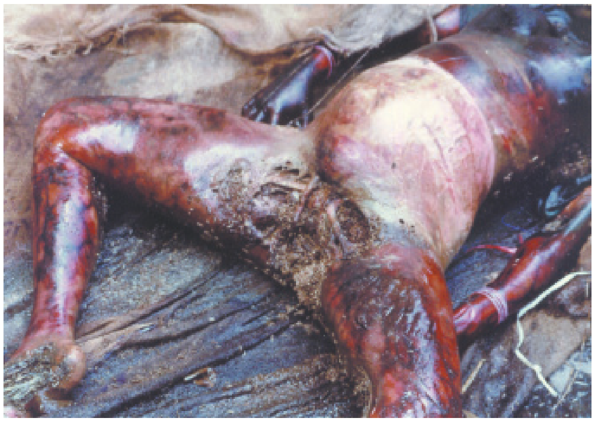


Fig. 13.21C: Decomposition, and maggots infested in the genital region (*Courtesy:* Capt. Dr B Santhakumar, Prof and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

initially to begin with, and then they gradually enlarge and coalesce to become larger in size. On puncturing such blisters release of postmortem gases occurs, which helps to differentiate it from blisters due to burns.

- *Denudation* of the cuticle
- *Degloving* and *Destocking* – This refers to postmortem slipping of the skin of hands and feet (Figs 13.22A and B) respectively.
- *Postmortem bleeding* from wounds.
- *Hair and nails detachment* – Hairs and nails become loose and can be pulled or detached easily.

5. *Extrusion of decomposition fluid* (*purge fluid/postmortem purge*) which is bloodstained and will drain from the mouth and nostril or rectum and vagina, due to the intra-abdominal pressure of putrefying gases on diaphragm. This may be misinterpreted by the inexperienced as bleeding due to antemortem head injury or intra-abdominal or pelvic injury, respectively.
6. *Emptying of heart*—due to pressure of gas in abdomen on diaphragm.
7. Changes in male genitalia—enormously *swollen* due to collection of gas (Fig. 13.23).
8. *Changes in female genitalia and uterus*—vulva turns *pendulous* (and may mimic findings of sexual offenses). If

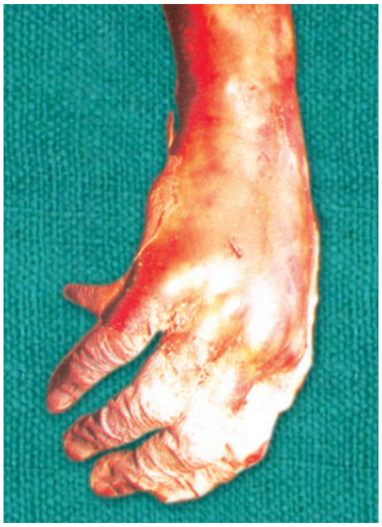


Fig. 13.22A: *Putrefactive changes:* Degloving

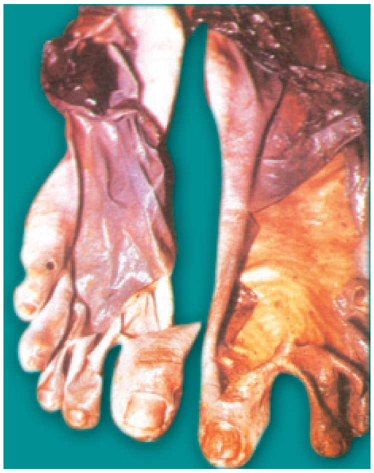


Fig. 13.22B: *Putrefactive changes:* Destocking

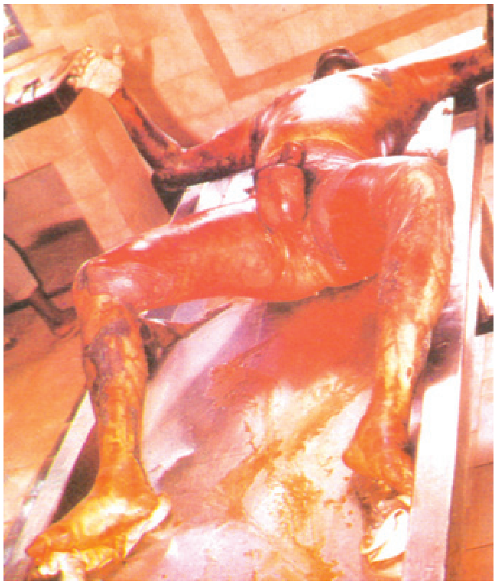


Fig. 13.23: *Putrefactive changes:* Effects of postmortem gas accumulation: Changes in male genitalia distended thorax and abdomen and stiffening of the limbs

the uterus is gravid it may present with expulsion of the fetus known as *postmortem delivery.*[11,20]

Postmortem Interval by Putrefying Gas Collection

In Summer-in General

- Gas collection inside the hollow viscera—12 to 18 hours.
- Gas collection under the skin and inside solid viscera—18 to 48 hours.

CADAVERIC ENTOMOLOGY

(Forensic Entomology/Fauna of Cadaver/Appearance of Maggots)

Any putrefying human body attracts houseflies (Musa) and such other insects easily. Once these organisms invade the dead body, they start to live on the cadaver, and undergo all stages of their life cycle, (*metamorphosis*). These flies may lay their eggs on fresh corps, between the lips, or the eye lids, in the nostrils, genitalia, or in the margin of a fresh wound, within a few minutes after death, and in some cases even before death during the *agonal period*. When the skin decompositions begins, the eggs can be deposited anywhere. In *eight to twelve hours* in summer these eggs hatch out *larvae,* i.e. *maggots*, which start invading the dead body, crawl into the *interior* of the body, produce powerful *proteolytic enzymes*, and *destroy soft tissues*. The maggots can burrow under the skin and make tunnels and sinuses, which *hasten* putrefaction by allowing *air* and *bacteria*.

Depending on the various *stages of life cycle* of a type of insect, at given time in a cadaver, *postmortem interval (PMI)/ time since death* can be assessed. A rough idea of establishing postmortem interval by this methodology is presented in Figure 13.24. Thus, if a cadaver recovered in a state of *putrefaction* and is infested with *maggots*, (See Figs 13.21A to C) time since death can be assessed to be 4 to 5 days. The scientific study of *insect fauna* on the cadaver constitutes a special branch '*forensic entomology*'.

Colliquative Putrefaction[17,20,27]

(*Synonym*: *Colliquative liquefaction, colliquative liquefying putrefaction*)

During this stage *liquefaction* of tissues takes place. Here the wall of the abdomen softens and *bursts open* with protrusion of stomach and intestines. The thorax also bursts, especially in children. The diaphragm is pushed upwards. The body fats especially the omental, mesenteric and perirenal may *liquefy* into a translucent yellow fluid filling body cavities between organs. The tissues become soft, loose and are converted into a thick, semi-fluid, black mass, to fall off separately, exposing the bones. The cartilage and ligaments are similarly softened (Fig. 13.25). Decomposition may differ from body to body, from environment to environment and from one part of the same body to another. At times one part of the body may be *mummified*, while rest of the body may show *liquefying putrefaction*.

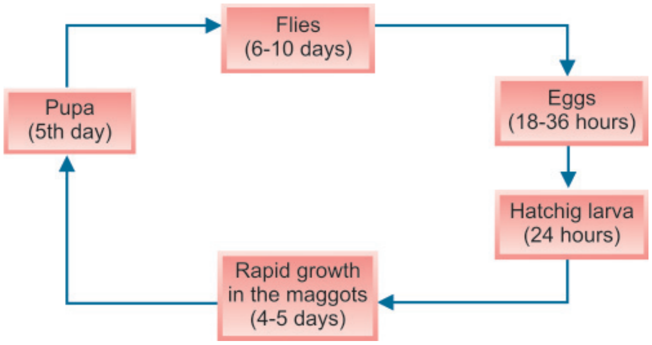


Fig. 13.24: Life cycle of housefly and establishing the PMI

Colliquative putrefaction and time since death—It usually commence on *fifth* day after death and is complete by *tenth* day.

Internal Changes

Along with the external changes, internal organs also undergo putrefactive changes. However, this process is greatly affected by the structure of each organ. Based on the ascending order of frequency of occurrence of putrefaction, the various organs are listed as follows:

Rapidly Putrefying Organs

- Larynx and trachea
- Brain of infants
- Stomach, intestines and spleen
- Liver (change called *foamy liver*) and lungs
- Adult brain
- Pancreas

(Note: The lining of the intestines, Adrenal medulla and Pancreas autolyse within hours of death).

Slowly Putrefying Organs

- Heart
- Kidneys, bladder, uterus (gravid)
- Skin, muscles and tendons
- Blood vessels—initially the intima (inner surface) of major blood vessel such as *aorta* start showing *brownish discolouration*
- Uterus (nulliparous) and prostate.

Putrefactive changes observed in each of these various viscera are presented in Box 13.2.

Medicolegal Importance – Presence of prostate or uterus (nulliparous) helps in the identification of sex in a highly putrefied body *(Note: Prostate and non-gravid uterus resist putrefaction to the greatest extent).*

Conditions Influencing Putrefaction

Conditions that influence the initiation of putrefaction are:[20,27]

- Presence of good air,
- Plenty of suitable microorganisms,
- Availability of moisture and
- Enough of warmth.

However, the rate of putrefaction depends on other factors such as:

- Condition of body,
- Cause of death,
- Surrounding atmosphere and
- Medium in which the body lies.

Each of these factors is discussed in detail individually:[20, 26-30]

Condition of Body

Following factors can affect putrefaction:

- Fat, flabby body with heavy clothing (which keeps body warm), sepsis and presence of plenty of moisture - *hasten* putrefaction.

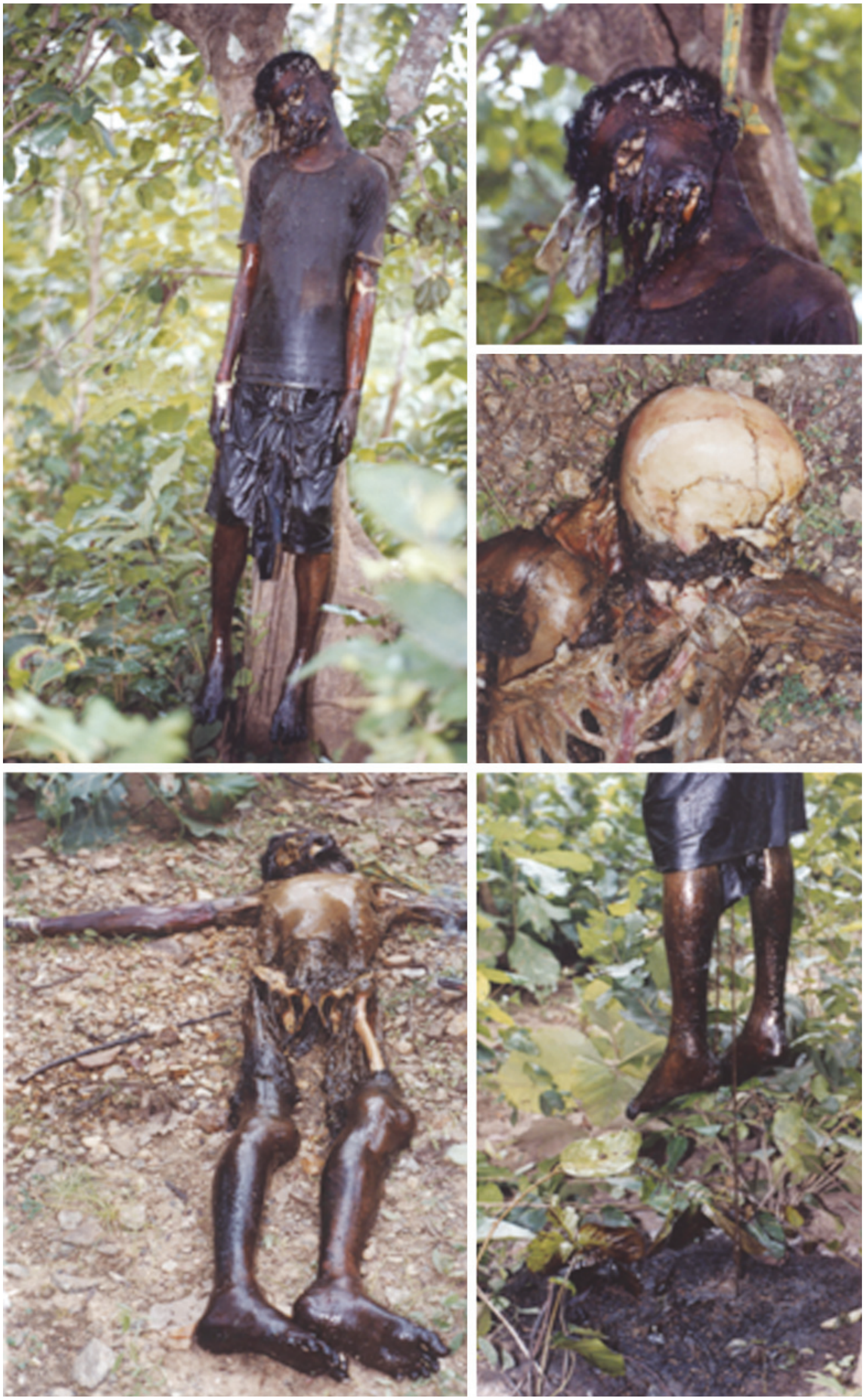


Fig. 13.25: Colliquative putrefaction. Note the changes body has undergone and thick, semi-fluid black mass fallen at the feet of the victim and body is partially skeletonized (*Courtesy:* Dr Binoy Kumar Bastia, Professor of Forensic Medicine, JNMC, Belgaum, Karnataka)

- Newborn infant—putrefaction is *slow* (reason: first half of the *intestines* is sterile).
- Slim and lean body with tight clothing, body lying on a stone or metallic surface, which can cool rapidly by conduction, can retard putrefaction.

Box 13.2: Common viscera changes of putrefaction (in summer)

1. Larynx and trachea
Mucosa in 12-24 hours (summer)/2-3 days (winter)
↓
Brownish red
↓
Greenish
↓
Softened

2. Stomach and intestines
In 24-36 hours (summer)/3-5 day (winter)
↓
Dark red irregular patches
Firstly involving posterior wall
↓
Later on involving anterior wall
↓
Gas blebs in the submucosa
↓
Transform into cysts of varying size into the lumen
↓
Softens, dark-brown colour
↓
Green/black soft, pulpy mass

3. Spleen
In 2-3 days
↓
Soft, pulpy mass
↓
Liquefy

4. Omentum and Mesentery
In 1-3 days (summer)
↓
Greyish green and dry

5. Liver
In 12-24 hr (summer)
↓
Soft and flabby
↓
In 24-36 hr (summer)
Multiple blister formation
↓
Cl. welchii form characteristic small clumps in a tissue space and produce gas
↓
Increase size (bubble like)
↓
Whole organ become greenish
↓
Coal black finding (newborn children liver putrefies earlier)

6. Gallbladder
Resists putrefaction for a long time but bile pigments diffuse early to adjacent tissue

7. Pancreas
Softens and haemorrhagic

8. Heart
Softens → Flabby → Cavity appears dilated

9. Lungs
Gaseous bullae appear under the pleura which are small, pale-red, on scattered and later initially coalesce
↓
Soften, collapse, and reduce into small mass

10. Kidneys
Renal tissue → Flabby with dull surfaces + appear parabolic, Cortex → dark → greenish

11. Adrenals
Cortex-softens from within around softened medulla
↓
Medulla liquefies
↓
Cyst like cavity

12. Bladder
Resist putrefaction for long-time if empty and contracted

13. Prostate
Resist putrefaction for very long time

14. Uterus
a. Nulliparous uterus is the last organ to putrefy
b. Gravid uterus/uterus after delivery – putrefy rapidly

- Embalmed cadavers – dead bodies are embalmed to delay decomposition. However, success depends on quality of embalming, the climate and nature of burial ground.

Cause of Death

Various manners of death that may affect putrefaction are as follows:

- Death by septic diseases, puerperal sepsis, kidney diseases with generalized (anasarca) oedema, etc. can *hasten* putrefaction.
- Death by chronic alcoholism can *retard* putrefaction.
- Death by poisons such as potassium cyanide, barbiturates, fluoride, phosphorous, endrin, datura, strychnine, yellow oleander, nicotine, arsenic, mercury, copper, antimony, lead, thallium, etc. can *resist* putrefaction.

The Surrounding Atmosphere

Various surrounding atmospheric factors which retard or prevent putrefaction are enumerated below:

- Airtight coffin buried deep (>2 meters)
- Death *in utero*, with intact membranes
- Too high temperature (>48°C)
- Too low temperature (<0°C)
- Body immersed in water, where no air enters.

The Medium in which the Body Lies

Temperature being same the ratio of rate of putrefaction of a dead body in air, water and buried in ground has been reported to be *1:2:8* respectively by *Casper*, constituting *Casper's dictum*. Thus, a dead body putrefies within one week when left open to air, while it requires two weeks when drowned in water and eight weeks when it is buried under the soil. Similarly *'Dictum of Rule of thumb'* describes that dead bodies decay *twice* as fast in air as in water. Both these *sayings* of the past though are considered *grossly inaccurate* at present; they certainly lay emphasis on the *slower* rate of decomposition of immersed or buried corpses.

Putrefaction in Water

Putrefaction in water is slower than on ground, because of lower ambient temperature, non-availability of air and protection from insect and small animal predators. The rate is still *slow* if it is running water. But, once the body is removed from water the putrefaction will take place *rapidly*.

- *First week*—little changes seen
- *Second week*—changes begins to appear in face, neck, etc. in bodies floating supine, as these parts are exposed to air, rendering identification of face difficult. However, the usual posture of freely floating body in water is *face down* as the head is relatively *dense* and does not develop early gas formation as in the abdomen and thorax. This *lower position* favours fluid gravitation and hence *more* marked decomposition, so the face is often *badly* putrefied in an immersed body making visual recognition difficult or impossible.
- *Two to six weeks*—distension of abdomen, skin peels off now
- *Six to eight weeks*—the whole body becomes discoloured and postmortem blisters or bullae of gas are formed
- *Third month*—putrefaction continues
- *Floating of dead body*—this occurs within 12-20 hours in *summer*, as a result of accumulation of gases in the body tissues, which makes it lighter. It will sink again on escape of gases.

Destruction by Animals[16,21,29]

(Postmortem damage by predators)

Dogs, fish, ants, etc. can eat the dead body, especially around natural orifices, such as nostrils, mouth, anus, vaginal orifice, etc. and eyes (Figs 13.26A and B). All such injuries by predators vary in appearance according to the size and shape of the teeth or jaws.

The animal eaten parts of the body may be *mistaken* for antemortem injuries are called as—*pseudoabrasion*. However, all predator induced postmortem injuries have certain features in common, which help to differentiate them from antemortem wounds. Firstly there is no bleeding apart from the minute quantity actually present in the vessels of the damaged part. Secondly, there will be no oedema and reddening of the edges (vital reaction).

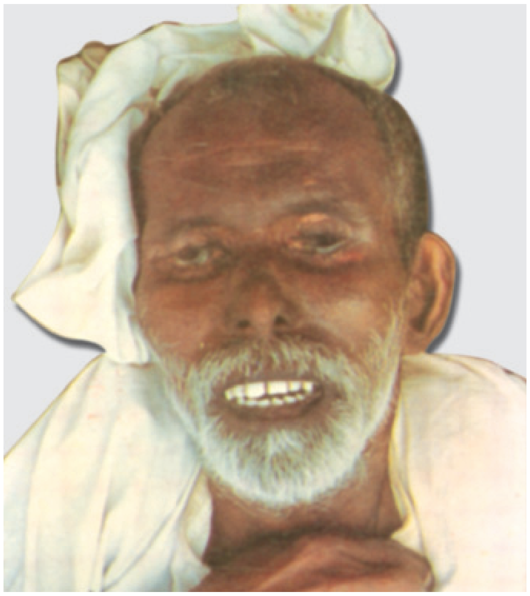


Fig. 13.26A: Pseudoabrasions postmortem injuries by ants—around the eyes (*Courtesy:* Dr SC Mestri, Prof and HoD Forensic Medicine, JSS Medical College, Mysore)

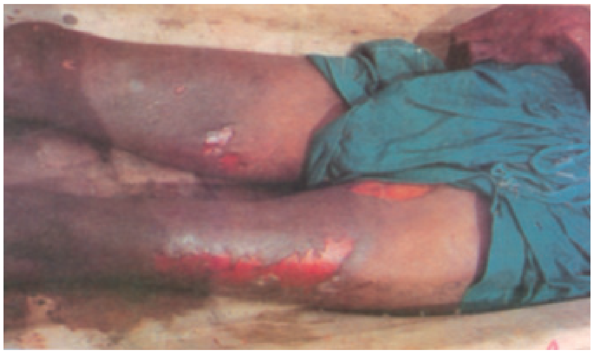


Fig. 13.26B: Pseudoabrasions postmortem injuries by ants—Over the thighs (*Courtesy:* Dr SC Mestri, Prof and HoD Forensic Medicine, JSS Medical College, Mysore)

SKELETONISATION

In forensic sciences, skeletonisation refers to the complete decomposition of the non-bony tissues of a corpse, leading to a bare skeleton. In a temperate climate, it usually requires three months to several years for a body to completely decompose into a skeleton, depending on factors such as temperature, presence of insects, and submergence in a substrate such as water. In the tropics, skeletonisation can occur in weeks, while in the Andes Mountains or Tundra, skeletonisation will never occur if subzero temperatures persist.

The rate of skeletonisation and the present condition of the corpse can be used to determine the time of death.

After skeletonisation has occurred, the human skeleton takes about twenty years under optimal conditions to be completely dissolved by acids in the soil leaving no trace of the organism. In neutral pH soil or sand, the skeleton will persist for at least several thousand years before it finally disintegrates. Infrequently, however, the skeleton can undergo fossilisation, leaving an impression of the bone that can persist for millions of years.[39]

The process of skeletonisation takes varied time under varied conditions. A body exposed to air, will get completely skeletonised from decomposition by 14-20 days, but the time limit may be less, if exposed to ants, flies, maggots, cockroaches, rats, vultures, dogs, jackals, etc in addition. Similarly, fishes, crabs and other aquatic animals may eat up soft tissues of a drowned body, which may get skeletonised within a period of few days or weeks.[20,27,28]

A buried body laid down in a coffin will take more than a year to get skeletonised depending upon the coffin material and place of burial, etc. An uncofined buried body in ordinary conditions in a tropical place will get skeletonised within six months maximum.[40] However, in case of mass grave and shallow burial, uncofined body will get skeletonised within a week or two.[20,27,28]

After skeletonisation, the bones usually decompose by 3 to 5 years or more; these will ultimately crumble to dust leaving no trace of it within 10-15 years. Decomposing bones lose weight and become fragile from loss of organic matter. The dead body will be finally reduced to a skeleton, when all the soft tissues and the viscera progressively disintegrate along, leaving with the ligaments and tendinous tissues attached to the bone. This depends on environment, as a corpse in the open air will suffer far more from rain, wind and especially animal predator, compared with one in a locked room or a coffin. A body dying outdoor in winter will '*survive*' longer than one dying in summer. In broad terms, a corpse in outdoors in a temperate climate in India is likely to be converted into a skeleton carrying tendon tags, within 6-18 months, and to a '*bare-bone*' skeleton within 3 years; there are of course, numerous exceptions depending mainly on the local environment.[16,17,20,39,40]

ESTIMATION OF TIME SINCE DEATH/ POSTMORTEM INTERVAL (PMI)

There are various factors considered to be useful in estimating the postmortem interval.[13,19,20,27-30,42-48]

- Postmortem cooling (refer page 143)
- Corneal changes in the eyes (refer page 144)
- Postmortem lividity (refer page 145)
- Rigor mortis (refer page 146)
- Putrefaction (refer page 149)
- Cadaveric entomology (refer page 153)
- Skeletonisation (refer page 156)
- Adipocere formation (refer page 158)
- Mummification (refer page 158)

Changes in Gastrointestinal and Urinary Tract

PMI can be assessed depending on the conditions or changes in these tracts.

Examples:

- Stomach contains undigested food particles- since digestion of food requires 2 to 3 hours normally, presence of undigested food particles in stomach suggests the deceased must have died after taking food.
- Fully distended urinary bladder—normally at night bladder gets fully distended, as people do not get up frequently to urinate once asleep. Thus, a distended bladder in a cadaver suggests death must have occurred at night.

Changes in Biological Fluids

Blood, CSF, vitreous humor, and other body fluids on biochemical analysis are found to contain certain chemicals in their own norms in life.[16,17,27-30,42-45] After death, metabolisms in the body tissues being ceased by all means, these norms cannot be maintained. Hence, variations inevitably show either an increase or decrease of concentration of these chemicals in all biological fluids progressively, and can be used as a marker in establishing PMI.

For example:

- *Vitreous humor potassium level*: It is reported to *increase* (rise) after death (liberated from retinal blood cells, by leaking from the retina through the now permeable cell membranes, into vitreous body). The vitreous electrolytes after death will show rise in Potassium, fall in Chlorine and Sodium levels. However, the postmortem interval determined by this method is having a variability of ±10 h, in the *first 24 h*; ±20 h in *first the 48 h* and ±30 h in the *first 72 h* after death, which has rendered it less reliable practically.[42-45]
- *CSF chloride level*: It is found to decrease and a concentration below 440 mg per cent indicates PMI to be less than 25 hours.[27-30]
- *Blood creatinine value*: It is found to increase after death. A concentration of 5 mg per cent indicates PMI to be less than 10 hours, while values of 11 mg per cent indicate PMI to be less than 28 hours.[16,17]

Growth of Hair and Nails

Normally in life, hair grows at the rate of 0.4 mm/day and the nails by 1 to 3 mm/week. Both hair and nails cease to grow after death. However, hair and nails present an *apparent growth* after death, due to drying up of the skin after death, which could help in establishing PMI or time since last shave.

Scene of Death Particulars

Valuable information can also be availed from scene of death in estimating PMI.

For examples, a wrist watch if found on the table besides a dead body may indicate a time somewhat nearer to the actual time of death, as it might have stopped, because of lack of winding which the deceased would have done if alive.

Carbon Dating

Radioactive carbon (C-14) is formed in atmosphere by the action of cosmic radiation and enters in all living beings through various channels, it gradually diminish after death. It is reported that weakening of C-14 radioactivity takes more than 5000 years to reach half its initial activity.[27-30,40,46-48]

A simple carbon compound, such as CO_2 or acetylene, or even carbon itself is prepared from the bones, and radioactivity is estimated. This means dating of the skeleton can be done even after burials for several years by this method, giving clues on PMI.

CONDITIONS THAT REPLACE PUTREFACTION

The phenomena of putrefaction is *replaced* occasionally by any one of the following three namely:

- Adipocere formation
- Mummification
- Maceration.

At times two or all the three may also be noticed in a same case. However for a better understanding each one is discussed independently in detail.[11-17,20-21,26-30,48] These can also help in determining Postmortem Interval (PMI).

ADIPOCERE FORMATION

Definition

Adipocere formation is a modified process of putrefaction, wherein the fatty tissues of the body change into a substance known as adipocere, by hydrolysis and hydrogenation (Fig. 13.27).

Note: Saponification is no more considered as synonymous terminology for adipocere formation as in the past as during adipocere formation soaps are formed only as a minor by-product. More over adipocere formation is an enzymatic process and not biochemical change.

Mechanism

In a dead body on exposure to moisture, e.g. when buried in damp moist soil/immersed in water, body fat undergoes certain changes, instead of putrefaction. The schematic representation of events of changes occurring under this is shown in Figure 13.28. These fatty acids being acidic inhibit putrefactive bacteria:

Factors Influencing

- Cold weather-retards
- Hot weather-accelerates.

Properties of Adipocere

- Odour—it has an ammoniacal odor initially, which gets later on replaced with a distinct rancid/sweetish odor.
- Fresh adipocere on touch—is soft, moist, greasy and waxy. It becomes dry, brittle, chalky and friable in months/years after time of formation.
- Colour—varies from dead white to grayish white or brown.
- Inflammability—it is inflammable and burns with yellow flame.
- Solubility—it is insoluble in water, but dissolves in alcohol and ether.
- Distribution—It is generally distributed in all sites wherever fatty tissue is present, such as subcutaneous fat (buccal pad of fat/cheeks, breasts in women, anterior abdominal wall, buttocks, etc and internal organs rich in fat like heart, etc)
- Chemically—adipocere contains palmitic, oleic and stearic fatty acids together with glycerol.
- Adipocere formation is often mixed with other forms of decomposition, i.e. one end of the body may be putrefied/skeletonized, while the other parts may be mummified/adipocere.

Time Required

Time required for adipocere formation: in 3 weeks time in summer temperate zones. However in India, it is reported to have formed in 3 days-3 months.

Medicolegal Importance

Body appearances, features (facial) are retained (although distorted compared to the immediate postmortem shape) —help in establishing identity.

- All injuries are well preserved—can assess cause of death.
- Can help in establishing time since death and place of death.

MUMMIFICATION

Definition

Mummification is a modified process of putrefaction, wherein the water content in the dead body gets evaporated making it dehydrated or desiccated and shriveled up, the natural appearances and features of the body are retained (Fig. 13.29).

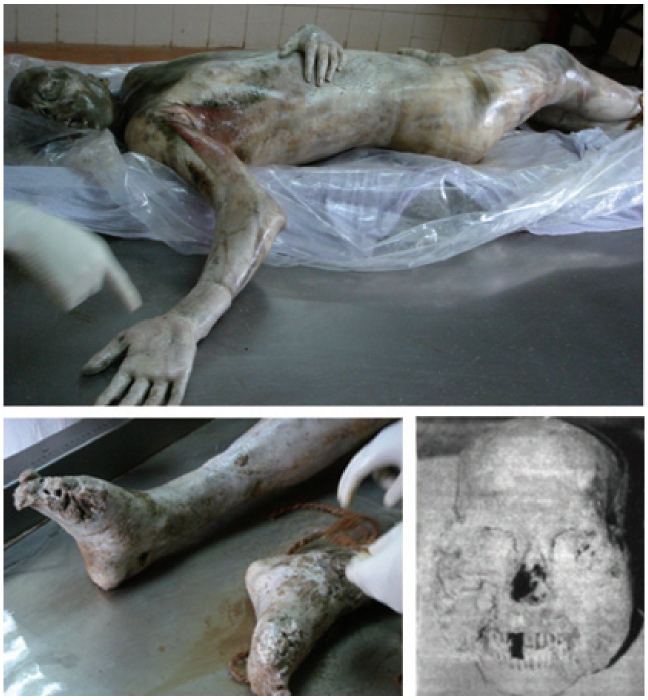


Fig. 13.27: Adipocere Formation. Note the moist greasy, waxy changes in the body (*Lower two figures–Courtesy:* SC Mestri, Prof & Head, JSS Medical College, Mysore)

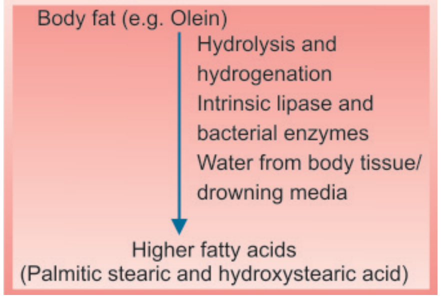


Fig. 13.28: Mechanism of adipocere formation

Factors Influencing

Body in dry place with warm dry air circulation, e.g. body buried in sandy soil, the soft parts become desiccated and putrefaction is inhibited.

Appearance

Whole body gets converted into a hard, dry, leathery mass and the features will remain as they are.

Time required—several weeks.

Medicolegal significance – Like the other modes of decomposition, this can be partial and can co-exist with them in different areas of the same body.

MACERATION

Maceration is due to autolysis in an aseptic environment. When fetus dies in uterus naturally, it will be in a sterile medium in the amniotic fluid. So, no organism can enter, hence, no putrefaction can occur, until and unless the membranes are intact. The body (Fig. 13.30) becomes:

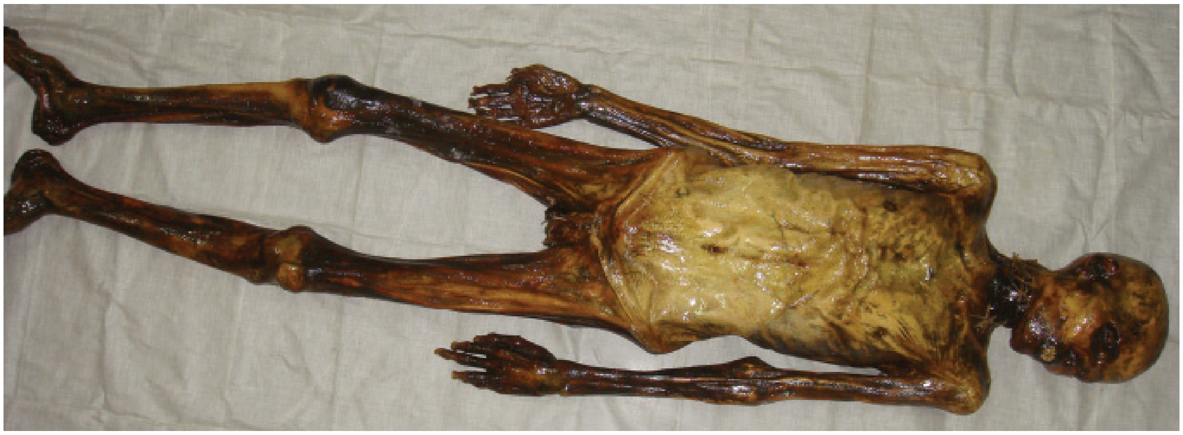


Fig. 13.29: Mummification. (*Courtesy:* Dr Kiran J, Prof. and Head, Department of Forensic Medicine, Devraj Urs Medical College, Kolara, Karnataka)

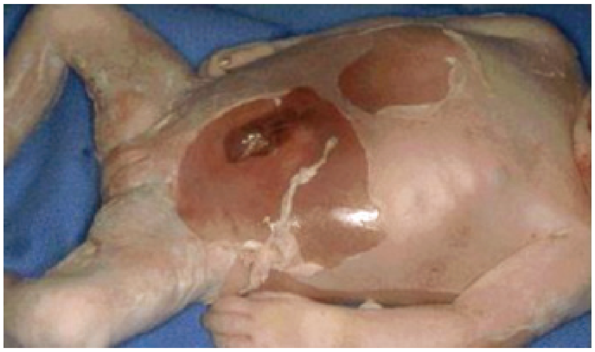


Fig. 13.30: Macerated fetus, the skin is peeling and the tissues are jelly like. The fact that the umbilical cord is tightly wound round the neck does not mean that it must have been the cause of stillbirth

- Discoloured – *brownish-pink* after exposure to air.
- Skin is peeled off and slimy.
- Arms flaccid and frail.

Time Required

48-72 hours after IUD.

Confirmation by Radiograph Picture

Radiograph shows over-riding of skull bones and ribs known as 'Spalding's sign'.

PROCESS OF PRESERVING THE DEAD BODY[20-21,41,49,50]

Three methods are approved and they are–*embalming*, *freezing* and *taxidermy*.

Embalming

The contents of the gastrointestinal tract, bladder and blood vessels are effectively drained out with the help of trocar and suction apparatus passed through a puncture wound in abdomen, and then embalming fluid consisting mainly of 40 per cent formaldehyde solution is injected into the femoral and carotid arteries, chest and abdominal cavities.

The process renders the proteins to get coagulated, tissues fixed, organs bleached and hardened, blood coagulated and converted into pinkish brown mass. If proper embalming is done soon after death, the decomposition can be withheld for a long time; even may be for months. Even when it is done in a body where the decomposition has already started, it can prevent it's further progress and the body can thus be kept preserved for a long-time after death. In an embalmed body, it is difficult to

Table 13.9: Putrefactive changes in relation to time since death[11,20]

Changes observed	*Time since death**
Greenish discolouration of	
• Right iliac fossa	12-24 hours
• Whole body	48 hours
Marbling changes	
• Just commenced	24 hours
• Becomes prominent	36-48 hours
Postmortem blister formation	18-48 hours
Foamy liver formation	18-48 hours
Loosening of scalp hairs, nails and teeth	>48 hours
Bloating of facial features	36-48 hours
Putrefactive blister formation	36-48 hours
Postmortem slipping of skin (hand, feet, etc)	48-72 hours
Changes in the external genitalia	48-72 hours
Postmortem delivery in gravid uterus	48-72 hours
Bursting open of the abdomen	48-72 hours

* *In India, in summer*

detect alcohol and some other substances including alkaloids as the process destroys them.

Freezing

If a body is kept in deep freeze or amidst thick layers of ice, it can remain in good preservation for a long time. Bodies left frozen in ice caves in arctic circles can remain preserved indefinitely.

Taxidermy

Taxidermists prepare dead bodies for the purpose of anatomy dissections by injecting solutions of *arsenic sulphide or oxide, lead sulphide, potassium carbonate with or without formalin* or at times formalin alone into the femoral and other big arteries.[41]

The aforesaid processes can be adopted for preservation of the dead body, especially when it is to be taken from one place to another for burial or to avoid putrefaction before cremation or burial, which might take some time.

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Chapter 14

Postmortem Examination

Postmortem examination is also known as, *autopsy* or *necropsy*. The word *autopsy* means *self-examination.* In the terminology *postmortem,* word *post* means *after* and *mortem* means *death,* thus *postmortem examination* means *examination after death*. Similarly, *necros* of *necropsy* means *death* and *opis* meaning *view,* thus, the word *necropsy* means *viewing after death.*

DEFINITION

Postmortem examination or autopsy is a scientific and systemic study of a dead body.[1-3]

CLASSIFICATION

Autopsies are of three types[4-17]: (i) clinical or pathological autopsy, (ii) medicolegal autopsy, and (iii) anatomical autopsy. Each of these is discussed below in brief:

Clinical/Pathological/Academic Autopsy

To determine the disease causing death. A Pathologist performs it with consent of the relative of the deceased.

Medicolegal Autopsy

To solve mysterious unnatural death. A forensic pathologist or medicolegal expert preferably does it.[1,7] However, in India, due to lack of adequate qualified experts, all registered medical practitioners can also perform this with an authorisation by State.

Anatomical Autopsy

To learn the normal structure of the human body by medical students. It is usually done on an unclaimed dead body, handed over to anatomy department by municipal or such other governmental authority. It is also performed on the voluntarily donated dead body. An anatomist performs this.

CONSENT FOR AUTOPSY

Clinical autopsy: Obtain consent from the relatives.

Medicolegal autopsy: No consent is required but legal permission or authorisation *(requisition from police)* is a must.

Anatomical autopsy: Here permission must be obtained from government authority and the dead body is mostly an unclaimed. In a voluntarily donated dead body consent from the legal heirs is essential prior to the dissection.

MEDICOLEGAL AUTOPSY

A medicolegal autopsy is thus a special type of autopsy or postmortem examination, ordered by the government/legal authorities in all unnatural deaths, such as *homicide, suicide, accident, etc.* It is a challenging problem in India for reasons such as:[7-9,11,12,15-17]

- Dearth of properly trained experts in the medical specialty.
- Weather condition in India sets up the putrefactive changes early, rendering all the trace evidence lost in a dead body at times.
- Scope and facilities for such investigation and examination are not satisfactory.
- Medicolegal autopsy can be studied in detail under following heads.

OBJECTIVES

Objectives of medicolegal autopsy are definite and they are basically to establish:[7,16,17]

- Identity of the deceased
- Time since death
- Cause of death
- Live birth, period of viability, etc.

Identity of the deceased: This is simple and easy if the person dead is well known. However, the problem is difficult when the dead body recovered is not known to anyone. Racial characteristics, age, sex, stature, belongings on the dead body, birthmarks, moles, facial features, complexion, tattoos, scars, deformities, finger-prints, poroscopy, footprints, occupational stigmatas, anthropometry, teeth etc might help in establishing identity of it unknown *(refer Chapter Forensic Identity).*

Time since death: Estimation of probable time since death (postmortem interval) is done by the various changes occurring in a dead body after death such as postmortem cooling, postmortem lividity (hypostasis), rigor mortis, putrefactive changes (contents of the stomach/bowels/urinary bladder), biochemical changes in the body fluids such as CSF, vitreous humos, etc. are of great help *(refer Chapter Thanatology).*

Cause of death (Fig. 14.1): Establishing the case of death is an interpretive two-step intellectual process, deriving and depending upon sound evaluation of morphologic evidence of injury or of injury and disease, results of toxicological, biochemical, microbiological and histopathological studies. First step involves recognising structural organic changes (morbid anatomic features) or chemical abnormalities responsible for cessation of vital functions. Second step in establishing cause of death requires an understanding and exposition of mechanism by which the anatomic and other deviations from normal actually caused death. The mechanism of death is the physiologic derangement or biochemical disturbance incompatible with life which is initiated by cause of death, e.g. haemorrhegic/hypovolaemic shock, metabolic disturbances, cardiac tamponade, sepsis with piofound bacterial toxaemia, etc. The

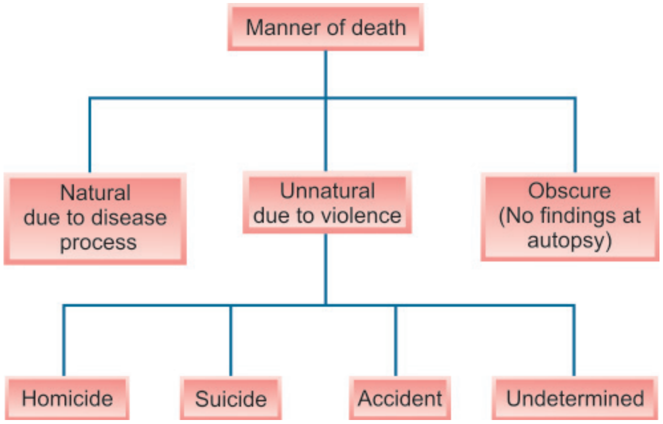

Manner of death
Natural due to disease process
Unnatural due to violence
Obscure (No findings at autopsy)
Homicide
Suicide
Accident
Undetermined

Fig. 14.1: Illustrates the manner of death

fashion in which cause of death arose refers to as manner of death (See Fig. 14.1). If death resulted solely from a disease, the manner of death is natural. If death is solely by injury or is hastened by injury in a person rendered vulnerable by natural disease, manner of death becomes "violent" or "unnatural". The violence could be homicidal, suicidal, accidental or undetermined. Investigation by the police outside the autopsy room is essential in deciding this accurately.

Live birth, period of viability *(refer Chapter Fetal Autopsy).*

Legal Formalities

Legal formalities in taking up a case for medicolegal autopsy are:

- Authorisation order
- Identification of the deceased
- Facts about the case
- Place of performing autopsy
- Qualifications.

AUTHORISATION ORDER

Authorisation order is usually in the form of requisition letter, which must be received by the doctor prior to autopsy and it depends on type of the case (Table 14.1). However, never perform an autopsy without an authorisation order.

Identification of the Deceased

A dead body is better identified prior to autopsy. If the deceased is a known person it is mandatory to get it identified. If the deceased is unknown, efforts are made in noticing the factors which could help in establishing the identity later.

Known body: Confirm identity by the police officer or constable or the relatives or legel heirs. Always take the signature of the person identifying. If police constable is identifying the deceased, note down his or her *PC No, name and name of the police station he or she belongs to, etc. on* the postmortem report form itself.

Unknown body: Ask police for taking the photograph, fingerprint, details on the particulars of the dress worn with the tailor's label if any, etc. Doctors should also make note of all available factors (external/internal) of establishing identity *(refer chapter Forensic Identity)*.

Facts about the Case

For better autopsy results always try to study all available facts about the case prior to autopsy and it includes:

- Inquest report—issued by *police*
- Hospital records (if any) such as wound certificate, case file/ sheet, etc.

Note: Confirm HIV and HBV status of the deceased whenever facilities available, as to take proper self-care and care of the other mortuary staff.

Place of Performing Autopsy

- Preferably done in an equipped mortuary
- Autopsy is also done at site of recovery of the cadaver in cases of *exhumation* and *putrefied body*.

Qualifications

Minimum qualifications render a doctor qualified for this work is that he or she should have MBBS degree and that he or she must be a registered medical practitioner.

Specialist qualifications vary and include any one of the following qualification[7]:

- MD (forensic medicine)
- DNB—forensic medicine (diplomat of national board—forensic medicine)
- DFM (diploma in forensic medicine)
- DMJ (diploma in medical jurisprudence) from UK.

Other specialist qualifications

- MD (pathology)
- MS (anatomy/surgery)
- Any other specialists may also be allowed to do the autopsy in India on account of shortage of subject specialists. However since 1985 MCI insists to recruite only MD (forensic medicine) qualified only to be appointed in forensic medicine departments of a medical college allowing all those who are already in profession to continue.

Medicolegal Autopsy Guidelines

- Avoid unnecessary delay in performing autopsy
- Do not allow unauthorised person into the mortuary. If allowed record a statement from him or her giving reasons for his or her presence and signature for being present during the autopsy. The investigating officer (IO) may be allowed, if desires.

Table 14.1: Type of cases and authorising officers

Type of case	*Authorising officer*
1. Routine cases (e.g. accidents, suicide, etc.)	Police sub-inspector, Station house officer
2. Homicidal death	Circle inspector of police
3. Exhumation	Executive magistrate of class I
4. Death in Prison	Executive magistrate of class I
5. Death in Police custody	Executive magistrate of class I
6. Death in Police firing	Executive magistrate of class I

- An autopsy is better done in the **daylight (dawn to dusk)**. However, medicolegal autopsy may also be conducted even after dusk or in the night, with proper artificial light having day light (incandescent light) effect.
- Prepare the PM notes during autopsy or arrange to tape record the dictation.
- Prepare the PM report immediately and sign it duly.
- Handover this report and viscera (if preserved for chemical analysis) only to an authorised police officer/constable maintaining the 'Chain of custody'.

Autopsy Procedure

Procedures vary and depend on the skill and experience of forensic expert. A checklist for postmortem examination can be given under the following headings in general.[1,7,10,11,16-18]

External Examination

It is the most important part of the medicolegal autopsy procedure as most of the information gathered at this stage can always help a lot in solving the most mysterious deaths. This includes examination of:

- Clothes
- Stains of mud, blood, urine, stool, etc.
- Identity
- Body orifices
- Finger/toe-nails
- Injuries/surgical intervention
- Rigor mortis
- Postmortem staining
- Decomposition/other changes.

Internal Examination

This includes dissection and examination of the three major body cavities and their contents, namely:

- Skull/cranial cavity
- Thoracic cavity
- Abdominal cavity.

No autopsy is completed until all parts of the body are dissected and examined in detail.

According to Ludwig[7] principal autopsy techniques are deseribed and they are:

1. Technique of R Virchow: organs are removed one by one. Here the cranial cavity is opened first, then spinal cord, followed by thoracic, cervical and abdominal organs in that order.
2. Technique of C Rokitansky: This is characterised by 'in situ' dissection, in part, combined with removal of organ blocks.
3. Technique of A Ghon: Thoracic and cervical organs, abdominal organs, and the urogenital systems are removed or organ block ('en bloc' removal).
4. Technique of M Letulle: Cervical, thoracic, abdominal and pelvic ogans are removed as one organ mass ('en masse' removal) and subsequently dissected into organ blocks.

Note: While undertaking a medicolegal autopsy following points may also be remembered:

- Depending on type of a case, any of the body cavity can be opened first. Table 14.2 provides an idea as to open which cavity first, depending on type of a case.
- Spinal cord is routinely not opened.
- Arrange for histopathological examination, chemical analysis, etc. as needed, especially when cause of death is not clear.

Table 14.2: Type of case and choice of opening body cavity

Indication/type of case	*Cavity*
Head injuries	Cranium
Hanging, strangulation, throttling, etc.	Cranium
All other cases	Thorax and abdomen

Note: The autopsy surgeon may however adopt a technique to his convenience

DISSECTING CRANIAL CAVITY

Dissecting cranial cavity includes following steps:

- Scalp incision
- Removing the skull cap
- Opening the duramater
- Removing the brain
- Dissection of brain and its parts.

During each of the steps enumerated above, proper examination of each of the following is done:

- Scalp—any injuries
- Skull—any fractures
- Membranes—haemorrhages, pus, etc.
- Brain—pressure manifestations, injuries, congenital anomalies, abscess, tumours, etc.

Note: Keep a wooden block under the shoulder making the head rest firmly.

Scalp Incision

Incise the scalp in coronal plane, extending from one mastoid process to other. However, examine the scalp thoroughly for injuries or any other findings prior to the incision, as well as after incision on its inner aspect also after incision and reflection. Figures 14.2A to D illustrate the method of incising and reflection of the scalp.

Note: First separate hairs along the dotted line to avoid cutting of hairs.

Removing the Skull Cap

Comprise of following steps: Incise the temporalis muscle and cut it along its origin and reflect down on both sides. Next, saw the skull bone a little above superciliary ridges in front and occipital protuberance behind (Fig. 14.3A). Now, let both lines meet at an angle of 120° above mastoid process and then remove the skull cap, exposing dura mater (Figs 14.3B and C).

Opening Dura Mater

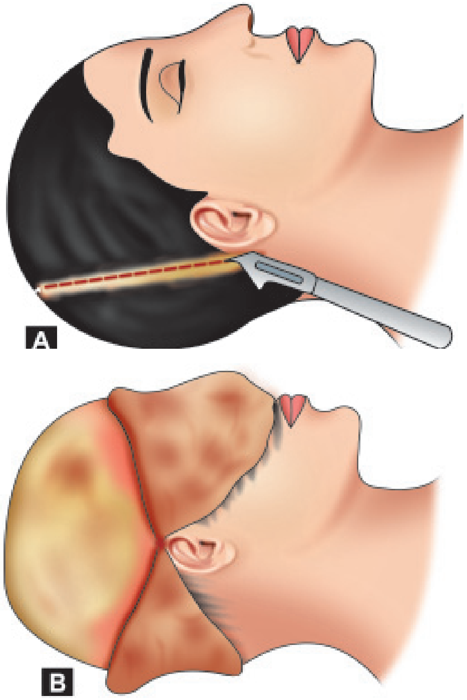
Using a sharp pair of scissors make a nick on either sides of midline anteriorly and extend it by cutting parallel to midline anteroposteriorly and then cut along the coronal plane on either side. The dura mater is then reflected into four flaps (Figs 14.4A and B, Figs 14.5A and B). With this examine the brain still intact and note down the observation.

Removing the Brain

Removal of the brain is essential procedure and it comprises of following steps (Figs 14.6A and B).

- Insert four fingers of left hand between frontal lobes and skull.

- Draw the lobes backwards gently and cut optic nerve and then other nerves and vessels with right hand as they emerge out from the skull.
- Cut the tentorium cerebelli along superior border of petrous bone and along its attachments in posterior cranial fossa.
- Cut spinal cord, first cervical nerves and vertebral arteries as low as possible through foramen magnum.
- Support the brain in left hand and remove with the cerebellum.



Figs 14.2A and B: (A) Dotted lines showing-scalp incision, (B) Reflection of incised scalp anteriorly and posteriorly

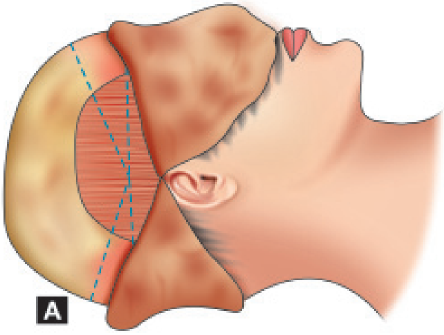


Fig. 14.3A: Dotted lines showing the lines of sawing the skull cap

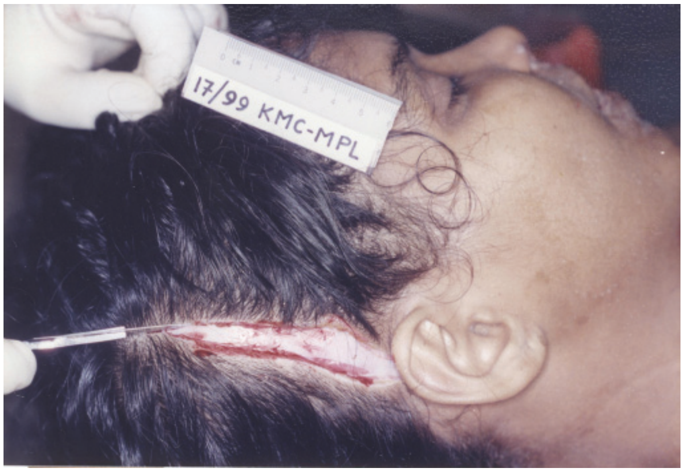


Fig. 14.2C: Incision of scalp along line of parting the hairs in coronal plane

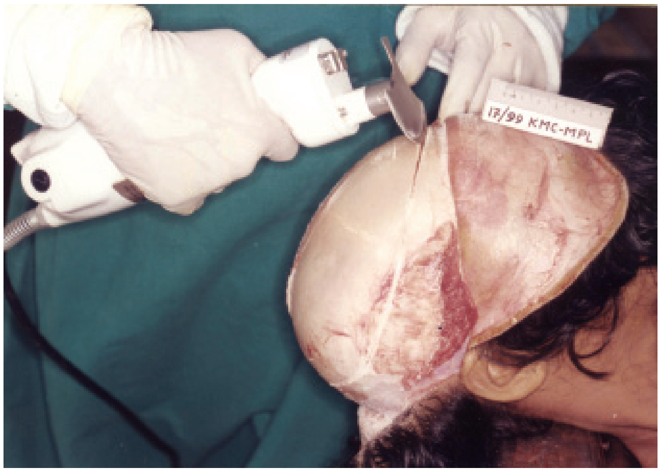


Fig. 14.3B: Sawing the skull by electric saw

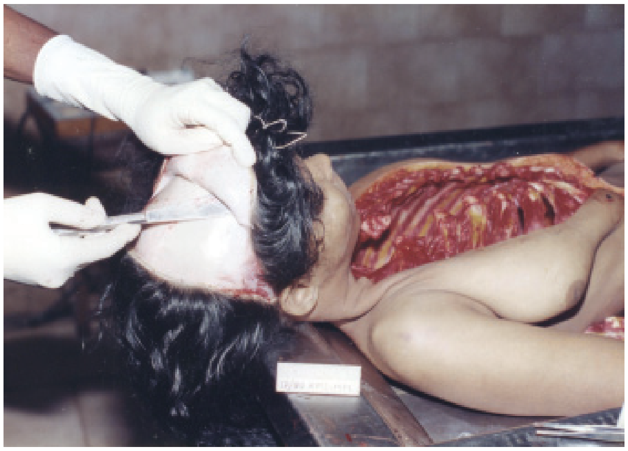


Fig. 14.2D: Reflection of scalp

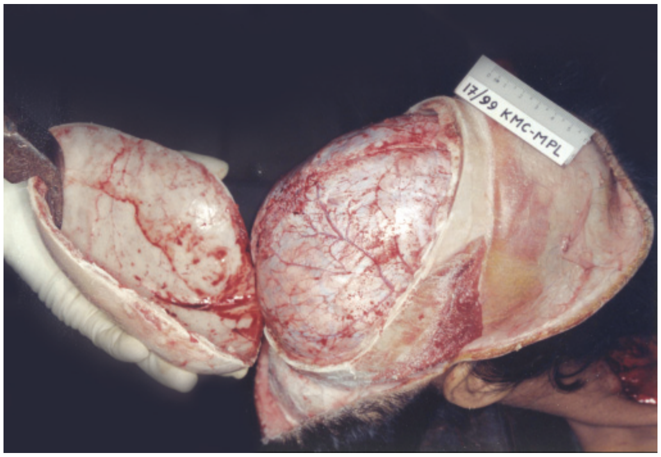
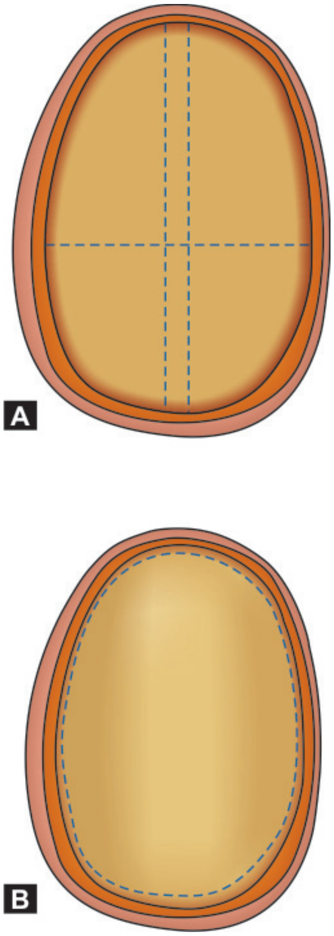


Fig. 14.3C: Cranial cavity opened, exposing dura mater



Figs 14.4A and B: Dotted lines showing — (A) Opening of the dura mater into four flaps, and (B) Opening of the dura mater in a single piece

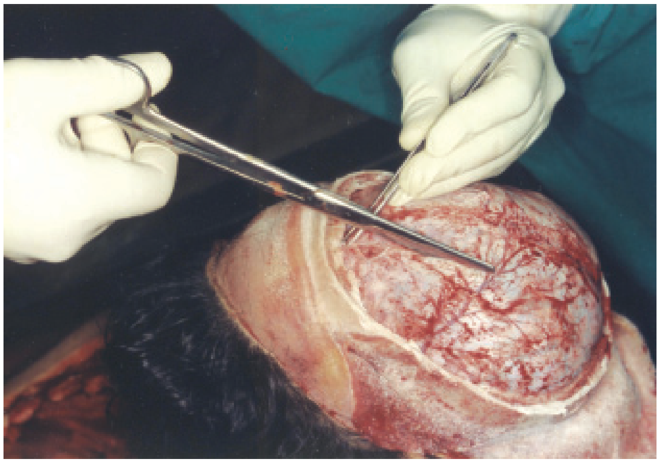


Fig. 14.5A: Opening the dura mater

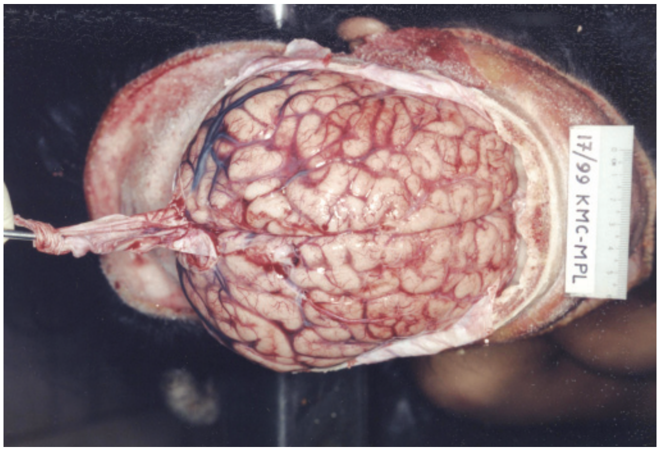


Fig. 14.5B: Brain exposed on reflecting the 4 flaps of dura

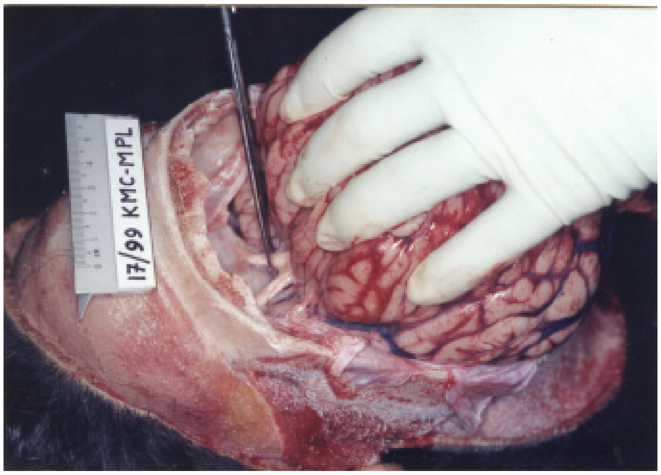


Fig. 14.6A: Cutting optic nerves

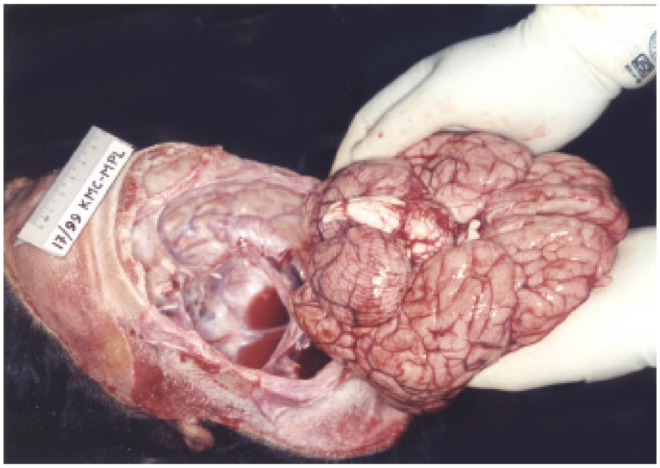


Fig. 14.6B: Removing the brain on cutting spinal cord deep in foramen magnum

Dissection of Brain

The brain is placed on a board with frontal pole away from dissector. Steps of dissecting brain are enumerated below:

- The cerebral hemispheres are separated first by left hand
- Using a brain knife, placing it in the longitudinal sulcus, cerebral hemispheres of brain are sectioned on either side, just above the level of corpus callosum, exposing basal ganglion, the lateral ventricles, the choroid plexus and interventricular foramen, which are then examined (Figs 14.7A and B)
- Next cut the fornices and corpus callosum and reflect it backwards. Examine thalamus and caudate nucleus.
- Third ventricle is now exposed; pass a probe through aqueduct of Sylvius (Fig. 14.8).

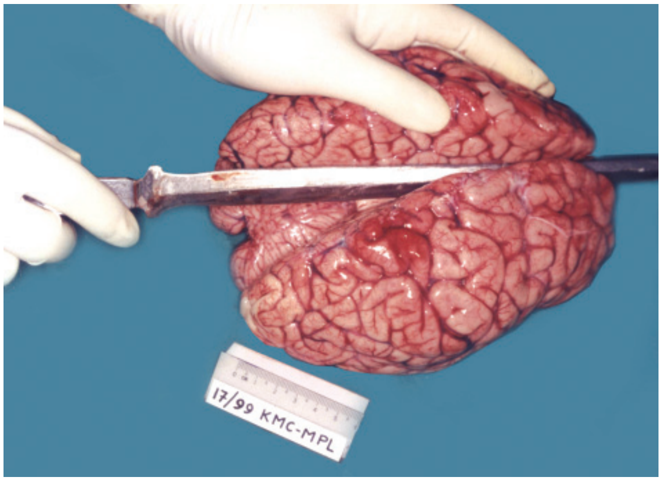


Fig. 14.7A: Sectioning the brain for exposing the lateral ventricle and basal ganglion

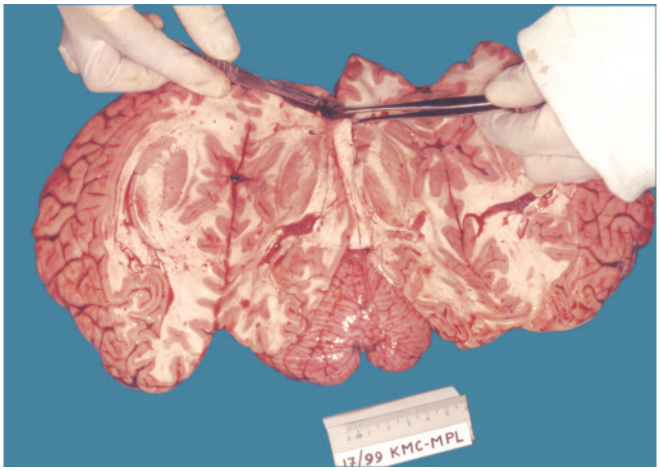


Fig. 14.7B: Cut section of brain exposing lateral ventricles and basal ganglion

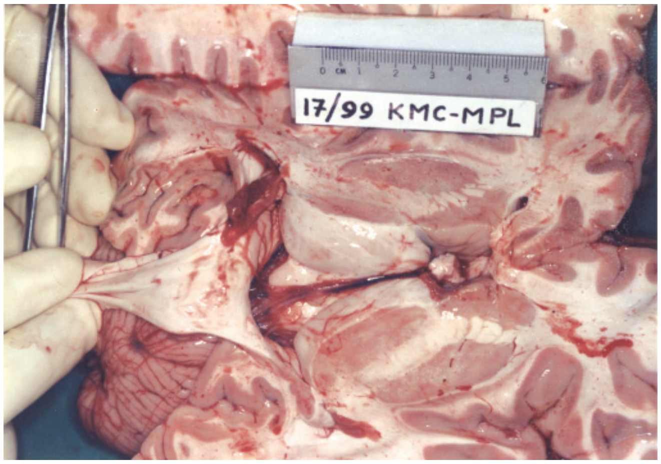


Fig. 14.8: Exposing third ventricle

- Expose the fourth ventricle now by cutting along vermis in midline by a scalpel.
- The internal and external capsule and basal ganglia are now exposed and examined.
- Remove cerebellum and brain stem now by cutting through cerebral peduncles.

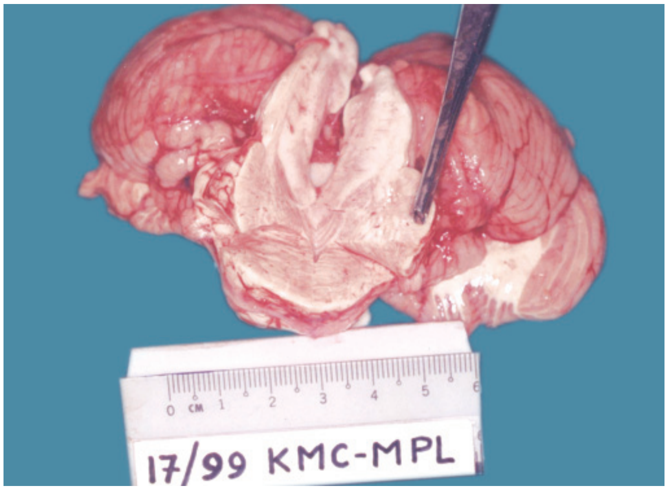


Fig. 14.9: Cut section of pons and brainstem

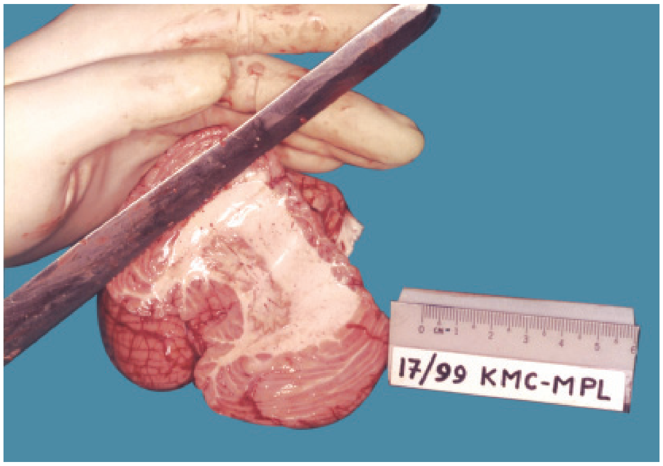


Fig. 14.10: Cut section of cerebellum exposing dentate nucleus

- Make sections through pons, medulla and remaining cord (Figs 14.9).
- Expose dentate nucleus by cutting cerebellar hemispheres (Fig. 14.10).

DISSECTING THORACIC AND ABDOMINAL CAVITIES

The procedure includes several steps and they are:

- Incisions
- Removing the abdominal and thoracic viscera *enmass (Letulle’’s method)*
- Closing the body
- Handing over the body
- Preserving/dispatch of viscera to FSL or to other laboratories.

Incisions

Several types of incisions are mentioned (Figs 14.11A and B):

- I-shaped incision
- Y-shaped incision
- Modified Y-shaped incision
- Elongated X-shaped incision

However, only three types of incision are in practice routinely and described initially, followed by uncommon ones.

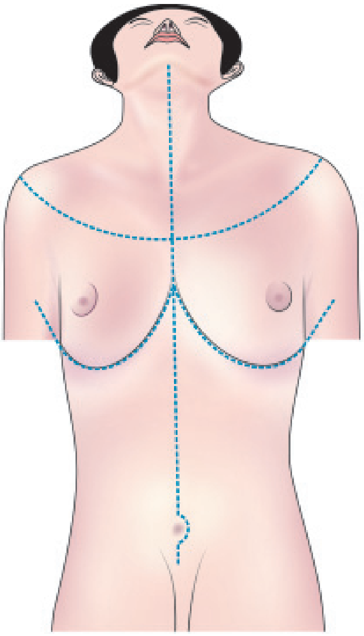


Fig. 14.11A: I-, Y- and modified Y-shaped incision

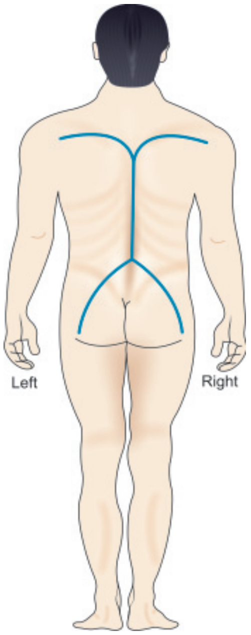


Fig. 14.11B: Elongated X-shaped incision of the back for subcutaneous dissection in custodial death cases

I-Shaped Incision

Extends from symphysis mentii to symphysis pubis (Fig. 14.12A), taking curve towards left around umbilicus.

Indication It is used routinely in practice.

Advantages It is simple and convenient.

Y-Shaped Incision

Commence at angle of mandible above on either side and the incision from both sides brought forwards, downwards to meet at the suprasternal notch and then run downwards as in "I" shape incision to symphysis pubis.

Indications This is preferred when a detailed study of neck structures required, e.g. asphyxial death due to neck compression.

Advantages It has a better exposure and allows study of neck structures.

Modified Y-Shaped Incision

Commences at anterior axillary fold on the chest on either side, runs downwards and anteriorly beneath the breasts as to meet at xiphisternum in the midline and then run downwards as in *I-shaped incision* to symphysis pubis (Fig. 14.12B).

Indication Preferred in females—

Advantages: It prevents cutting of the chest skin in midline and also allows detailed study of neck structures.

Elongated X-shaped Incision

This is a aspecial incision used to dissect out subcutaneous structures in the back to identify and evaluate the extent of blunt injuries, which are usually missed where superficial imprints are faint, particularly when present on skin not overlying bone (See Fig. 14.11B). This is helpful in reconstruction of events prior to death and should be practiced in causes of custodial death.[7]

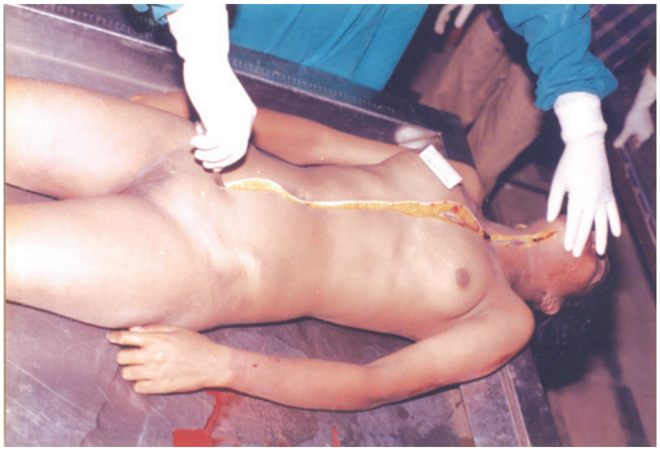


Fig. 14.12A: I-shaped incision

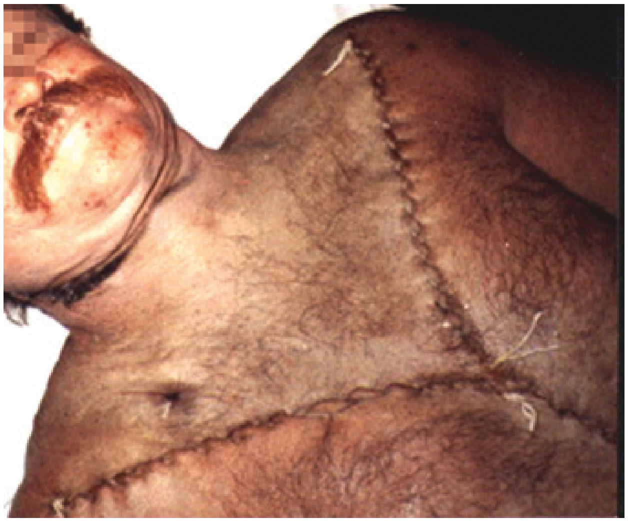


Fig. 14.12B: Sutured 'modified Y-shaped' incision

Removing the Abdominal and Thoracic Viscera

Opening Abdominal Cavity

1. The rectus abdominis muscle is incised first and then a small nick is made into the peritoneum
2. Next, introduce the index and middle fingers of left hand into the peritoneal cavity, keeping adequate gap inbetween the tip of the fingers pointing towards head-end or foot-end as needed; lift the abdominal wall and extend the cut upwards up to xiphisternum and then downwards up to symphysis pubis and open the abdominal cavity
3. As the abdominal cavity is opened look for following:

- Thickness of fat in abdominal wall, in the omentum and around kidneys
- Presence of fluid, pus or blood in the peritoneal cavity
- Evidence of perforation, obstruction, twisting, gangrene, etc. of gastrointestinal tract
- Mobilize the large intestine by cutting along peritoneal attachments
- Milk the contents of sigmoid colon and rectum proximally and cut it between two ligatures.

Opening the Thoracic Cavity (Figs 14.13A to E)

- The skin, subcutaneous and soft tissues in the neck and chest are then reflected sideward (Bruising of the thoracic wall, fracture of ribs, etc. should be noted if present).
- Now cut along the costochondral junction, and reflect the chest plate (Fig. 14.13A).
- Introduce the hands into pleural cavities and explore and look for blood/fluid collection (Fig. 14.13B).
- Disarticulate the sternoclavicular joints on either side, cut the cartilage of first rib and separate the chest plate and remove it (Fig. 14.13C).
- Examine the mediastinum for injuries and other findings and notice accordingly.

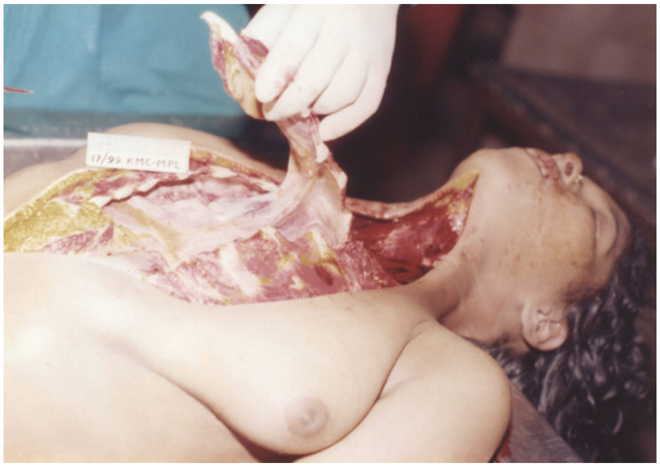


Fig. 14.13C: Removing the sternal plate

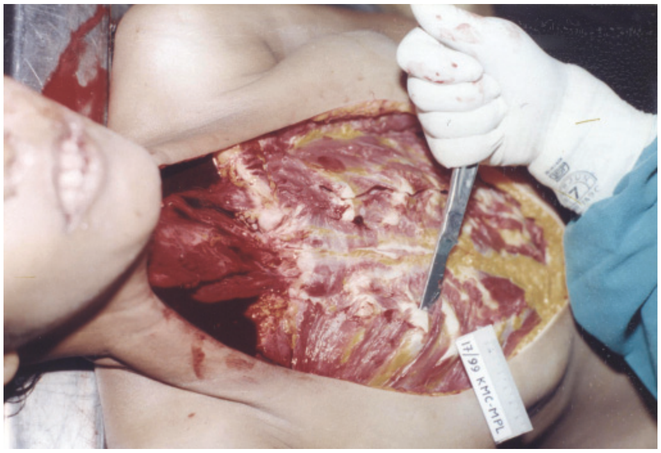


Fig. 14.13A : Opening thoracic cavity cutting along costochondral junctions

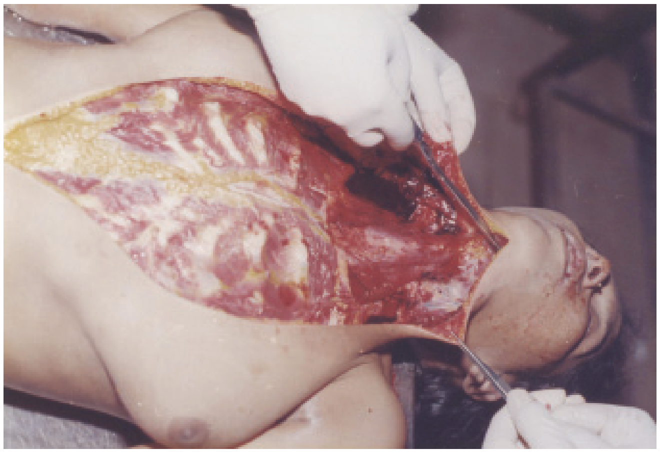


Fig. 14.13D: Cutting the floor and roof of the mouth

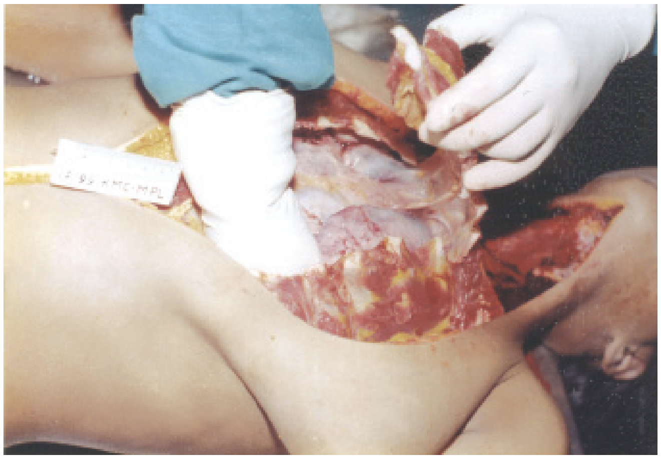


Fig. 14.13B: Exploring the pleural cavity by hand lifting the sternal plate

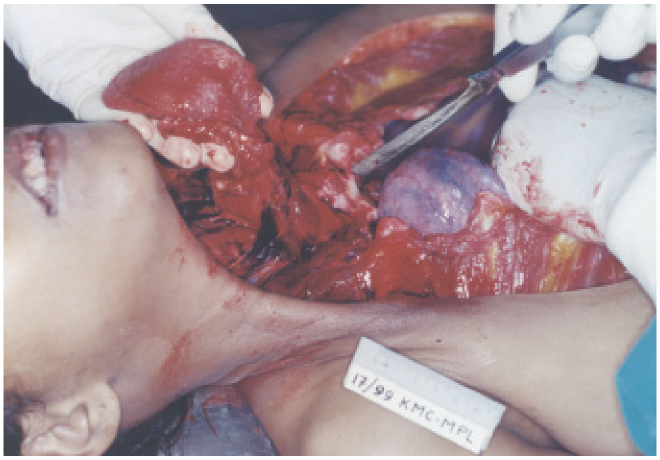


Fig. 14.13E: Detaching subclavicular ligament after releasing the tongue and neck structure

- Now cut the tissue of the floor of the mouth along inner sides of lower jaw with tip of a long pointed knife and release the attachments of tongue (Fig. 14.13D).
- Pull-down the tongue and cut behind the pharynx and larynx and release the attachments from vertebral column (Fig. 14.13E).
- Cut the costocervical fascia and large blood vessels at the root of the neck.
- Hold the tongue with a piece of cloth wound around it and trachea and by applying gentle traction, pull-down the thoracic organs caudally.
- Cut the crux and attachments of diaphragm muscle and pull further caudally.
- Pull-down further and remove all the organs *enmasse* from abdomen (Figs 14.14A to E).
- Keep the 'en masse' viscera thus removed on a dissection table in prone position and cut open the aorta from below upwards starting at little above its bifurcation (Fig. 14.14B), up to the arch of aorta. Similarly, now oesophagus and trachea are cut opened. Oesophagus is cut at the gastroesophageal junction between two ligatures (Figs 14.14C and D). Trachea is opened from above next, turning to either sides at its bifurcation.
- Each of the viscera is then removed and examined separately grossly and on sectioning, weight of each of the organ being measured before its dissection, except heart, which is weighed after the dissection.
- ***Stomach:*** The stomach is first removed by cutting between ligatures at its cardiac and duodenal ends (Figs 14.14C and 14.15A), and cut open along greater curvature (Fig. 14.15B) studying the contents and changes of the wall (Fig. 14.15C). Smell the contents for any abnormal odour.

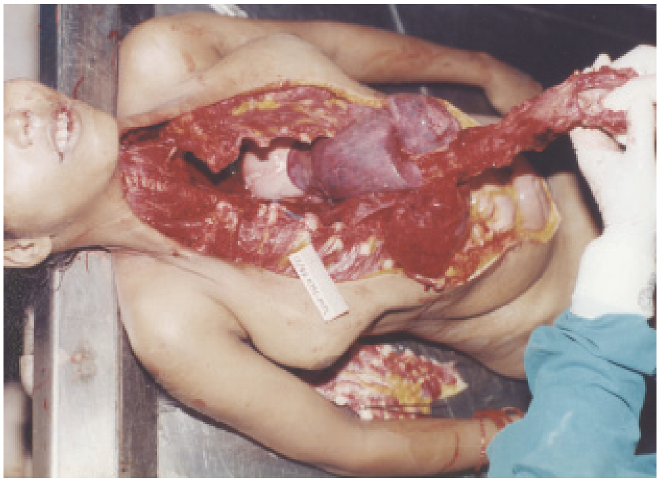


Fig. 14.14A: Thoracoabdominal viscera 'en masse' held lifted up in hand prior to its removal outside

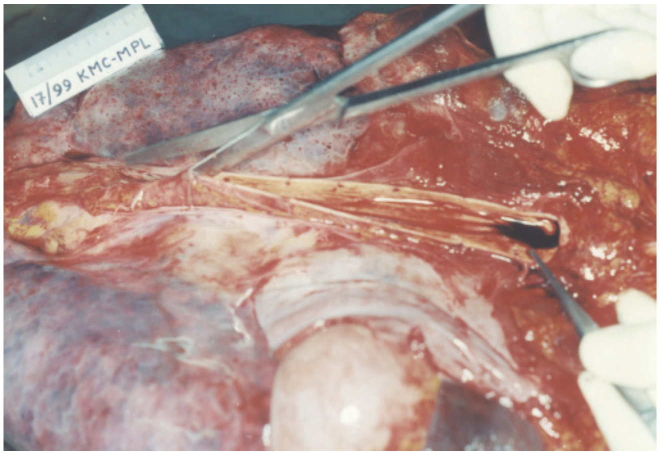


Fig. 14.14B: Opening the aorta

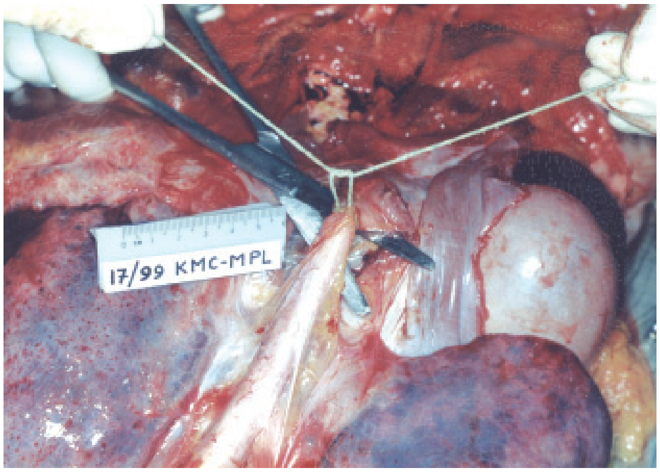


Fig. 14.14C: Ligating the oesophagus at the junction with stomach

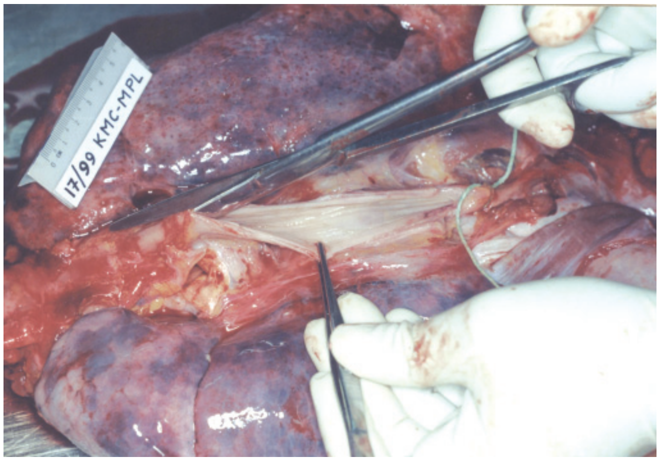


Fig. 14.14D: Cutting open oesophagus

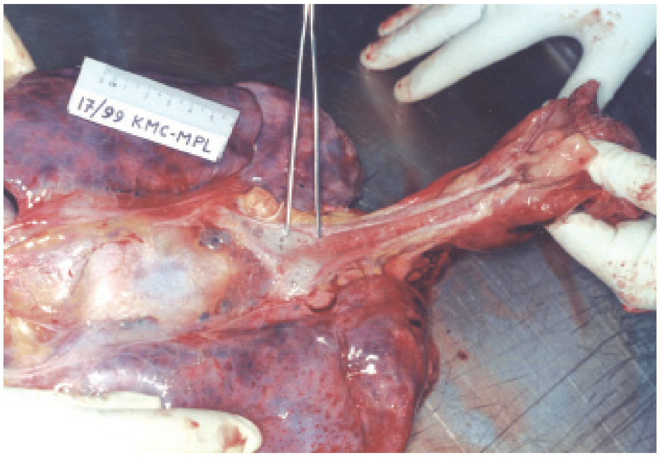


Fig. 14.14E: Cutting open trachea and bronchi (note frothy oozings)

- ***Heart, lung, liver, spleen and kidneys:*** Remove the heart, lungs, liver, spleen, kidneys, etc. and note down all changes grossly and on sectioning (Figs 14.15D to G). Heart is dissected in direction of blood flow is discussed separately. Lungs, liver, spleen and kidneys are sectioned from its outer/convex border to hilum. While lungs and kidneys are sectioned into equal halves (Figs 14.15D and E), liver and spleen are sliced (like a bread) into multiple sections (Figs 14.15F and G) allowing to visualise pathological/other changes due to disease/trauma.
- ***Uterus with appendages*** (Overies and follopian tubes): It must be removed 'en block' through abdominal incission, and dissected separately, noting the changes, especially the products of conception (foetus), presence of foreign body, tumour, or signs of delivery, criminal abortion, etc. prior to its sectioning.

Dissection of Heart

Holding it at its apex and cutting the aorta and pulmonary vessels as far away as possible from base, separate the heart. It is then dissected in the direction of blood flow (Figs 14.16A to G).

- To begin with pass a forceps through the openings of *superior and inferior vena cava* and by cutting between the arms of forceps, open the right atrium. Open also the auricular appendage and look for the thrombi, if any.
- Now introduce two fingers into the right ventricle through *tricuspid valve* and assess its size (normally three fingers can be introduced easily). Next, introduce a long pointed knife (heart knife) through it and pierce the apex and cut along the right lateral margin in one stroke (Fig. 14.16B). Note the thickness (normal 3 to 5 mm) and examine the valve cusps.

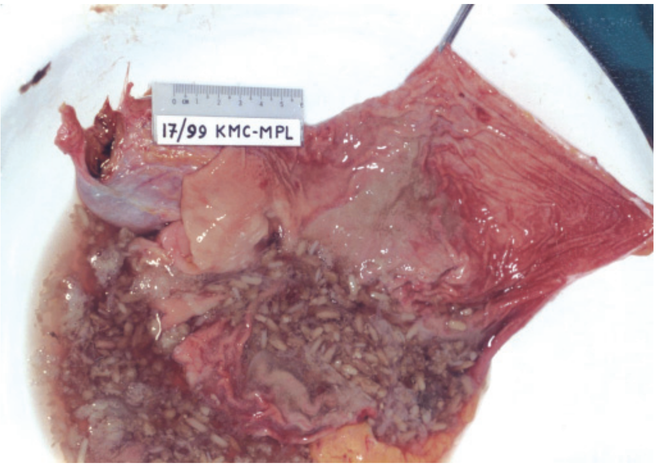


Fig. 14.15C: Note the stomach contents and stomach mucosal changes

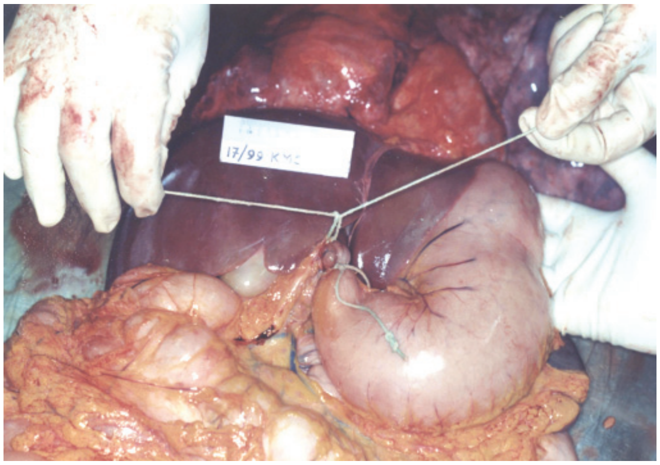


Fig. 14.15A: Apply two ligatures at duodenal end of stomach

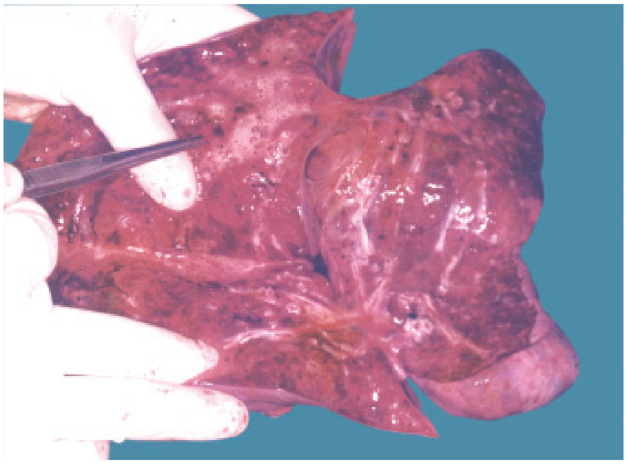


Fig. 14.15D: Sectioning the lung

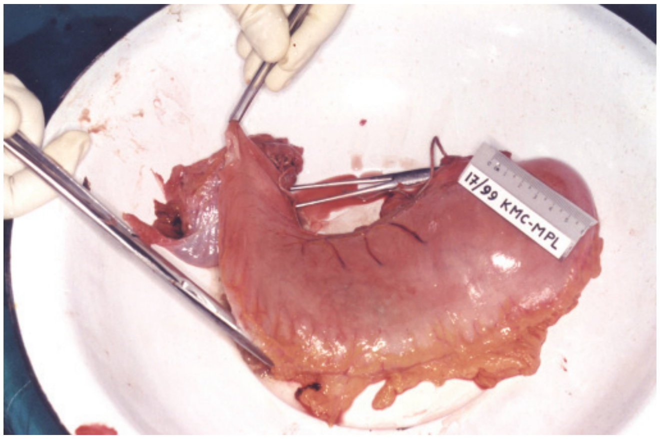


Fig. 14.15B: Dissecting the stomach along greater curvature

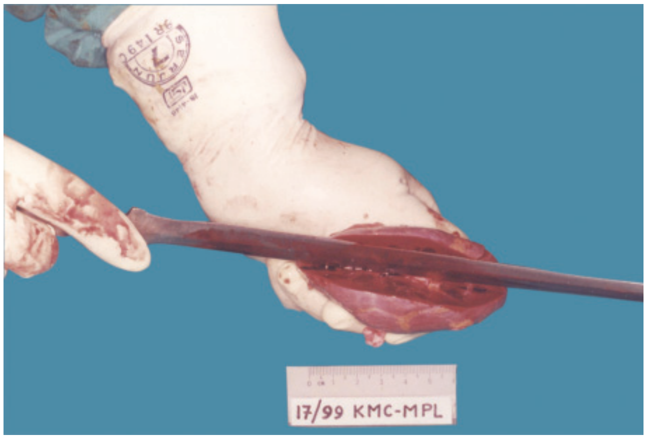


Fig. 14.15E: Sectioning the kidneys

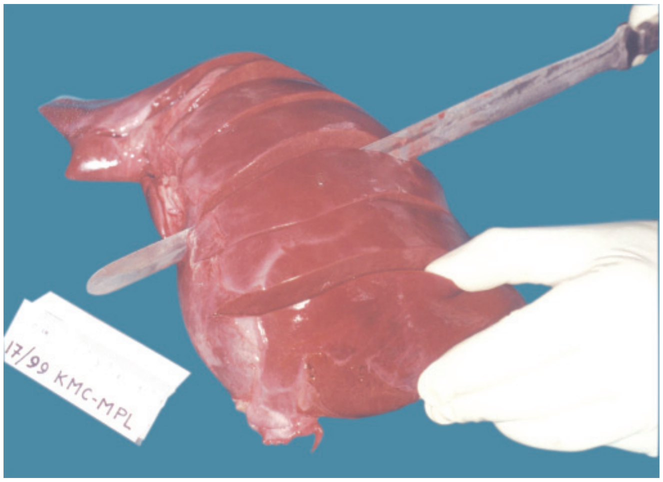


Fig. 14.15F: Sectioning the liver

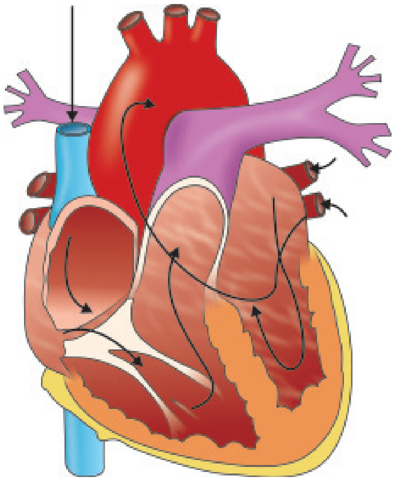


Fig. 14.16A: Dissection of heart along the blood flow—indicated by arrows

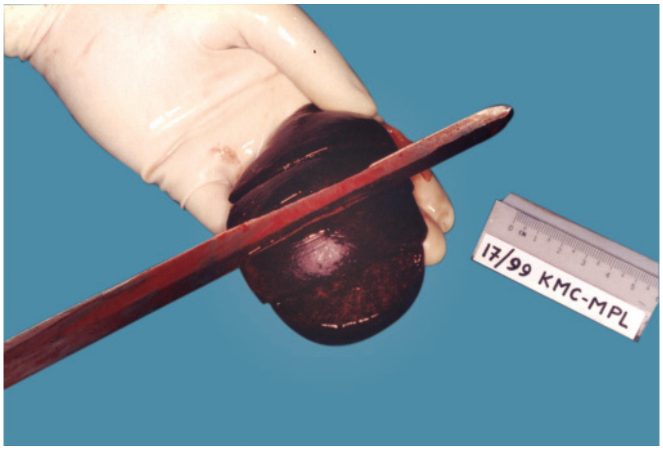


Fig. 14.15G: Sectioning the spleen

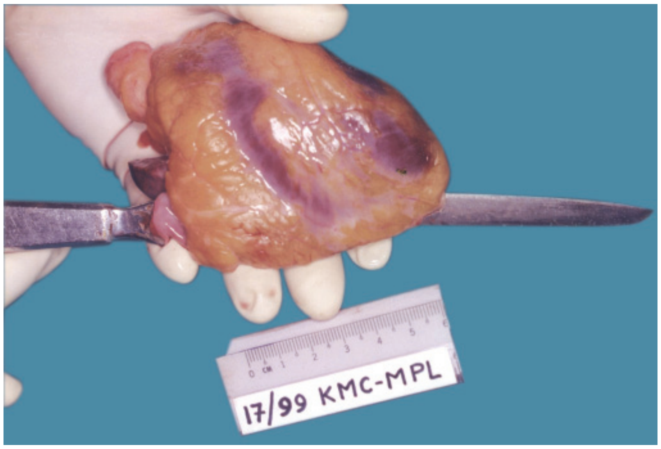


Fig. 14.16B: Dissection of the heart along the blood flow—opening the right chambers

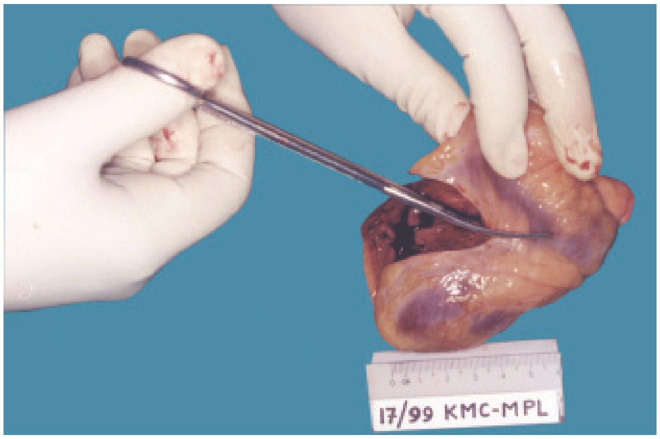


Fig. 14.16C: Dissection of the heart along the blood flow—opening pulmonary trunk through right ventricle

- Next from the apex cut open the pulmonary artery, along its long axis (Fig. 14.16C) and examine the valve, cusps and look for the presence of any embolus.
- Then, pass the scissors into *pulmonary veins, jointly cut them and enter into left atrium*. Cut the left auricular appendage and look for the thrombus, if any.
- Introduce fingers into *mitral valve* and examine it (normally two fingers allowed). Now introduce the tip of a sharp pointed knife into mitral valve and pierce through the apex and *cut along left lateral margin* in one stroke (Fig. 14.16D).
- Examine the wall, valves, papillary muscles, chordae tendinae, septum, etc.
- Next open the aorta from left ventricle (Figs 14.16E and F) and examine the aortic valve and coronary ostia. Pour thin stream of water into these leaflets of the valves from a tap (Fig. 14.16E) and confirm their intactness.
- Coronaries are dissected longitudinally or by ***serial sectioning*** along their course for their patency (Fig. 14.16G).

  Note: Heart is preferably weighed after its dissection.

Closing the Body

After complete dissection study, put all the viscera into the trunk and the body is closed properly suturing along the incisions using curved needle. Clean the body and dress it properly in an appealing way, while being handed over to police or deceased party.

Handing over the body to the police: Always handover the body to concerned police constable or officer who brought

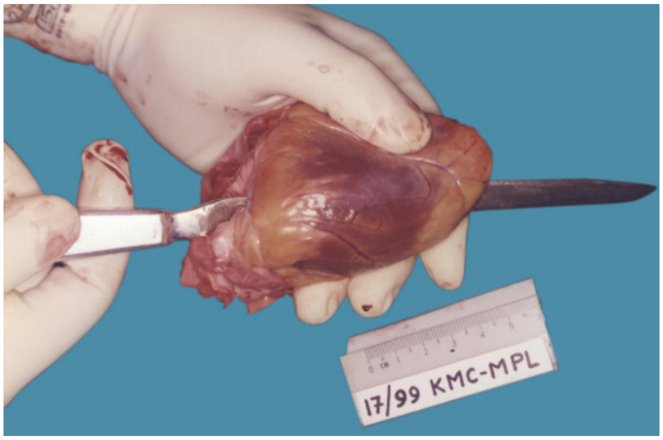


Fig. 14.16D: Dissection of the heart along the blood flow—opening the left chambers

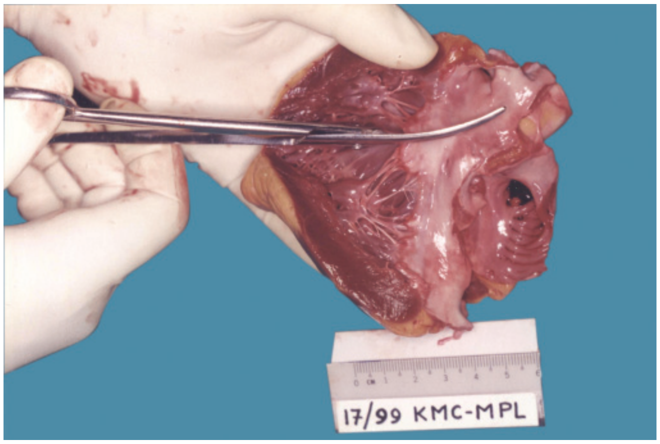


Fig. 14.16E: Dissection of the heart along the blood flow—opening the ascending aorta through left ventricle

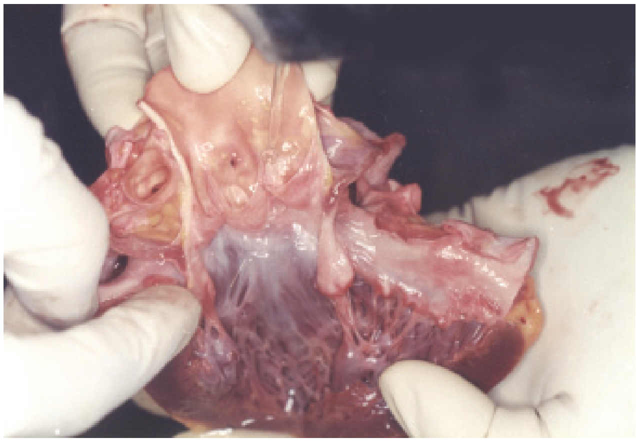


Fig. 14.16F: Dissection of the heart along the blood flow— aorta opened and testing the patency of aortic valves by making the tap water flow into the valve leaflets (Arrow)

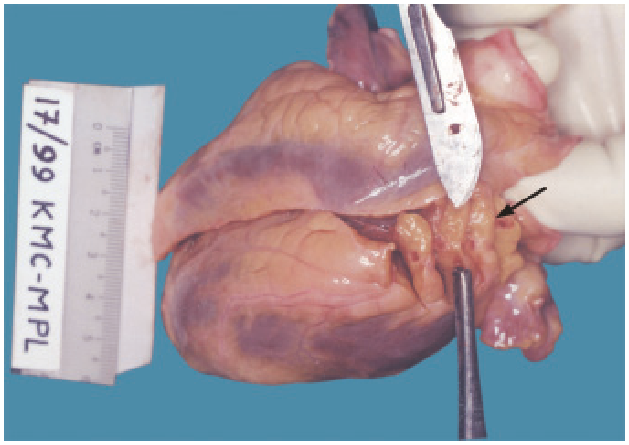


Fig. 14.16G: Dissection of the heart—serial sectioning of anterior descending branch of left coronary artery for confirming the patency of the lumen (Arrow)

it for autopsy. Take a written statement for receiving the autopsied body by the police mentioning the actual date, time, etc. accurately.

Examination of Spinal Cord

Routinely not examined. However, whenever needed it should be examined from the back. A midline incision is given in the back along entire length of neck and trunk, skin reflected out on either side laterally for about 2.5 cm. Vertebral column is then chiseled out along the medial margin of the transverse processes of the vertebrae. An electric saw may also be used in cutting. Entire length of spinal cord can be then taken out without causing much postmortem trauma.

Special Procedures for Specific Cases

These vary according to types of cases, and are enumerated below and discussed separately:

- Poisoning
- Mechanical asphyxia such as hanging, strangulation, drowning, etc.
- Burns
- Criminal abortion
- Road traffic accident
- Newborn/infant/foetus—infanticide case
- Firearm injuries
- Exhumation
- Examination of skeletal remains.

It may be remembered at this juncture that at times unless these special procedures are opted, it may render difficulties in compiling final opinion in the postmortem report.

Poisoning Case

Take care to note following observations as found during the autopsy examination:

- Smell
- Color of PM stain
- Froth around mouth and nostrils
- Corrosions
- Any injuries, fang marks, etc.
- Gastrointestinal tract findings.

Note: Send viscera for chemical examination (FSL).

Hanging/Strangulation Cases

Take care to note following during the autopsy examination:

- Ligature material and mark
- Salivary dribbling marks
- Face findings (eyes, pupils, and tongue)
- Injuries to spinal vertebrae.
- *Bloodless dissection of neck* Here the cranial and thoracoabdominal cavities are dissected first, and neck and neck structures dissected last.

Firearms Injuries

Take care to note the following during the autopsy examination:

- Clothes
- Cadaveric spasm
- Radiological examination
- Details of wound (terminal ballistics)
- Projectile if any must be collected, preserved properly with proper marking, etc. and dispatched to FSL.

Drowning

Take care to note the following during the autopsy examination:

- Changes in hand, feet, skin, etc.
- Froth around mouth and nostrils
- Cadaveric spasm
- Injuries (reconstruction of case)
- Air passages and lungs
- Diatom test
- Viscera for chemical examination (FSL)

Burns

Take care to note following during the autopsy examination:

- Smell
- Nature of burns (note antemortem postmortem, by looking for vital changes)
- Age of burns or time since burns injury
- Color of postmortem stains
- Look for presence *of soot particles* in air passages suggestive of antemortem burns.

Criminal Abortion

Take care to note following during the autopsy examination:

- Evidence of pregnancy and gestation period
- Criminal abortion—evidence
- Toxic substance—evidence
- Sepsis, emboli, complications, etc.

Road Traffic Accidents

Take care to note following during the autopsy examination:

- All injuries must be described
- Any foreign particles—preserved
- Alcohol—stomach, blood, urine
- Eyes—vision impairment evidence.

NEGATIVE AND OBSCURE AUTOPSY

It is a fact that a medicolegal autopsy result may not be favorable in deciding every case. It could be *negative* or *obscure*. Each of these is discussed in brief.

Negative Autopsy

An autopsy, which fails to reveal the cause of death with gross, microscopic, toxicologic and other laboratory investigations is defined as *negative autopsy*.

Causes

Various causes are enumerated as follows.

- Inadequate history especially in cases of death due to vagal inhibition, hypersensitivity, etc.
- Lapses in external examination as in case of death due to snake bite, other insect stinging, etc.
- Inadequate or improper internal examination, e.g. air embolism or pneumothorax missed.
- Insufficient laboratory examination.
- Lack of toxicological analysis
- Lack of proper training to perform an autopsy properly.

Obscure Autopsy

An autopsy done *meticulously, properly and perfectly*, but may present with no clear-cut findings as to give a definite cause of death, leading to *perplexity* of the forensic pathologist is defined as *obscure autopsy*.

Causes

Causes of obscure autopsy are usually unexplainable. However, several possibilities which are speculated and thought of in a favourable direction to decide the case, are as follows:

1. Natural causes such as:
   - Obscure morbid anatomy
   - Obscure histopathology
   - Emotional causes
   - Paroxysmal fibrillation, etc.
2. Biochemical disturbances like uremia, diabetes, etc.
3. Concealed trauma, such as concealed punctured wound.
4. Endocrine disturbances such as conceded fimctired wound.
5. Poisoning due to:
   - Anaesthetic over-dosage
   - Delayed narcotic poisoning
6. Miscellaneous—such as putrefied body.

Medicolegal Importance of Negative or Obscure Autopsy

1. In autopsies, which are obscure, medical examiner must not hesitate to state that no *opinion can be given regarding the cause of death.*
2. No doctor should entertain a guesswork in establishing cause of death for the reason that medicolegal autopsy is to exclude foul play and derive justice.

FOETAL AUTOPSY

Like a medicolegal autopsy of an adult, foetal autopsy is also an essential work done by a medicolegal expert. The various aspects of foetal autopsy can be studied under following heads.

Objective

In a case of alleged infanticide wherein a foetal autopsy is most commonly called for, we should ascertain the following:

- What is the intrauterine age of the foetus?
- Is it viable or not?
- If viable, is it liveborn or not?
- If liveborn, how long did it survive after birth?

  An examination of the foetus may also be required in cases of criminal abortion and concealment of birth to establish:
- Material recovered is a human foetus or not?
- If yes, ascertain the intrauterine age of the foetus.

Legal Formalities

As in any other autopsy, the investigating police officer has to give a requisition for the foetal autopsy with brief history of the case, maintaining all other formalities as meant for the adult medicolegal autopsy.

Procedure

The special objectives in a case of infanticide require certain special examinations and tests to be done besides the routine dissection to find out the cause of death. A detailed examination is done as an adult autopsy, and has been discussed under following two heads.

External Examination

A thorough external examination is done noting following facts.

Clothes and wrappings—if any may be noted for purposes of establishing the identity.

Postmortem changes—describe in detail.

Signs of maceration—the skin of the macerated foetus is coppery red in colour. The body parts are flaccid and flat when placed on a table. The bones appear to be separated.

Umbilical cord—tied or torn, or with signs of inflammation.

Placenta—note whether attached or not, measure the weight, and look for any infarcts, disease, etc.

Signs of maturity *(intrauterine age and viability)*—the various factors which are helpful in this context are as follows (Figs 14.17A to F):

- Crown heel/rump length with a flexible tape
- Weight
- Midpoint of body in relation to sternum and umbilicus.
- Skin—wrinkled or smooth with presence of fat, covered with *vernix caseosa*.
- Nails—appeared or not. If present–extent of growth.
- Scalp hairs—appeared or not.
- Eyelashes and eyebrows—appeared or not.
- Eyelids—adherent or open.
- Testicles—ascertain the position by incising the scrotum and inguinal canal if necessary.
- ***Ossification centres:*** Special emphasis may be given to note for ossification status of *certain bones only* in certain parts of the body by *special procedures of dissection* as described below (Figs 14.17A to D):
  - ***Knee joint:*** Open the joint by a transverse incision on the front. Reflect the soft tissues upwards and bring out the lower end of the femur. Make thin transverse slices with a cartilage knife starting from the periphery and look for the reddish ossification centre (Fig. 14.17A). Make further slices. Make sure that the diaphyseal end is not mistaken for the epiphysis. Section the upper end of the tibia similarly.
  - ***Ankle bones:*** Make a longitudinal incision on the sole of the foot from the space between the third and fourth toes to the heel. Reflect the lateral flap exposing the outer border of the foot. Make slices in a sagittal plane to expose the centres of the calcaneum, talus and cuboid (Fig. 14.17B).
  - ***Sternum:*** Later the sternal plate is removed and making a midline incision, bisecting it may expose the centres (Fig. 14.17C).
  - ***Sacral segments****:* The sacral centres (Fig. 14.17D) could be examined after other organs are examined.
- Marks of violence on body—look for the mouth, neck, head, etc. in and around for trauma or foreign bodies.
- Cyanosis—look for this in the face, and fingernails.
- Caput succedaneum—over the head (scalp) or buttocks.

Internal Examination

Adopting following steps may accomplish methodical internal examination.

Examination of Skull and Brain

Reflect scalp as usual and cut through the membranous connections of the skull bones. Look for bruising of the scalp, fracture of skull bones, site and extent of caput, moulding, tears in membranes, hemorrhage in meninges, puncture in anterior fontanel, etc. Remove and examine the brain.

Examination of Thorax and Abdomen

- Make a midline incision from chin to pubis avoiding umbilicus. Open the abdomen first and ascertain level of diaphragm. Then reflect the chest muscles, remove the sternal plate exposing the viscera. Note position of heart and lungs *in situ*.
- The floor of the mouth, larynx and deeper tissues of the neck are now examined—for foreign bodies, mucus, meconium and marks of violence.
- Ligate the trachea halfway to its bifurcation to prevent foreign bodies from entering. Apply a similar ligature to esophagus to prevent entry or escape of air. Remove the thoracic organs enmasse by gentle traction.
- Put the whole piece consisting of the heart, thymus and both the lungs in a tall jar containing water and note whether it sinks or floats.
- Look for the presence of Tardieu's spots under the serous membranes. Open the trachea and bronchi and note down the contents. Remove the lungs and heart.
- Make a transverse section of the heart through the ventricles and note any difference in colour of blood on two sides.
- Examine the lungs for its weight, colour, consistency, edges, presence of distended air cells under pleura, crepitation and for conditions like collapse or consolidation. Make further observation when the organ is sectioned. Perform hydrostatic test at this stage.

HYDROSTATIC TEST (*FLOATATION TEST)*

Hydrostatic test is a test done to confirm whether the lungs tested are from a respired newborn or not.

Principle

If the newborn has respired after birth, the air that has entered the lungs shall remain within the lungs as residual air, which cannot be removed even after death, renders the lung lighter and makes it float in water giving positive result.

Procedure

The procedure of the test includes following steps:

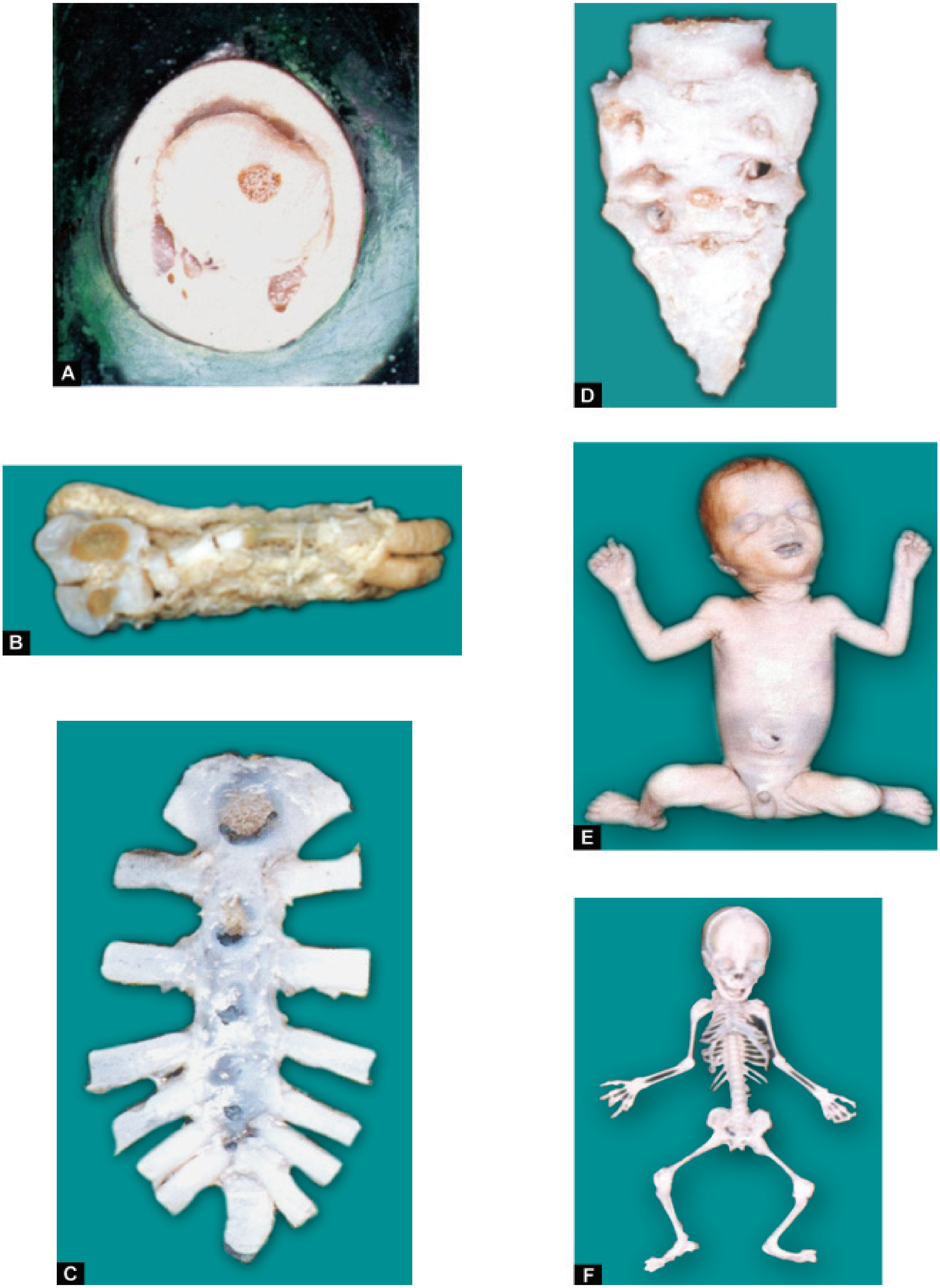
- Put each lung separately into a jar of water, see whether it sinks or floats.
- Cut each into 10 to 12 pieces observe the blood exuding. Test each of them for buoyancy.
- Pick up the floating pieces, and press firmly under water. Observe the nature of bubbles streaming up. Note whether they continue to float or not.
  Note: Fix a few lung pieces for histopathological examination.

Interpretation

- If the entire lung floats, it means that the child had not only respired but respiration had been completely established. In the absence of putrefaction this test itself is conclusive.
- If some pieces float but others sink, the child has of course respired but for a very short period and ineffectively. Yet it is a live born child.
- If all pieces sink, the child never respired and is stillborn.

Fallacies

- *Putrefaction:* An unrespired lung may float due to gases of decomposition.



Figs 14.17A to F: Foetal ossification centres dissected—(A) Knee joint cut opened showing centre for lower end of femur; (B) Ankle bones; (C) Sternal plate and (D) Sacrum, (E) Viable foetus (note the well developed scalp, hairs, eyebrows and eyelids and, nails), (F) Foetal skeleton

- *Oedema lungs, congenital syphilis, pneumonia, lung collapse, etc.:* These conditions can make a respired lung sink in water. But unaffected areas may give a clue to respiration having been established if that is the case.

STOMACH BOWEL TEST (BRESLAU'S SECOND LIFE TEST)

This test is done to determine whether the child was born alive or not.

Principle

Some air is swallowed during respiration in a live born child and detecting the presence of this air in these viscera constitutes the basis for this test.

Procedure

Procedure comprises of following steps.

- Remove stomach and duodenum separately by cutting in ligatures.
- Place them both in water. See whether they float or sink.
- If they float, make a small cut while under water to see air-bubbles coming up.

Inference

- A floating viscera giving out air-bubbles when opened under water is positive test and suggestive of live birth.
- A positive test proves live birth even in the absence of a positive hydrostatic lung test. This may happen, if there had been some obstruction in the respiratory passages.
- A negative test does not mean stillbirth since air does not necessarily enter stomach in adequate amounts during the breathing act.
- Putrefaction invalidates the result.

Other Findings Suggestive of Live Birth

- Open the stomach along the greater curvature and look for mucus and milk, which is suggestive of not only live birth, but also indicates that the infant had lived for some time.
- Examine the large bowel for meconium and urinary bladder for urine, presence of which indicates that the child had lived for some time.
- The umbilical vessels are removed and examined histologically, which is of help in determining exactly how long the child, lived.
- *The ossification centres:* Certain parts of the fetal body are dissected and exposed last to confirm the presence or absence of ossification centre in order to determine the age (see above for details).

AGE OF THE FOETUS

Determination of age of the foetus (Table 14.3) is very essential and crucial in cases such as:

- *Infanticide:* A child which has completed 7 months of IU development is deemed *viable*, i.e. capable of being born alive and reared. The possibility of live birth is ruled out if it is below the age of viability and a charge of infanticide cannot be sustained.
- *Criminal abortion:* To know whether the mother was *quick with the child.* At about 14-18 weeks of pregnancy she feels the *foetal movements from within*. Abortion induced after this period brings *enhanced punishment*. The foetus is examined and its age fixed (Table 14.3) in such cases (Refer Hasse's Rule in Chapter Identity).

WHETHER THE CHILD WAS BORN ALIVE OR NOT

A charge of infanticide can be sustained only when it is proved that the child was born alive, and it was killed by criminal means of acts of commission or omission.

Live Born Child

Live born child is one, which is partly or wholly born external to the mother and showed some signs of life. It is found out by the presence of certain well-defined changes that occur in the body after birth and known as "signs of live birth."

Dead Born Child

Dead born child is one, which had died in uterus long before labor started. It is diagnosed by the presence of maceration, i.e. a peculiar change that a dead fetus undergoes when it remains in the uterus without being expelled.

Stillborn Child

After being born the child never showed any sign of life. It might have died during delivery. It shows neither the signs of maceration nor positive signs of live birth. A complete autopsy may give the cause of stillbirth.

To ascertain whether the child is born alive or not, the following should receive consideration *(see procedure above).*

1. *Signs of maceration:* If signs of maceration are present, live birth can be ruled out.
2. *Signs of immaturity:* Estimate the age of the fetus by observing the length, weight, midpoint of the body, skin, nails, scalp hair, eyelashes, eyebrows, eyelids, testicles and ossification centres. If the child has not attained viable age, it could not have been born alive.

Table 14.3: Various changes in a foetus helpful in determining IU age

Findings	*5 months*	*6 months*	*7 months*	*8 months*	*9 months*
Length	10 inches	12 inches	14 inches	16 inches	18-20 inches
Weight	1 lb	1-2 lbs	3-4 lbs	4-5 lbs	5-7 lbs
Midpoint	At Xiphoid	—	Midway	—	At umbilicus
Skin	Wrinkled	Vernix	Fat, Vernix	Fat, Vernix	Plump
Scalp hair	Light	Distinct	1 cm	1.5 cm	Dark and 2 cm long
Eyebrows	Nil	Appears	Distinct	—	—
Eye lashes	Nil	Appears	Distinct	—	—
Eyelids	Closed	Closed	Open	—	—
Nails	Appear	Distinct	Near finger tips	At finger tips	Project beyond finger tips
Testes	On psoas	—	At int. ring (Inguinal)	In canal (Inguinal)	At ext. ring or scrotum
Ossification	Calcaneum	Calcaneum Sternum	Talus	5th piece sacrum	Lower end of femur, cuboid

3. *Signs of respiration (most important in criminal cases):* There are several gross and microscopic findings, which can suggest whether a newborn has respired, or not. Many of these are depicted in Table 14.4.
4. *Confirmatory signs of live birth:*
   - Milk in the stomach
   - Absence of meconium in the large bowel, exception being breech delivery.
   - Exfoliation of the skin
   - Closure of foetal channels
   - Changes in the umbilical cord.

Note

- If the child shows undoubted signs of maceration it is proof that it died *in utero* and the question of live birth does not arise. However, putrefaction should not be interpreted as maceration.
- Similarly the child has not attained the period of viability, it is not possible to be born alive. In both above cases further examination is unnecessary.
- On the other hand the child does not show signs of maceration and has passed the age of viability, the question of live birth should still be proved.
- In criminal cases proof of live birth requires proof of having respired. Of the different signs of respiration, only two are reliable, *viz.* hydrostatic test and stomach bowel test.
- The confirmatory signs listed above are conclusive evidence that the child was not only born alive but survived for some time.
- In the absence of these signs of live birth one can safely pronounce the child as having been stillborn.

MURDER, SUICIDE OR ACCIDENT

Strictly speaking the doctor's responsibility in connection with a death is only to determine, and often certify, the medical "cause of death." For example, a stabbed person may be certified as *"stab injury of the chest,"* a poisoned person as *"organophosphorous toxicity"* and a presumed natural death as 'myocardial infarction'. However this medical cause of death requires further clarification for legal purposes, into manner of death, i.e. homicide, suicide, accident or natural cause.

World Health Organisation (WHO) has published a booklet "Certification of Death," instructions are given in this book to doctors to enter the manner of death on death certificate, along with medical cause.[7,9] However, in most of the western countries this is not appropriate, as the decision as to the manner of death is reserved to a legal official, not to a doctor.

In most countries of the world including UK, many commonwealth countries, parts of USA, and even in India at Mumbai (not at present), the "Coroner" has this function. In most of the other countries with continental system of law, a judge, the police or some other official authority makes the decision, as mentioned in Chapter 3, Doctor and the Law. In certain parts of USA where there is a prevalence of "medical examiner" system, it is indeed a doctor who makes the decision as to the manner of death, but this is because a medical examiner is an official state appointment combining the functions of pathologist and coroner.

However, in many of the parts of the world including developing countries, the certifying doctor himself whether he or she be a clinician or pathologist, may have to decide on manner of death, as well as the medical cause. Of course, in all jurisdictions, the legal official relies heavily on the doctor, especially one who has carried out an autopsy, to provide much of the evidence which will decide between accident, suicide or murder, especially when an eye witness or other circumstantial witness is deficient. Therefore, some knowledge of facts, which help to differentiate between these four main categories, is certainly useful for all doctors.

The differentiation between accident, suicide and murder varies according to type of fatality involved, so these are discussed under the various type of trauma, more details of which can be found in other chapters of the book.

FIREARMS INJURIES

Following features can assist the distinction between murder, suicide or accident.

- Range of firing beyond arms length cannot be suicidal, unless otherwise certain special mechanical devices are used.
- Absence of weapon at the scene of death excludes suicide.
- Suiciders use *"sites of election"* to kill themselves with guns. These include sides of the forehead (temples), mouth, under the chin and over the heart. It is also not true that a right-handed person would always shoot in the right temple or vice versa. Though this is common there are several exceptions.
- Homiciders usually choose sites like inaccessible parts of the body (for a victim)–such as back of the neck or trunk.
- Regarding wounds inflicted on eyes, back of the head, abdomen, etc. accident or murder is highly likely. Back of the head or nape of the neck is a traditional "execution site" in terrorism or assassination.
- Women rarely shoot themselves, or others. Accidental firing by women is also unlikely for the reason that they rarely have interest in firearms as men have.

All other factors a doctor has to take note in firearm injury case are to take a swab of skin of the hands, for gunpowder

Table 14.4: Enumerating the signs of respiration

	Characteristics	*Before respiration*	*After respiration*
a.	**Shape of chest**	flat	arched
b.	**Diaphragm** at the level of	4-5 ribs	6-7 ribs
c.	**Lungs: size**	small	voluminous
	Fodere's test	500 grains	1000 grains
	Ploquet's test	1 : 70	1 : 35
	Edge	sharp	rounded
	Colour	reddish brown	mottled-pinkish red
	Consistency	liver-like	spongy and crepitant
	Section	dark blood	red blood
	Hydrostatic test	sinks	floats
d.	**Stomach-bowel test**	sinks	floats

residue. When a bullet, pellet, wad, or other missiles are detected on autopsy or surgery, they should be carefully preserved without damage. It is usual to excise the wound at autopsy and send it without formaline fixation to forensic science laboratory for analysis for gunpowder/propellant residues.

KNIFE WOUNDS

Suicidal knife wounds have a characteristic pattern, though a murderer can even simulate these very occasionally. Following help to decide suicide:

- Presence of tentative cuts or hesitation cuts
- Site—usually seen on the wrist, throat, etc.
- In right-handed victims, the cuts in the throat are often deepest on the left side while it tails off to the right, passing obliquely downwards across the larynx, which may be badly damaged.
- Homicidal cut throat is usually more severe and lacks tentative cuts. However, multiple neck wounds due to skidding away at an angle to main cuts due to the movement of the victim may also be observed in homicidal neck wounds.
- Homicidal cut wounds are usually deep even into the vertebral column.
- In self-destruction, the stabs are most often into chest, though some may be made into the head through skull. Abdominal wounds are uncommon, as are gunshots, as it is well known that death may be uncertain or lingering and painful.
- Suicidal chest wounds are usually over left side, where the heart is generally known to be situated. Stabs are usually single, but multiple stab wounds by no means indicate homicide, even if each wound is potentially fatal. Many of the suicides who stab themselves in the chest, pull aside the clothing first, a feature which may be useful in interpreting the motive, as homicidal stabbings invariably penetrate the clothing.
- Homicidal stab wounds have no particular characteristics and can only be identified if they are into inaccessible parts of the body such as the back or if the weapon is present at the scene of death.
- Fatal accidental stab wounds with knives is unusual, but may occur in butchers, slaughtermen, etc.
- Fatal stab wounds from falling through windows and glass doors, etc. are also well recorded, but the circumstances are usually obvious.

HANGING, STRANGULATION AND SUFFOCATION

Often these asphyxial deaths give rise to considerable difficulties for doctor and investigators in distinguishing between accident, suicide and homicide.

Hanging is almost always suicidal and although more common among men, is not unusual in women. Homicidal hanging is very rare and unless the victim is drugged or drunk, hanging cannot be accomplished in resisting conscious person, unless restrained by tying up, etc. Self-strangulation is virtually impossible with the hands. In ligature strangulation, whether murder or suicide, the mark on the neck takes generally a horizontal course, even though this might be high up under the chin and angles of the jaw. In hanging, there is usually a rising ligature mark to the place where the knot is situated, generally leaving a gap *missing gap* in the skin where the rope leaves the body surface to ascend to the knot. However this distinction is not always reliable, if the hanging ligature has a slipknot, the rope will tighten on the neck and fail to leave a *missing section* in the mark. Also it may not *rise* to the knot, if the attachment of hanging rope is relatively low, making the ligature mark look horizontal and complete, indistinguishable from actual ligature strangulation.

In young adolescent/adults/even middle-aged *sexual asphyxia* must be thought, as *masochistic—autoerotic hanging* is common in this age group. These activities usually amount to accidental hanging, here death not being intended by the man who is indulging in *sexual fantasies* produced by *temporary cerebral ischemia*, by a controlled hanging *(refer Asphyxial Death)*. The police often who are unaware of this syndrome may label the death as suicide or even murder.

Suffocation, by external soft fabrics, pillows, cushions and plastic sheets almost never leaves any signs and, thus, cannot be differentiated from other natural deaths if the obstructing object is removed before the doctor or other witness attends the scene.

Sufficient pressure exerted may cause abrasion; contusion, etc. which may create suspicions. At the same time care must be taken not to mistake postmortem blanching around the mouth and nose as signs of deliberate suffocation. Thus in a case of suffocation, in such case is dependent on circumstantial evidence mostly.

DROWNING

Drowning again it is circumstances, which will be more helpful in distinguishing the manner of death. Removal of clothing, spectacles, etc. and kept on the river banks are suggestive of suicide than accident or homicide. A full examination of dead body is essential to exclude natural diseases, which may have caused a fall into water, and injuries sustained before entering the water may point to homicide. The facts that tying of hands and legs/tying some heavy objects like stone, bricks, etc. by a determined suicider need to be convinced that the tying is by the victim himself or herself, as otherwise it might be done by a criminal who might have tied his or her victim prior to pushing them into the water.

SELECTED SPECIAL PROCEDURE OF AUTOPSY

Certain selected autopsy procedures and techniques that are helpful in the routine performance of medicolegal postmortem examinations are discussed here[9-11] and they include:

- Demonstration of air embolism
- Demonstration of pneumothorax
- Demonstration of thrombi in the calves
- Reconstruction of the skull for personal identification and determination of type of violence
- Removal of the jaw for dental identification
- Removal of spinal cord by anterior approach
- Fixation of the brain
- Autopsy photography.

DEMONSTRATION OF AIR EMBOLISM

Presence of air in the vascular system is demonstrated by easy and accurate methods. Interrupted blood column, i.e. fragmentation of the blood line in the coronary arteries, meningeal arteries or arteries elsewhere are usually artifactual and must not be mistaken for air embolism. Usual indications for air embolism are:

- Suspected criminal abortion
- Open wound of the neck.

Principle: To demonstrate air in the vascular system, i.e. heart.

Procedure

Heart is exposed by routine autopsy incisions and sternal plate is removed with ribs up to the costochondral junction (Figs 14.18A and B).

- Now without disturbing the tissue arround as to prevent negative pressure and aspiration of air in to the vessels, perform following carefully (Figs 14.18C and D).
- The pericardial sac is now incised anteriorly, and the edges are grasped with haemostats on either side and held firmly. It is better that an attendant holds the haemostats.
- Now fill the sac with water till the heart is submerged.
- The right side of the heart is then punctured with a scalpel under the water level. The scalpel should be twisted several times to ensure the heart wound is open.

Observations

If air is present in the heart, bubbles will rise, otherwise the blood will flow out from the wound.

Measuring the Amount of Air

Invert water filled measuring glass cylinder (300 ml) over the heart, with the mouth of cylinder under pericardial water column. When the heart is stabbed, the gas will escape into the cylinder displacing the water in it allowing to measure it directly.

DEMONSTRATION OF PNEUMOTHORAX

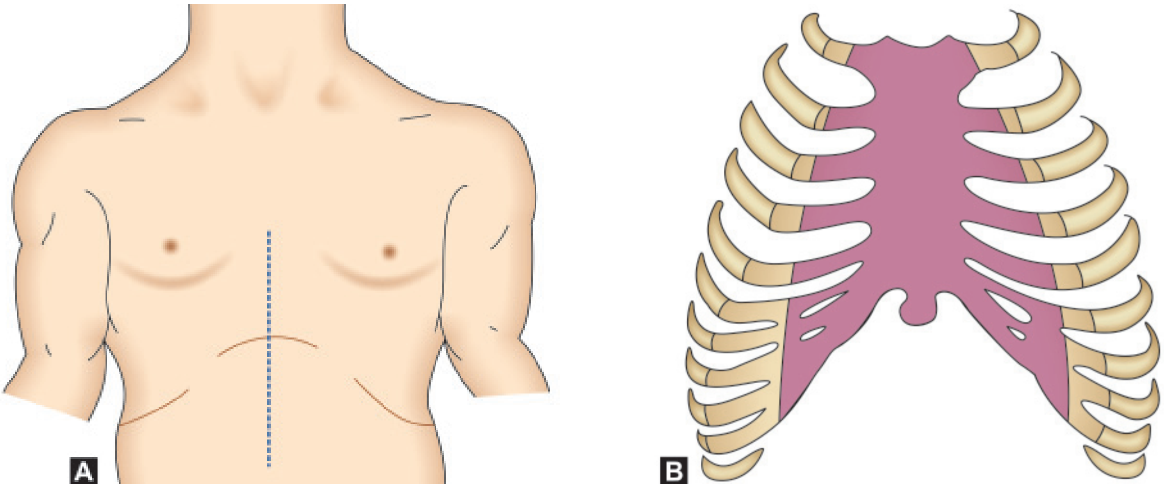
Pure form of pneumothorax is rare. It is usually associated with injury to lung, resulting in blood in the pleural cavity or hemopneumothorax. Thus every case of chest injury is an indication for checking for the presence of air or pneumothorax.

Principle: To demonstrate air in the pleural cavity.

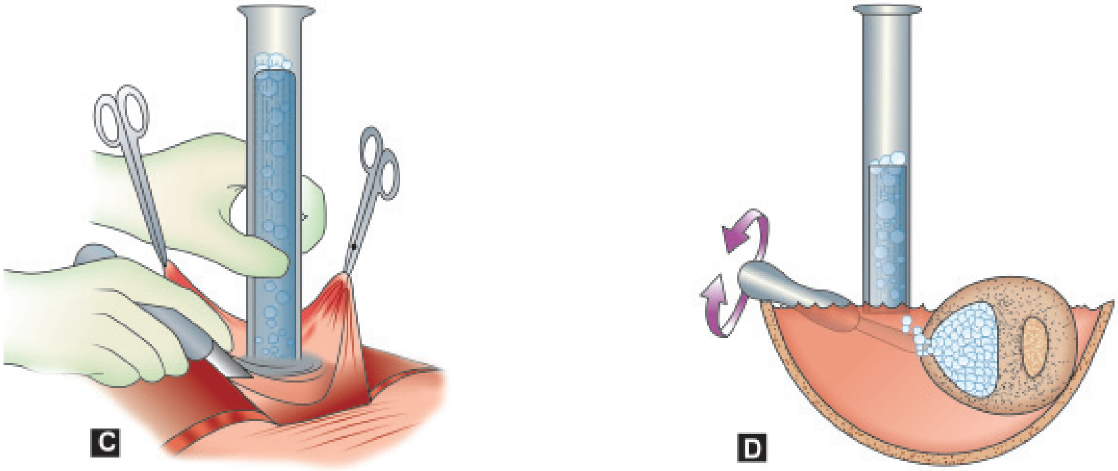
Procedure

Perform following steps carefully (Figs 14.19A and B):

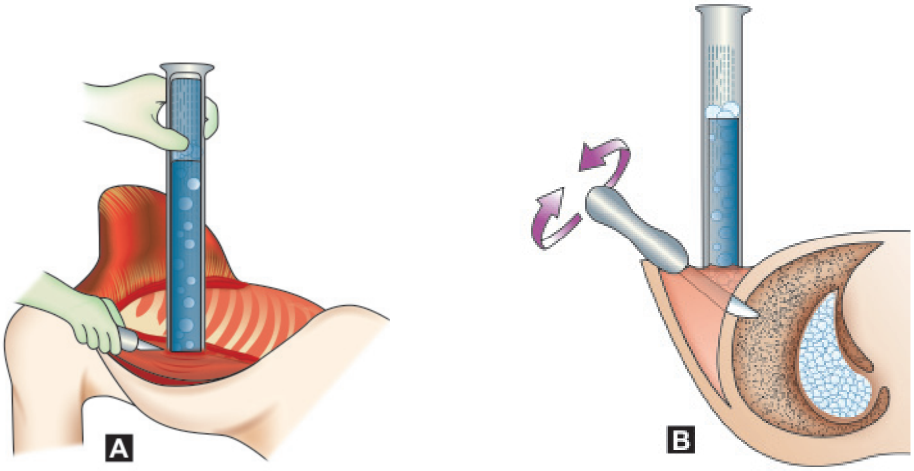
- Incision of the body as in any routine medicolegal autopsy examination and the skin and muscles on the injured side are reflected and dissected to form a *pocket*. This pocket is then filled with water.
- Invert now water filled measuring glass cylinder over the pocket, with the mouth of cylinder under water in the pocket.
- A scalpel is then introduced under water level into the costodiaphragmatic sinus through an intercostal space.
- To ensure the wound is open, the scalpel is twisted a few times.
- If air is present in the pleura, bubbles will come out from the wound and get collected into the glass cylinder and directly measure the amount of air in the pleural cavity.



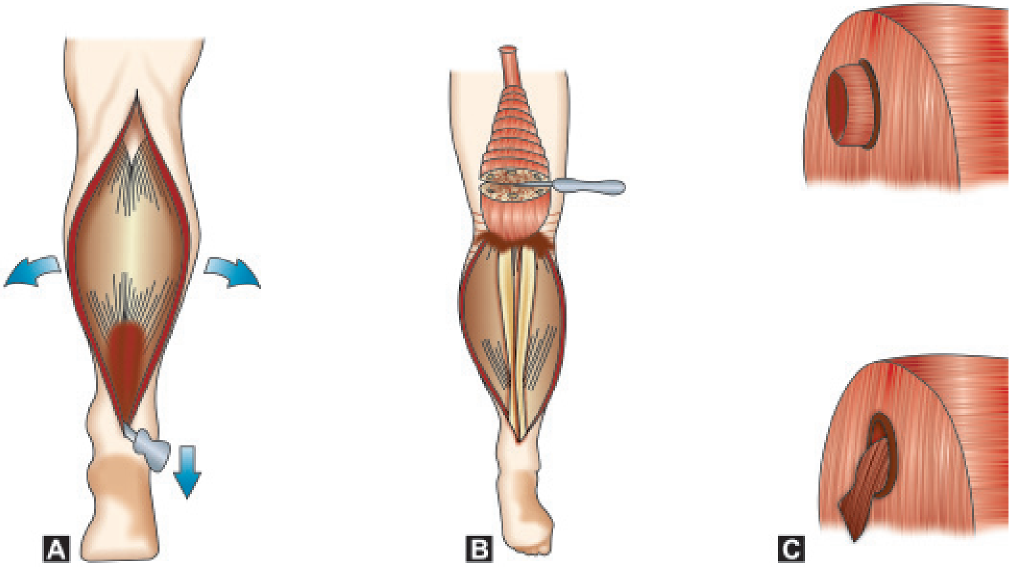
Figs 14.18A and B: *ML autopsy special procedure* demonstrating air embolism in the heart. (A) I-Incision of the skin to expose heart; (B) Line of sectioning costochondral junction and removing the sternal plate



Figs 14.18C and D: (C) Incising the pericardial sac and filling with water. Artery forceps used to elevate the sac and water filled measuring jar inverted and kept in the right side of the heart, allowing air when released by puncturing the heart collected in the cylinder; (D) Cross section view showing scalpel puncturing the heart and air entering into the jar



Figs 14.19A and B: (A) Create a pocket with water in the side of the chest and invert a measuring jar with water into it. Next pierce the intercostal space under water and see air bubbles out and flow into the jar. (B) Side view, presenting the scalpel piercing the intercostal space and gas bubbles entering into the jar



Figs 14.20A to C: (A) Skin incision exposing the calf region and muscles. (B) Separate the muscles from its bone attachment and reflected upward, is then sliced transversely, will disclose presence of thrombi, as it pops out as sausages from transected vein (C above). If it is a postmortem clot it will be flabby and does not pop (C below)

DEMONSTRATION OF THROMBI IN THE CALVES

Causes

Formation of thrombi in the calves is common complication of prolonged bed rest or may be due to direct trauma to the legs. Dislodging of a leg vein thrombus may cause fatal pulmonary embolism.

Procedure

Perform following steps carefully (Figs 14.20A to C):

- Incise the calf from the heel to the poplitial fossa, and reflect the skin.
- Divide the tendon of Achilles.
- With a long knife the calf muscle is then separated from the bones from the heel upward.
- Avoid excessive traction and handling of the muscles.
- The calf muscle is then sectioned 2.5 cm apart
- Thrombi if any will pop out as firm, solidly structured sausages, on pressure, which cannot be mistaken for postmortem artifacts.

The major arteries of calf pass between the tibia and fibula and they remain intact in this procedure if carried out properly.

RECONSTRUCTION OF THE SKULL FOR PERSONAL IDENTIFICATION AND DETERMINATION OF TYPE OF VIOLENCE

At times the skull is fractured to such an extent that facial features are not recognizable and the type of violence responsible for the mutilation cannot be ascertained. Restoration of the contour of the skull frequently provides the answer to these questions. Using an electric drill and copper wire or quick fix gum one may carry out replacement and fixation of the bone fragments.

The fragmented skull and badly mutilated face in a victim of explosion is a good indication for this type of reconstruction.

REMOVAL OF THE JAW FOR DENTAL IDENTIFICATION

Identification of a dead person by dental comparison is often the only choice in case of extensive mutilation of the body or advanced putrefaction. Disarticulation of the mandible and excision of the upper jaw may be necessary under such circumstances to ensure adequate examination and possible preservation as evidence. Using an electrical or manual saw, the upper jaw is excised by means of horizontal cut placed above the hard palate.

EXAMINATION OF THE CERVICAL SPINE FOR WHIPLASH INJURY

An autopsy is incomplete without a thorough examination of cervical spine in cases of RTA or death due to fall from height. The so called whiplash injury is usually situated in the level of C-4 to C-6, with C-1 and C-2 being involved frequently. The whiplash injury range from severe fracture dislocations to a few deep hemorrhages due to tear of individual muscle fibres in the musculature. Tears of articular capsules and ligaments with intra-articular or periarticular hemorrhage is common in the first two vertebrae. Following technique facilitates demonstration of whiplash injury during autopsy examination.

Procedure

Perform following steps carefully:

- Keep the body in prone position (Face down) on a wooden block under the chest with head bent downward. This position will stretch the cervical spine.
- Now open by midline skin incision and dissect the neck musculature layer by layer down to the vertebral column.
- Atlanto-occipital joint capsule are then incised and the articular surfaces are examined.
- Subsequently, the atlas is disarticulated and removed.
- Laminectomy is then performed on the remainder of cervical spine; the duramater is incised, and the spinal cord is inspected *in situ* and then removed.
- The under surface of the base of the skull can now be inspected as forward as the pharynx. Replacement of atlas and tight suture of the incision in the skin permit good reconstruction with fair stability of head.

REMOVAL OF SPINAL CORD BY ANTERIOR APPROACH

Spinal cord is the most neglected structure during autopsy. It remains untouched, unless threre is a specific indication. Basically this is due to the fact that, it is not only a laborious procedure but also time-consuming. However, search in literature in clinical pathology, explains an easy anterior approach which render removal of the spinal cord within 2-3 minutes.[21] The method required no special requirements. A Stryker saw, a sturdy knife, a chisel and a hammer are the only requirements. With this spinal cord can be removed intact and is good for both gross and histopathological examinations. This method also avoids incision of the back.

Procedure

Perform following steps carefully: When all other viscera are removed completely from all the three major body cavities, removal of spinal cord is done as follows:

- Initially, any intervertebral disc in the upper lumber spine is transacted, with another disc in the lower thoracic area.
- Now using Stryker clectric saw cut out the segment of vertebral bodies between the two separated discs. Chiseling may be helpful in removing the bony wedges. This will expose terminal dural disc, which must be now slit vertically and reflected sideways.
- Next, the lowest portion of spinal cord is cut and a moist cloth is wrapped round the proximal segment.
- Finally, with slow and gentle traction downwards, towards the feet, bringing the spinal cord out with its dural coverings in an excellent state of preservation there will be no artifactual tears, nor any distorted histopathology.

FIXATION OF THE BRAIN

The brain is usually suspended in a bucket of chilled formlin, using a string through the circle of Willis. The bucket with brain is better preserved in the body refrigerator. Adding a little of kitchen salt can help floatation of the brain in solution. However, drying of the area of brain, which floats above the level of solution, can be prevented by keeping it wet by covering the area with a paper/ towel, soaked in the solution. The shrinkage of tissue by salt dissolved in formlin is minimal and not detectable microscopically. This method of fixation is advantageous by preventing the following:

1. Contact flattening of surface in contact with the bottom of the container.
2. Rapid growth of anaerobic bacteria in the brain, developing air bubbles in the brain tissue.

AUTOPSY PHOTOGRAPHY

According to Spitz U W,[10] inspite of the growing popularity of amateur photography in pathology departments, photographs, though often in colour and generous size, fail to relate the desired information. Some suggestions in forensic photography, which are thought to be useful, are enumerated below:

1. Shoot always the photographs of the cadaver/part of the body at right angles from above, to reduce the perspective distortions.
2. Do not make photographs too gory. Photographs showing abundant blood on the body or in the background are often considered as offensive and are not admitted as evidence in the courts, as they tend to prejudice the law and preclude dispassionate judgement. A clean paper / cloth used as background for the body will be less distracting and also help obtain clearer and less gruesome picture. Extraneous objects, such as autopsy knife, scalpels, scissors, etc. must be excluded in case the photograph is used in evidence.
3. Cotton plugs in the nostrils, ears, hospital bandages and wrappings should be removed from the body and the part must be wiped clean and removing the dry blood adherent to the surface showing all particulars. Care must be taken not to remove the evidence of close range gunfire, when body is thus prepared for photography purpose.
4. The PM case number, date of autopsy and a suitable size scale should be included in every case photographed. This should be clearly legible and positioned in the same plane as principle points of interest. The case number must be placed in a corner or along one edge of photograph, enabling its removal, should this become necessary. Suitable scales are available commercially or can be manufactured

as required. A pointer such as an arrow, narrow triangle of thin cardboard, may be used where a lesion is not readily vissible.

5. In violent death cases, front and back views of the entire body with clothes and in the nude are recommended. It is important to show the condition of the body when first viewed. Two shots of the body in nudity are mandatory and they are: (a) distant shot to indicate the location of the injuries; (b) close up shot of major wounds to show details. The close up photograph must allow the viewer to get a clear concept of position with reference to the area of the body.
6. Identification of the victim is often made easy by a photograph of the face. Repair of the facial injuries and application of making up by a well-qualified mortician are often helpful in such cases.
7. Tattoos mostly help in the identification of the unknown. However, excessive skin pigmentation, too old tattoo, etc might obscure the tattoo. However, removal of the superficial layer of the skin is helpful in such cases.[10] Creating second-degree burns of the the tatooed area followed by wiping off the detached epidermis is the best method in visualising an invisible tattoo. Placing the tattooed area under a hot lamp will produce the desired effect in several minutes. An incidental observation is that tattoos frequently fade considerably and may be even disappear entirely with time. This is more commoner with home made tattoos in comparison to the professionally made tattoos. This is basically because of amount of pigments, which are less in such tattoos than in professional ones. Thus when a tattoo described by the relatives are not seen in the cadaver at autopsy, it could lead to mistaken identification. In such cases the best method is to *demonstrate the pigment* used for tattooing in the *regional lymph nodes* draining the area tattooed, microscopically by histopathology techniques.[8-10]
8. Palmar surface of the victim's hand is often necessary to be photographed show electrical burns, defense cuts, bullet wounds or discolouration of the skin due to handling of certain drugs, etc. These are often neglected or they are spoiled by tying strings to fingers to overcome effects of rigor mortis. Severing the flexor tendons of the fingers can be easily performed through a 1.5 to 2 cm horizontal incision at the wrist. This procedure will render complete opening of the hand and give better exposure with proper lighting and photography.

SECOND AUTOPSY

After the first autopsy (Postmortem examination) when a repeat medicolegal postmortem is performed with the authorisation by law it is called ***second autopsy***. Such a second autopsy is usually carried out at the postmortem centre affiliated to the Medical College, by a senior forensic expert. A panel of doctors may also perform the second autopsy. According to the merit of the case, other experts in medical profesion may also be consulted.

The second autopsy is ordered/performed in extraordinary circumstances such as:

- The relatives are not satisfied with the first autopsy report and they have pointed out certain lapses in first autopsy.
- The first autopsy was not performed in the light of the circumstances which are now available.
- The investigating authority wants the second autopsy for setting the investigation on right track.

The magistrate/commissioner of the police vested with such power, orders the second autopsy. With the written order/authorisation second autopsy is done. Before starting the second autopsy one has to confirm proper authorisation. Panel of doctors should be formed. The first autopsy report, first inquest report, relevant facts of the case should be studied carefully along with other relevant papers/reports, if any. If there is an ambiguity/discrepancy in the first autopsy report it may be necessary to have the presence of/discussion with the doctor who performed the first autopsy.

The second autopsy is performed in the same line as any standard autopsy. Complete external examination, internal examination, with necessary viscera (samples) preservation for chemical analysis or histopathology examination as required. In case of alleged history of injuries, the body parts should be incised at suspected areas to see deep contusions. Special attention should be given to neck and neck structures and brain. It is essential that objectivity be maintained in difficult circumstances and the autopsy surgeon should take utmost care to collect the evidence. Even if the viscera were preserved during first autopsy, whatever viscera available presently can be preserved for chemical analysis during the second autopsy.

Advantages

The second autopsy is beneficial as it clears all the suspicions of the relatives and also the descrepancies of the first autopsy and helps in the administration of justice. If in the first autopsy certain things are omitted/neglected or not done deliberatily and therefore certain areas not properly examined hence second autopsy certainly has greater value. Second autopsy helps to set the investigation on right track and can act as safeguard to contribute to the fact that justice is being done.

The second autopsy can also be done even if the dead body is buried after first autopsy. However, body has to be exhumed or disintered by the order of a magistrate.

In England and Wales there are three factors contributing to the increased frequency of second autopsy. The first reason is that it is done as an integral part of defense preparation even if there is guilty plea. Second reason for the increase of second autopsy goes hand in hand with an increasing hesitancy of the public to accept unquestionably their medical practitioner's opinion or advice on death of a relative or kith and kin. Third reason relates more specifically to the cases of homicides. The Criminal Justice Act, 1967, made it possible for the accused to be committed for trial 'without consideration of the evidence', and this with the consent of the accused, is now the norm.

A 35 years old British engineer who died while in the Middle East. A diagnosis of "acute hemorrhagic Pancreatitis' was made at the postmortem examination. At second autopsy performed ten days later the cause of death was reported to be due to an extensive haemorrhage in the transverse mesocolon,[19,20] justifies second autopsy as and when called for.

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EXAMINATION OF BONES

When skeleton or isolated bones are sent for opinion, the usual questions that a police-officer poses to a medical officer are:

i. Whether the bones are human or not?
ii. If human, whether they are male or female?
iii. Whether they belong to one or more individuals?
iv. The statures of the individual to whom the bones belonged.
v. The age of the individual to whom the bones belonged.
vi. The time of death.
vii. Where the bones have been cut, sawn, gnawed by animals or burnt?
viii. The probable cause of death.

Conflagrated Human Remains — Unburnt and Burnt Skeletal Remains

At times, burnt bones and ashes are sent to the medical officer for opinion, if foul play suspected and the body is partially or completely burnt. In a house fire, the temperature generated is usually less than 1200°C and therefore it is unlikely that the body of an adult would be burnt so completely as to leave no trace. If the body is not completely burnt, fragments of bones left could create sufficient evidence as to indicate whether the bones are of human or not. Burning of body is rarely so complete as to be reduced to ashes. Hence, by sieving the ashes, fragments of bones can be collected and identified by a careful examination.

Incineration of an adult human body for cremation requires about 1½ hours of time at a temperature of 1600-1800°C, resultant with ashes weighing about 4-6 kg. Such human ash contains bone pieces which may still be identified.[1]

When the soft tissue around the bones are less and thin, the bones show sharp heat induced fractures of the skull and limb-bones (usually transverse). However, charring, calcining and splintering are common in those bones with thick soft tissue, e.g. in femur, pelvis and nuchal areas of skull, with the substance of the bones showing molten appearaince.[1]

Bone is said to turns white when burnt in open, while it becomes black or ash grey when burnt in closed fire. It is also said that burnt bone preserves its shape, but turns into ash powder when touched by the fingers. When exposed to very high temperature, characteristically curved fractures may be produced in long bones and skull. Bones become so brittle and friable on prolonged exposure to fire that in victim of such intense heat, bones readily fractured during transport of the body, or its being moved, or during examination. Hyoid bone may similarly break on manipulation. A forceful stream of water from a fire-hose can fragment a bone like the femur, rendered brittle by exposure to such high temperatures. The skull bursts due to the formation of steam within the skull cavity as a result of intense heat. Such explosive postmortem fractures are accompanied by gaping defects and separation of ununited sutures and protrusion of brain matter through it. Intense heat can lead to desiccation of skull, resulting in postmortem thermal linear fracture, commonly located on either side of the skull, above the temples, usually consist of several lines which radiate from a common centre. In case the appearance is not typical, distinction from an ante-mortem skull fracture may be difficult. Besides this, victim may also sustain, postmortem mechanical fracture of any bone, due to the fall of a wall or a beam of building on fire. It is important therefore, to distinguish between postmortem thermal fracture and postmortem mechanical fracture.

In suspected cases of poisoning by arsenic, all the available ashes and burnt bones should be preserved for chemical analysis. This is because, despite its volatility, it is possible to detect arsenic in large pieces of burnt bones mixed with ashes in cases of arsenic poisoning, for the following reasons:[2]

- Much of the arsenic in bones is converted into arsenates, partially replacing the phosphates of the bones. Arsenates are non-volatile; hence arsenic can be detected in the bones even after burning for a long time.
- Even if all the arsenic were present in the bones in the form of arsenic trioxide or some other volatile form, all the arsenic is not likely to be lost during the process of cremation, as complete combustion of the body does not occur. Hence, some of the unburnt funeral pyre, may detect arsenic in it.
- Arsenic trioxide when heated with salts of sodium or earth group, part of the arsenic act converted into non-volatile arsenite.

EXAMINATION OF MUTILATED BODIES OR FRAGMENTS

Mutilation of a dead body is not always the act of a criminal who wants to destroy all traces of identity and thus to get greater facilities for its disposal. In India, animals, such as rats, dogs, jackals and hyenas, and birds such as vultures, may attack a dead body and mutilate it in a very short time, when exposed in an open field on the outskirts of a village or a town. Besides, it is not an uncommon sight to notice the dead bodies of lunatics, fakirs and pilgrims, lying on the roadside or on remote spots in the vicinity of villages, and being attacked by birds of prey, dogs and other animals. Generally, if the village *chaukidar* happens to find such a mutilated body, he hurriedly makes a report about his macabre discovery to the police, and the remnants of the entire body are forwarded to the medical officer for postmortem examination.

In such cases, the medical examiner should first ascertain if the parts sent are human or not. This is only difficult when a piece of muscle without the skin or a viscus is sent. In such a case, a definite opinion can be given by resorting to the precipitin test, which is equally applicable to blood as well as muscle or any other soft tissue, provided the tissue is not severely decomposed. If specialised laboratory facilities are available, the anti-globulin inhibition test is also definitive. Having determined that they are human, he should try to elucidate the following points:

1. All separate parts should be kept together, and determined whether they belong to one and the same body. Bones from different skeletons can be distinguished by exposing them to a short wave ultraviolet lamp, which shows different colour emissions from different skeletons.[3]
2. The nature and character of the parts should be described, as also the color of the skin, if any.
3. The manner of separation, as to whether they had been hacked, sawn through, cut cleanly, lacerated, or gnawed through by animals.
4. If the head or trunk are available, the sex can be determined from the presence and distribution or absence of hair, general conformation and shape of pelvis, sacrum or femur. If may also be determined from the recognition of prostatic, ovarian, mammary or uterine tissue under a microscope, if available, and unrecognizable with naked eye.
5. The probable age may be ascertained from the skull, teeth, colour of the hair, trunk, size and degree of development of fragments and ossification of the bones.
6. Identification can be determined from tattoo marks, fingerprints, scars, color of hair, condition of teeth, deformities, recent and old fractures, or from the discovery or certain articles of clothing known to have belonged to a missing person in association with the mutilated bodies or fragments of a skeleton. Height can be calculated from the measurements of long bones.
7. The probable time since death may be ascertained from the condition of the parts.
8. The cause of death can be ascertained, if there is evidence of a fatal injury to some large blood vessel or some vital organ.

   Case: A penetrating wound on the left side of the chest, cutting the left ventricle of the heart, was noticed on the mutilated body of the Hindu male packed in a steel trunk and found lying in a first class compartment of the No. 6 down train of RM Railway at Agra Fort Station The head, upper half of the lip, penis and extremities had been severed from the trunk.
9. Identity can be established by superimposing a life size photograph of the head of a person of the skull, and thus reconstructing the features. This superimposition techniques has a limited value and full of difficult problems, but corroborative and even conclusive evidence may be obtained.

   Case: In the case of Ram Lochan V State of West Bengal[4] in the Supreme Court, a superimposed photograph of the face of the deceased over a human skull recovered from a tank was accepted as evidence for establishing identity.

ILLUSTRATIVE CASES

Alavandar Murder Case

A Prabhakar Menon, with the help of his recently married wife Devaki, murdered one Mr. Alavandar, a forty-two year old former military sub divisional officer. The wife Devaki had confessed to her husband of having been seduced by Alavandar before marriage. They killed him in a room of Crown Hotel, Madras. Later on his headless trunk with arms and legs was found in the 3rd class compartment of Indo-Ceylon Express train at Mannamadurai, while the Police at the Royapuram sea beach found his head. Dr KC Jacobs established the identity on the following points:

1. That the trunk was that of Alavandar was established by:
   a. Finger print impression tallied with those in his military service book.
   b. Circumcised penis – Past history of circumcision.
   c. His wife identified his green socks and waist thread.
2. His head was identified by:
   a. Overriding canine teeth in the upper jaw.
   b. The peculiarity of two small holes in the right earlobe and only one in the left.
3. The points that proved that the head and the trunk belonged to the same person were;
   a. Unmistakable reciprocal appearance at the site of the decapitation was seen on comparing the lower part of the vertebra attached to the head and the upper part of the vertebral column attached to the trunk.
   b. Both the head and the trunk separately showed male features.

His height was estimated as 5ft 5"–the trunk being 4 ft 9" and head 8¼" while in his service register it was 5 ft 4½".[5]

The Drum Murder Case

Neighbours for the sake of robbery murdered one Devi Subbamma, aged about sixty – five years, in Masulipatnam. Her body was recovered from a heavy zinc drum, from the platform of Tondiapet Station, where some unknown persons pushed it down from a train compartment. An autopsy was done in Madras, and on the following medico legal points, Dr KC Jacob[6] established certain facts:

1. *Time of death:* Owing to a fairly advanced stage of putrefaction, it was opined that death could have taken place 48 hours before its arrival in the mortuary.
2. *Antemortem or postmortem wounds:* The pathologist, Dr Vishwanathan on examination with naked eye and microscopic evidence, opined that the injuries in the scalp and chest were antemortem.[6]
3. *The cause of death:* Head injuries caused by hitting with the rice pounder and fracture of the ribs caused by pressing on the sides of the chest with the knee could have produced death from shock.
4. *Age and identification of the dead body:*
   a. The presence of grey hair in the scalp and in the pubic region.
   b. The appearance of the gums with complete absence of the teeth in both jaws.
   c. The absence of marked distention of the breast in spite of advanced putrefaction suggested advanced age.
5. *Photograph:* Identified by close relations.
6. *Examination of bones:* From the extent of fusion of skull, the alveolar margins of the maxillae and the mandibles, the general shape of the mandible, the fusion of the manubrium with the body of the sternum and xiphisternum and the general consistent appearances in the pelvis and long bones, it was opined that her age must have been about 60 years.
7. A dhobie mark on her green-bordered sari was also a useful clue.[6]

The Ruxton Case

On the 29 Sept. 1935, several mutilated and dismembered human remains, consisting chiefly of two heads, thorax, pelvis, segments of the upper and lower limbs, three breasts, portions of female external genitals, and the uterus and its appendages, were found lying in the bed of Gardenholme Linn, below the bridge on the Moffat Edinburgh road.

With a view to effacing all evidence of sex and identity, the ears, eyes, nose and lips had been removed from both the heads. Even the uterus with its appendages was removed from one body. The skin of the faces had also been removed and the teeth had been extracted. The terminal joints of the fingers had been removed from the hands, so that no identification could be possible from finger prints or some peculiarities of the nails or fingertips. All the remains were assembled and found to represent two female bodies, apparently well developed and well nourished. From investigations carried out by several specialists, it was proved beyond doubt that these bodies were those of Mrs. Isabella Ruxton, the wife of Dr Ruxton, aged about thirty-five years and Miss Mary Rogerson, the nurse-maid of Dr Ruxton, aged about twenty years, and had both disappeared from the house of Dr Ruxton in Lancaster on the 15th September 1935, and were never again seen alive. Photographs were taken of the skulls and superimposed on those of the heads of Mrs. Ruxton and Miss Rogerson and were found totally in every respect. Casts made of the reconstructed left feet of both the bodies fitted perfectly shoes belonging to Mrs Ruxton and Miss Rogerson.

The police searched the house of Dr Ruxton and found numerous stains of human blood in the bathroom, on the banister, stair rails, stair carpets, pads, surgical towel, and particles of human tissue in the drains. The police subsequently arrested Dr Ruxton, who was charged with having wilfully murdered Mrs Isabella Ruxton, and Miss Mary Rogerson. He was found guilty of murder and sentenced to death.[7]

The Baptist Church Cellar Murder

On 17 July 1942, a gang of demolition workers, who were sent to clear out the damaged premises at 302, Kenninghton Lane, discovered, remains of a body covered with lime and buried under the floor of a cellar at the rear of a Baptist Chapel. The remains consisted of a head lying loose and the trunk with parts of the arms and legs missing. Someone had dismembered the body after death without particular skill and knowledge of the parts. The head was decapitated through the joints between the upper end of the spine and the base of the skull. In order to conceal identity, efforts had been made to destroy tissues by fire. Thus, the scalp and hair, face, eyes, lower jaw, hands and feet were missing. There were signs of burning on the head, down the left side of the trunk and at the level of each knee. Owing to the sprinkling of slacked lime the uterus and soft tissues especially of the neck were well preserved though they were dry.

The remains were removed to the Department of Forensic Medicine at Guy's Hospital, where after cleaning and reassembling the parts Dr Keith Simpson was able to determine the following points:

1. *Sex:* The body was that of a woman from the presence of the uterus, which contained a fibroid tumor, past records revealed that surgery was refused.
2. *Stature:* After making due allowances for missing bones and tissues the height was measured as 5ft ½". By using Pearson's formulae and Rollet's tables the height was also estimated to be 5ft ½" from the entire humerous of the left side, which was available.
3. *Age:* An X-ray examination of certain bones showed that the bones were those of an adult and probably middle-aged. The age was further fixed at 40 to 50 by study of the fusion between the plates of the vault of the skull and the palate. There was complete fusion between the brow plates, fusion was in progress between the top plates and fusion was lacking between these two groups. The sutures of the palate had also not united. A number of well preserved hairs on a minute fragment of scalp which lay crushed on to the back of the head showed that the color was dark brown, going grey.
4. *Cause of death:* Strangulation (throttling) was found to be the cause of death from the presence of some deep crimson blood clot extravasated between the tissues surrounding the upper horn of the right wing of the voice box and a fracture of the horn of that wing of the thyroid cartilage under this clot. The horn was forced inwards towards the windpipe. There was also a bruise to the back of the head, which might have been caused by the head being dashed against the ground while the throat was gripped or might have followed upon a fall backwards to the ground.

It was further established from the dental records cards of the treatment of the teeth in the upper jaw kept by the dental surgeon, from superimposed photographs of the available skull and the original portrait and from the fibroid tumour of the uterus that the remains were those of one Mrs. Rochel Dobkin, who was 5ft. 1" in height, and was 47 years old with dark brown hair growing grey. She went out to meet her husband Harry Dobkin, on 11 Sep. 1941, and was discovered fifteen months after date. The husband, in whose presence the body was discovered fifteen months after burial was charged with murder, was found guilty and sentenced to death.[8]

The Acid-bath Murder

In the early afternoon of 18 Feb. 1949, one John George Haigh went with Mrs Olive Durand-Deacon, a well-to-do widow to Crawley in his car from the Onslow Court hotel in Kensington.

On reaching there he took her into a store-shed at Leopold Road and shot her through the back of the head while she was looking at some plastic. He removed her Persian lamb coat and jewellary, and then put her fully clothed into a 45 gallon steel tank, into which he transferred strong sulphuric acid by means of a stirrup pump from a carboy. Three days later he found some fat and bones floating in sludge in the tank. This he removed in a bucker which he emptied on ground opposite the shed entrance and then pumped some more acid into the tank to dispose of any remaining tissues. On the next day, finding that decomposition had occurred completely, he poured off the contents of the tank on the ground opposite the door. The plastic hand bag had not been affected by the acid.

On the first of March, the residual acid sludge and the debris lying on the ground were lifted carefully by shovel and removed inboxes to New Scotland Yard for laboratory examination and sleving. All this mass of grease and earth weighted about 475 lbs.

For three days spreading it out over steel trays on the laboratory bench patiently searched the dirty, partly yellow greasy, partly charred oily residue, and the following were recovered from the whole mass:

1. A mass of some 28 lbs of yellow greasy substance resembling melted body fat.

2. Three facetted gallstones of human type.
3. Part of a left foot eroded by acid.
4. Eighteen fragments of human bone, all eroded by acid to a varying degree.
5. Intact full upper and lower dentures
6. The handle of a red plastic handbag.
7. A lipstick container cap.

The eighteen fragments of partly eroded bone on further examination were identified as:

a. A left ankle pivot bone (talus).
b. A small part of the centre of the right foot, with attached ligamentous tissues.
c. A right of caleis (heel bone).
d. A right ankle pivot bone (talus).
e. Three lengths of eroded long bone cortex, probably the femur (thigh bone).
f. Parts of each pelvic (hip-girdle) bone.
g. A fragment of pelvic wing (hip crest).
h. A small piece of the lower spinal column, together with eroded parts of two intervertebral discs.
i. Seven further small fragments, far too eroded for exact anatomical definition.

All these residual fragments were eroded by a strongly acid fluid, and sharply distinguished, therefore, from certain animal bones, which showed no such changes though lying in the same oil surface. A London Dental Surgeon as having been supplied by her to Mrs Durand-Deacon in the year 1947 identified the intact dentures.

It was reasonable to draw the following inferences from this examination:

1. The gallstones were human type construction, and a positive precipitin test proved beyond doubt that they were human.
2. Of the nineteen parts of tissue listed above, the foot was so obviously human as to be plainly so to a lay person; a plastic cast was made, and this enabled some comparison to be made between it and the left shoe of the suspected victim. The remaining very small fragments except seven were human as judged by their mere anatomy.
3. No evidence of more than one body was forthcoming from the remains.
4. Indication of female sex was derived from a preauricular groove seen on part of an os innominatum.
5. The somewhat fragile state of the bones and the presence of osteoarthritis in certain joints indicated late adult age.
6. The dentures were sufficient to place identity beyond all possible doubt.
7. The remains gave no evidence as to the cause of death.
8. Immersion in concentrated sulphuric acid would under certain circumstances, result in as extensive a destruction of the body as indicated by the remains within a period of several days. The body fat, gallstones, and certain plastic substances like dentures would resist this erosion.

In addition to the above, a group of very finely spattered human bloodstains were found on the white washed wall of the store-shed.

Haigh was charged with the willful murder of Mrs Durand-Deacon and was tried at the Sussex Assizes at Lewes. A plea of insanity was raised in defence. To prove this it was mentioned that after shooting his victim the accused made an incision into the side of her throat with a penknife and collected a glass of blood, which he drank. He was also in the habit of drinking urine. The jury, however, found the accused guilty and was hanged at Wandsworth.[9]

EXAMINATION OF DECOMPOSED BODIES

The autopsy examination of a decomposed body should be complete and be conducted like a routine autopsy. On a decomposed cadaver, a pair of steel hooks with bent, adequately long handles are very convenient for hooking up the abdominal and other incisions so as to keep the parts open and also for opening the pericardium and hooking up the heart, lungs and other organs.[10]

In a highly decomposed body, even after 5-6 days of death with skin peeled of epidermis, in violent asphyxial cases of strangulation and hanging, the ligature mark would be apparent. Body in advanced putrefaction, the presence of mud in the bronchus is diagnostic of death due to drowning. Valuable clue to the cause of death may be derived by presence of foreign bodies, such as a bullet, a piece of a weapon or some other objects in a decomposed body.

In fracture skull bones, presence of clotted blood may be found on their inner plates, or on the surface of the dura mater and on the brain, would not be enough to give an opinion that the fracture was caused before death. In all doubtful cases, it is better to give a guarded opinion that the injuries found on the body, if inflicted during the life, were sufficient enough to cause death. In cases where the cause of death cannot be found owing to advanced decomposition, necessary viscera should always be preserved for chemical analysis.

POSTMORTEM DESTRUCTION BY PREDATORS

Predation by animals is a part of the natural food chain. Jackals, dogs, crows, ants, flies and maggots, etc. are well known for badly damaging the dead body or carrying their parts. Predation varies greatly with geography, season and whether the dead body is indoor or outdoor. Lying open on the countryside, large predators can cause severe damage. If the dead body is in water (river/sea water) then damage by normal inhabitants of water is usual.

Damage by the canines and rodents is obvious as the impressions of typical teeth-marks are usually observed at the sites of localised loss of flesh. Edges of such wounds appear nibbled or crenated, and are of postmortem origin, with no haemorrhage or the inflammatory reaction.

Maggots, the larval stage of common house and blowflies are the most efficient tissue removers. These lay eggs over the body natural orifices and ulcerated areas. This is because these areas are usually moist and shaded, lowering the risk of desiccation to the eggs. These eggs then hatch into larvae or maggots. First attacking the natural orifices, they burrow into the tissues and invade the cavities too. They secrete proteolytic enzymes which helps in softening the tissues and making their way deep into the interior of the body. This also helps in easy access to the external microorganisms.

Ants and insects mostly attack the exposed parts and the moist areas of the body, such as around the eyelids, lips, axilla, groin and on the knuckles. Superficial ulcers with scalloped, serpiginous margins characterize the lesions. Cockroaches are common in the residential setting. They are omnivorous scavengers having predilection for devouring keratin. Postmortem insect bites may become desiccated giving appearance of brush-burns. Confusion may also exist because of the site, i.e. superficial abrasions at the neck region may simulate nail abrasions produced in the course of manual strangulation. Lack of hemorrhage, inflammatory reaction features of margins as described, make them easily distinguishable.

In water, all kinds of aquatic animals may be involved and if the corpse is lying in shallow water or on the bank of a pond or on a sea beach, land animals also attack the body.

ARTEFACTS (SYNONYM: ARTIFACT)

Any change or alteration, which is man-made as a whole or introduced in the natural state of the body likely to be misinterpreted at autopsy constitutes an artefact. Such artefact may be introduced before death, at the time of death or after the death and, labelled as:

- Therapeutic artefacts
- Agonal artefacts
- Postmortem artefacts.

Therapeutic Artefacts

The task of performing autopsy may sometimes become difficult in cases where the victim has sustained serious injuries and has survived for a fairly long time, undergoing surgical and other treatment, likely to affect the interpretation of findings at the time of conducting autopsy, if the autopsy surgeon is not conversant with their origin and significance. This focuses the necessity of going through all the records of the antemortem treatment and if need be, a discussion with the doctors who attended the victim during hospitalisation. The following are a few examples:

- Vigorous external cardiac massage may result in fractures of the ribs and sternum. These frectures may lead to lung contusion or anterior mediastinal and pericardial contusions.
- Automated pneumatic cuff used for blood pressure monitoring placed around upper arm can cause petechial ecchymoses, compartment syndrome from an intramuscular haematoma.[11]
- Injection marks against the cardiac region and ring-like bruising caused by a defibrillator, may be the other sources of confusion.
- Administration of fluids or multiple blood transfusions may introduce changes in the blood alcohol concentration or concentrations of other toxic agents.
- Shape and size of the injury/injuries may be altered by the surgical intervention. The appearance of entrance and/or exit wounds may be distorted by surgical interference or during washing/cleaning the wounds. Bullet or pellet(s) may drop out unnoticed while removing clothing in the Emergency Wing. Similarly, it may happen in the operating rooms too.
- Changes intervene in the injuries with the passage of time in the form of healing or becoming septic, etc.

Agonal Artefacts

Absence of appreciable hemorrhage does not necessarily indicate its postmortem origin nor does the presence of extravasated blood into the tissues always suggests its antemortem origin. During the terminal moments of life, the victim may pass rapidly into vascular collapse or shock, which may prevent any significant bleeding to occur. An individual may collapse and die along the roadside and may subsequently be run over by some vehicle, leading on to collection of blood in the body cavities and some into the tissues too. Agonal spillage of the gastric contents into the respiratory passage has been discussed under, 'Sudden and Unexpected Deaths'.

Postmortem Artefacts

Postmortem artefacts imply any alternation, modification, addition or substraction of some postmortem features due to certain factors originating after death. These may be classified as:

- Artefacts induced by transportation/storage/handlings, etc.
- Artefacts induced by embalming, decomposition, etc.
- Artefacts induced by predators or deliberate mutilation/dismemberment by the criminals.
- Artefacts induced by improper autopsy procedures.

Artefacts Induced by Transportation/Storage/Handling, etc.

- Postmortem lividity is usually purplish in appearance. However, this lividity appears pinkish in refrigerated bodies or bodies exposed to cold environment.
- Postmortem collection of blood in the occipital region is due to bumping of head.
- Protruding areas of the body may get abraded due to dragging of the body.
- Rigor mortis may be broken during lifting or handling of the bodies giving wrong clues towards time since death.
- Rarely, fractures of the long bones may be caused particularly in debilitated, elderly dead bodies with osteoporotic changes.
- During transportation, dead body may be contaminated with dirt, soil, grease, etc. This may give wrong impression about the place of occurrence of death.
- Tearing of the clothing during transportation may appear to be due to some scuffle during life.
- Attempt to remove ornaments from the body parts like nose, ears, neck, etc. may cause injury to these parts and may send wrong impressions.

Artefacts Induced by Embalming, Decomposition, etc.

- The embalming technician may pass trocar in any of the wounds already present upon the body or may make a fresh cut. Embalming fluid used may pose problems in chemical analysis of viscera.
- Decomposition of the body may lead to production of most common artefacts i.e.
  - Bloating, discolouration and blistering of a body may be mistaken for disease or injury. Dark bluish discolourction areas may be mistaken for bruising.
  - Distension of parts of the body having loose tissues like tips, eyelids, breasts, penile and scrotal regions and protrusions of tongue, may impart false sense of obesity.
  - Expulsion of blood-tinged fluid from the mouth and nose may be mistaken as bleeding originating during life.
  - If the deceased were wearing tight clothing or having a neck-tie, a groove may appear around the neck and this along with bulging of the eyes and protrusion of the tongue may be mistaken for strangulation.
  - Fissures or splits formed in the skin during decomposition may simulate incisions or lacerations.
  - Expulsion of semen or vaginal discharge due to pressure of putrefying gases may wrongly suggest involvement of sexual activities with the cause of death.
  - Marked bluish discoloration of the loops of intestines especially in the pelvic cavity may not be mistaken for infracted bowel.
  - Autolytic rupture of stomach can occur postmortem with release of the stomach-contents into the peritoneal cavity.
  - Pancreas too, may undergo autolysis due to proteolytic enzymes within it. On autolysis this organ may appear hemorrhagic and be mistaken for pancreatitis. However, histology will be helpful in resolving the tissue.

- Separation of skull sutures or bursting of abdormen with advanced putrefaction, may be mistaken for trauma.
- Putrefactive gas in the right side of the heart may be mistaken for air embolism.
- Small round holes due to maggots may be mistaken for bullet/pellet holes.

Artefacts Induced by Predators or Deliberate Mutilation/Dismemberment by the Criminals

Common terrestrial creatures attacking the dead body in and around the mortuary are rats, rodents, ants, cockroaches and crows, etc. Ants, insects mostly attack the exposed parts and moist areas such as face, arms, genitals, groins and axilla, etc. Rats, cats and dogs usually destroy the soft tissues of the exposed parts. All these are devoid of evidence of hemorrhage and vital reaction and their edges appear nibbled. Bodies recovered from jungle or open space, may be attacked by dogs, cats, vultures or jungle-animals and the bodies recovered from water may show gnawing by fish, crabs and other aquatic animals. Flies, maggots, larvae, etc may alter the wounds.

Sometimes, the criminals for easy disposal and removal may do mutilation or dismemberment of the corpse from the scene of crime. Injuries may also be inflicted after death merely to mislead investigations. Often, persons may be killed and thrown in water or the dead-body may be put on fire. Occasionally, the victim may be poisoned and hanged after death and so on. This has substantially been discussed under appropriate chapters.

Artifacts Induced by Improper Autopsy Procedures

- In usual practice, the vault of the skull is sawn and then removed gently by inserting and twisting the chisel at various places through the gap generated by sawing. Any vigorous sawing or using chisel and hammer may result in extension of the existing fractures or fresh fractures may be caused.
- In deaths due to compression of the neck, it is preferable to open the skull first. The draining out of the blood from the neck vessels due to prior removal of skull and brain, provides a clearer view for the study of the neck structures and will avoid occurrence of congestive-artifactual hemorrhages in the neck structures as cautioned by Prinsloo and Gordon.
- When the neck structures are pulled forcefully or improperly, air may enter the vessels of the neck or there may be seepage of blood into the tissues or there may occur fracture of the hyoid bone especially in the elderly.
- During removal of the sternum, damage to the heart or internal mammary vessels may lead to seepage of blood in the pleural or pericardial cavities.
- While abdomen or peritoneum is being cut open, coils of intestines may get involved.
- Improper pulling apart of the liver may cause tears in the diaphragm and denudation and laceration of the bare area of the liver.
- Collection of viscera in a single container or use of contaminated dirty bottles/jars or preservatives may result in wrong conclusions for visceral analysis.

Heat Effects

When the body is exposed to intense heat, the skin becomes tense, leathery, and hard and frequently exhibits splits which may be mistaken for wounds. Heat fractures may also be encountered. In conflagrations, when the head has been exposed to intense heat, scalp may show splits and the skullcap may present fissured fractures, which may be mistaken for fractures due to trauma. Furthermore, 'heat hematoma' within the burnt skull can resemble an extradural hemorrhage of antemortem origin. The frothy brown appearance of the false clot along with heating effects upon the adjacent brain, help in differentiation. The details have excellently been discussed under 'Thermal Deaths'.

CASE—Dead Bodies Recovered from Railways Track—Appreciation of Artefacts

During mid-February, 1998, two dead bodies were allegedly recovered by the police from the side of the railway-track on the information furnished by the watchman on duty and were transported for the postmortem examination.

The autopsy showed the faces of both the dead bodies as swollen and suffused obviously owing to their slanting position in which the bodies were lying alongside the railway-track with the heads at the lower level than the rest of the bodies. One of the bodies was also presenting some diffuse punctate haemorrhages distributed along the lower part of the neck and the adjoining inner two-thirds of the area against the clavicular regions. This, along with slightly protruded tongue and suffused face, initially conveyed erroneous impression of something concerned with some assault involving neck. But such appearance are usually artefacts as a result of prolonged posture in which the body was lying (probably for the entire night), coupled with round-necked clothing which the deceased was wearing, namely: a jacket with a central zipper, blue-coloured full-sleeve round-necked T-shirts, black-coloured full-sleeve round-necked warm vest.

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CARE IN THE POSTMORTEM ROOM

- The room where the postmortem examination is carried out and the annexe should be washed and cleaned regularly, in view of the high-risk autopsies like HIV, Hepatitis B, etc.
- Cleaning with bleaching powder (10%) solution after autopsy helps in removing the HIV virus.

CARE OF THE TECHNICAL STAFF

- Instruments should also be soaked in 10 per cent bleaching powder solution.
- Except while intentionally noting the smell, in the remaining period during the autopsy, the autopsy surgeon is advised to wear a cap, mask, plain goggles, gumboots, gloves reaching up to the elbows, and double gloves in all high risk cases.

- A chest X-ray once in six months,
- Tetanus toxoid every six months,
- Hepatitis – B immunisations.

EMBALMING

It may be required to keep the body embalmed to enable the relatives from far off places to have a better glimpse. It is done by injecting formalin solution 9 per cent by gravity or with the help of power-pump.

ENTOMOLOGY OF THE CADAVER AND THE POSTMORTEM INTERVAL

Study of insects or their larvae infesting a dead body as a means of ascertaining possible time since death, constitutes the subject of forensic entomology.[1] The application of entomology was first reported by Bergeret in 1855 and Megnin, an entomologist, who placed forensic entomology on a sound footing with his publication of *La Faune des Cadavers* in 1894.[2] It may be emphasised here the need for exercising caution while making any estimate of the time since death, i.e. it must be assumed that the eggs were deposited on the body at or nearing the time of death. Further, it is also important to identify the species, as the life cycle of each species varies. When the larvae are identified it is possible to determine the minimum postmortem interval based on the larval age, i.e. if it is estimated that the maggots on the corpse are four days old, the deceased could not have died with a postmortem interval of less than four days, although the body might have been dead for a longer period than that. Since blowflies usually arrive at the corpse and lay eggs on it within an hour or two after death (unless the body is buried or placed in a sealed bag or concealed in any other manner so as to prevent access to the flies) the minimum time of death-estimate may effectively be the actual time of death.[2]

Admittedly therefore, it is the field of the 'specialist' when the issue of time since death is involved in the criminal investigations and it is essential, wherever practicable, the forensic entomologist should attend the scene and collect the material. It also enables him to study the environment. Obviously, no attempt will be made here to deal with the identification of various species and their life-cycles but the doctor engaged in the medicolegal work must be aware of the technique of collecting and dispatching samples for examination by the forensic entomologist, demands the knowledge of collection, preservation and dispatch of the specimens.[2]

Collection, Preservation and Dispatch of the Specimens

The main concern of the doctor, conducting the medicolegal postmortems, is to do the careful collection, preservation and dispatch of specimens to the FSL or forensic entomologist. Different steps are presented and discussed below:

- Maggots (including different stages, i.e. mature, immature, pupae, empty pupa cases and eggs, etc.) should be placed in separate tubes and placed directly in acetic alcohol (three parts 70% alcohol and one part glacial acetic acid). If no preservative is available, killing of the specimens may be done by putting them in hot water.[2,3]
- Some 'live' larvae should also be collected and placed in a tube with a fragment of meat or a portion of muscle from the body, acting as a food for the maggots. Larvae should be taken from different parts of the body and proper labelling be done on the tubes regarding. This large sampling makes it possible to interpret the population of maggots more confidently.[2]
- A sample of the soil beneath the body along with the various stages of maggots should also be sent.
- Available facts about the environment in which the body was found also be recorded and sent to the entomologist. Recording of temperature of the body and ambient temperature of the scene must be recorded and dispatched. If recording of the temperature has been not done, local records from the nearest meteorological centre may be called for.[2,3]

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PSYCHOLOGICAL AUTOPSY

Introduction

Psychological autopsy is a recently developed postmortem investigative tool that aids in the determination of a person's cause of death. Often there are many questions left unanswered when a person dies in a mysterious manner. In such cases, scientists, investigating officers, and psychologists can help to uncover hidden secrets deriving justice. Here interviews, eyewitness reports, and medical records are used to determine a manner of death: natural, accidental, suicidal or homicidal.[1]

Psychological autopsy is used most often in cases of suspected suicide or homicide. It has definite role in criminal investigations. In 1961, two psychologists named Shneidman and Farberow developed the psychological autopsy procedure which is often used presently in some of the developed countries. Here about 16 categories are identified for possible inclusion in this process and enumarated and described below.[2]

- Identifying particulars (name, age, sex, address, marital status, occupation, religion).
- Details of death (police records).
- History (siblings, illnesses and treatments, suicide attempts, etc).
- Death history of deceased's family members, if any.
- Patterns of stress reaction.
- Recent tensions or confrontations.
- Role of alcohol and/or drugs in the overall lifestyle and death of the deceased.
- Interpersonal relationships.
- Fantasies of the deceased.
- Dreams of the deceased (or nightmares).
- Thoughts and fears of the deceased relating to death, accident or suicide.
- Change in habits, hobbies, eating, sexual patterns or other life routines just prior to death.
- Information relating to the "life side" of the deceased (upswings, successes, inspirations).
- Assessment of intention (role of the deceased in his/her own demise).
- Rating of lethality reaction of informants to deceased's death.
- Comments and special features.

However, one of the major weaknesses of psychological autopsy is lack of standardised procedure.[3] Psychologists have developed recently, a guide with 26 categories to help investigators. Here it is important to remember that this is only to assist investigators and that it may not be applicable in all categories in every case[1] and are enumerated as follows:

- Alcohol history.
- Suicide notes.
- Writing/diaries.
- Books.
- Relationship assessments on the day before death.
- Marital assessments.
- Mood.
- Psychosocial stressors.
- Pre-suicidal behaviours.
- Language.
- Drug history.
- Medical history.
- Reflective mental status exam of deceased's condition before death
- Psychological history.
- Laboratory studies.
- Coroner's Magistrate's report.
- Motive assessment.
- Reconstruction of events.
- Assess feelings regarding death as well as preoccupations and fantasies.
- Military history.
- Death history of family.
- Family history.
- Employment history.
- Educational history.
- Deceased's familiarity with methods of death.
- Police reports.

Group of investigators who perform a psychological autopsy are called Death Investigation Team (DIT).[4] DIT's most important task is the interview process and that there are two specific types of interviews:[1]

I. *Interviews with eyewitnesses:* These interviews are usually conducted with persons who witnessed the actual death event and/or persons who found the deceased's body.
II. *Interview with character witnesses:* These are persons who were related to, or acquainted with, the deceased in some or other way, such as family, friends and co-workers.[4] One specific type of interview is called the Structured Interview of Reported Symptoms (SIRS), which can help detect malingering.[5]
III. *Physical autopsy of corpse:* According to psychologists there is a third interview, which involves a physical autopsy of the corpse. Quite often, the dead will reveal hidden secrets about themselves or others of which no witnesses were aware (such as substance dependence, cancer or abuse).

Applicability of Psychological Autopsy

As mentioned earlier, psychological autopsy is mostly used in murder and suicide investigations.

Murder investigations—The results can reveal vital information about why the victim was chosen by the killer. This may help to detect similarities between different crimes committed at different times, possibly enabling detectives to pinpoint a criminal's signature.[6]

Suicide investigations—Psychological autopsies are mostly indicated in cases of suspected suicide. This is sometimes the only way to shed light on the characteristics of suicide victims, and to identify the possible warning signs that may have been present prior to their self-demise. European psychological autopsy studies have employed the use of case-control designs to better estimate the role of various risk factors for suicide. These studies document suicide mortalities and reducing the frequency of these tragic incidents.[7]

The procedures of psychological autopsy in suicide cases are aimed at the production of *four outcomes:*

1. To establish a method of death.
2. To determine why the deceased chose to take his or her own life at that specific time and place.
3. To learn better ways to predict suicidal tendencies and treat people before their behavior reaches harmful levels.
4. To provide the friends and family of the deceased with explanations for the loss of their loved one, which can serves as a therapy for their suffering and grief.[8,9]

Results of a thorough psychological autopsy may suggest that the deceased did not commit suicide but, in fact, was murdered or may have died of natural causes.

Use or abuse of alcohol or controlled substances might be a misconception. Often its difficult to assess whether an overdose was self-induced or accidental. Psychologists usually probe for a specific interpersonal loss and/or conflict up to three years prior to death. Usually this is referred to as a *life event* and is often perceived as the major reason for the loss of hope that causes suicidal actions. Job loss and divorce are some of the examples of negative life events.

Investigations at times might also reveal the presence of mental illness or dementia, which could be the cause of confusion about certain life events. It is reported in a study of 57 adult suicide victims, 30 were diagnosed with alcoholism and 27 were diagnosed with mood or anxiety disorders.[10] In an another study, it is reported that 16 out of 18 suicide victims in later life (50 to 92 years old) had diagnosable psychopathology, of late onset major depression.[11]

In a community-based psychological autopsy study involved 73 cases of elderly suicide and addressed the issue of first-attempt fatal suicide in elderly individuals, claimed that most of the elderly persons, who choose suicide possessed a "life-long character fault," which remains unseen until aging forces the issue to surface.[12] The elderly population has the highest suicide rate in the United States and that they are also more likely to be successful in their attempts than their younger counterparts.[13]

After the elderly, adolescents age group has the second highest suicide rate in this country. Reports are there which claims that depression can occur in as young as puberty, but there are cases documented of suicides committed by children as young as three years old.[14] However, it is believed that the suicides of many children are concealed and it is most likely that the suicide mortality for youth and adolescents is actually higher than recorded.[15,16]

Studies have also shown that, unlike in adults, there is no significant difference between adolescent suicide victims who have and do not have diagnosed psychiatric disorders. Some of the disorders that are often seen in adolescent suicide victims are major depression, bipolar disorder, substance abuse/dependence and conduct disorder. Homicidal ideation is also quite common among these troubled youths.[17,18] It may be remembered here that the most important aspect of psychology is the integrity of the patient and in psychological autopsy, the patient is the deceased. Always all facts and data to be handled with the utmost care and respect. However, three points deserve particular attention here: integrity of the deceased, respect of the interviewees and health of the investigators. One should also be aware of the fact that unless there is a court order, all witnesses are entitled to refuse interviews. Psychological autopsies also has a potential problem that all who participate may contaminate the results of this delicate process needing proper caution and security on data colleted.

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AUTOPSY ON A BODY OF HIV INFECTION

Identifying and isolating HIV infected bodies is though controversial among the pathologists, there lies no difference of opinion regarding precautions to be taken, while conducting an autopsy on a body suspected to have been infected with HIV.

In India good number of bodies will be autopsied by forensic pathologists not being very sure of its HIV status.[1] It is extremely difficult to isolate cases, which are in window period as ELISA test will be negative. ELISA test will also be negative in a full-blown case of AIDS. Under such circumstances detection is only by a viral culture. Since the forensic pathologist cannot afford such lapse of time he/she has to depend on brief clinical history,[2] and clinical conditions or findings[3] which should give a suspicion of HIV infection, are enumerated below:

1. *Clinical history:*
   - Persistent low-grade fever with malaise, arthralgia of more than one month duration.
   - Weight loss, progressive type of greater than 20 per cent.
   - Persistent diarrhoea of more than one month duration.
   - Persistent gastrointestinal tract infections like
     i. CMV
     ii. *Giardia*
     iii. *Cryptosporidia*
     iv. *Isospora.*
   - Recurrent vaginitis/cystitis.
   - Skin rashes non-response to general treatment.
   - Oral candidiasis, dysphagia, odynophagia (Severe pain on swallowing due to a disorder of the oesophagus).
   - Jaundice with progressively deteriorating liver function.
   - Gullian Barrie syndrome.
   - Rieters syndrome with bells palsy.
   - CMV induced Retinitis.
   - Intense psoriasis and psoriatic arthritis.
   - Hansen's disease nonresponsive to general treatment.
   - Dead bodies of children of HIV positive mother who were breast fed during the probable window period.
   - Dead bodies whose spouse is suspected to be suffering from HIV infection.
   - Pulmonary and military tuberculosis non responsive to triple drug regime.
   - Systemic herpes zoster infection.
   - IT purpura.
   - Recurrent *H Ducreyil* and *treponema* infections
2. *Clinical conditions or findings:*
   Dead bodies with following can give clues on HIV infection:
   - Anorectal diseases.
   - Gingivitis and seborroic dermatitis.
   - *Pneumocystis carinii* infections.
   - Hairy leukoplakia over tongue.
   - Polymyositis
   - Generalised lymphadenopathy not secondary to Koch's/Hodgkin's.
   - Hypodensities in white matter
   - Multiple ring lesions (demonstrated by CT scan)
   - Cryptococcal/tubercular meningitis.
   - CSF abnormalities like raised protein/cell count/IgG.
   - Kaposi sarcoma
   - Anal squamous cell carcinoma
   - Nonmelanomatous skin cancers

Hence a little extratime spent, before cutting open a dead body, over history taking and detailed examination to look for the signs enlisted above, shall avoid the forensic pathologist getting infected with the deadly virus.

It is true that HIV virus cannot enter an intact skin, but can enter through an intact membrane like conjunctive, mucous membranes of oral and nasal cavities. Hence, additional precautions may be adopted with routine precautionary measures to avoid an accidental occupational exposure and are listed below:

Additional Precautions[4]

- Wearing head cover and face mask.
- Wearing protective goggles.

- Wearing protective disposable gown.
- Wearing double gloves for hands.
- Wearing thimbles for left hand fingers (to avoid needle prick)
- Incinerating all the protective disposable wears.
- Preserving needles and thimbles in 70 per cent alcohol for 30 min. before reusing them.
- Autoclaving the instruments before reusing them (since the virus is thermolabile).
- Getting the autopsy room washed with household bleach.
- Advising the relatives of the deceased to incinerate/cremate the dead body.
- If the body is to be buried (as in cases of unidentified dead bodies) embalming them with 0.1 per cent formalin to reduces the risk during exhumations.

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Chapter

15

Violent Asphyxial Death

LEARNING OBJECTIVES

- Definition of Asphyxia
- Etiology of Asphyxia
- Signs and Symptoms of Asphyxia
- Pathophysiology of Asphyxia
- Mechanical/Violent Asphyxial Death
- Define and describe the postmortem findings and the medicolegal aspects of:
  - Hanging
  - Strangulation
  - Suffocation
  - Drowning.

INTRODUCTION

Deaths due to asphyxia are caused by failure of cells to receive or utilize oxygen. The deprivation of oxygen may be partial (*hypoxia/ suboxia)* or total *(anoxia/anoxemia)*.[1] Literal meaning of word 'asphyxia' is *'no pulse'* (pulse less). It is also understood as a 'state of low oxygen' (*hypoxia/suboxia, anoxia,* etc). Thus the terminologies *anoxia, anoxemia, suboxia, hypoxia, etc.* are though considered as better ones, the terminology "asphyxia" remains accepted in true medicolegal sense globally. However, the pathophysiology would include biochemical and neurological mechanisms.

DEFINITION OF ASPHYXIA

Asphyxia is defined as lack of oxygen in blood and tissues due to impaired or absence of exchange of oxygen and carbon dioxide on a ventilatory basis, leading to death.[2]

ETIOLOGY OF ASPHYXIA

Asphyxia can develop because of several causes such as:

- Physical causes—breathing in high altitudes with rarefied/ vitiated medium, deficient of O_2, e.g. mountaineering.
- Inhaling irrespirable gases, such as gases interfering with normal O_2 carrying capacity of blood haemoglobin, e.g. CO, CO_2, H_2S, etc. or gases which prevent cellular oxidation enzymes, e.g. HCN.
- Drugs/poisons—directly depressing the respiratory centres, e.g. narcotics (morphine), anaesthetics, etc.
- Mechanical asphyxia—this is due to mechanical interference to respiration, e.g. hanging, strangulation, throttling, smothering, choking, drowning, etc (Fig. 15.1A).
- Pathological asphyxia—this is due to diseases, e.g. lung pathology.
- Allergic reactions causing laryngeal oedema.

PATHOPHYSIOLOGY OF ASPHYXIA

Conventionally, the term *asphyxia* has been applied to all conditions in which oxygen supply to blood and tissue has been reduced appreciably below the normal working level by any interference with respiration. In death from asphyxia, usually it falls below the minimum necessary for continuance of life. Serious deprivation of oxygen for 5 to 10 minutes can result in permanent damage to central nervous system and cardiovascular system resulting in death. It is interesting to note that in a healthy adult body, normally blood that is circulating has about 1 litre of oxygen held in combination. Any interference with this results in asphyxia, which in turn triggers the consequences. This renders the pathophysiology of asphyxia, depicted in Figure 15.1B in a cyclical form and is nonstopping and continuous event till death of the person; hence it is a vicious cycle.[3-6]

ANOXIA

According to physiological concepts, asphyxia can lead to *lack of oxygen* supply to cells and tissues. Among all, nervous tissues are affected first by oxygen deficiency. This lack *of oxygen supply* is known as *anoxia.*

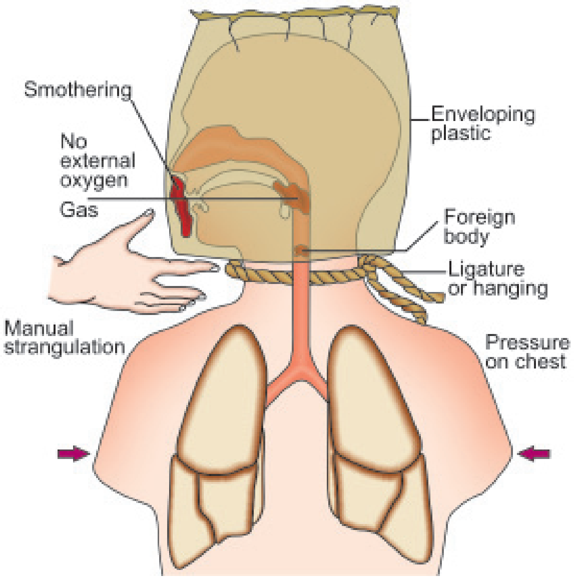


Fig. 15.1A: Causes of mechanical asphyxia

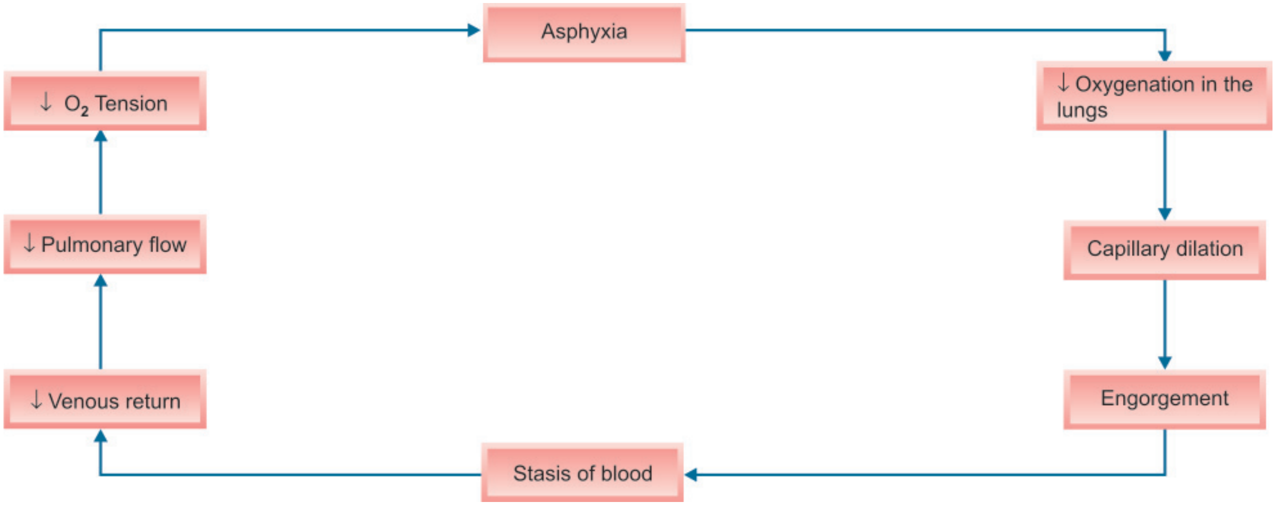


Fig. 15.1B: Pathophysiology of asphyxia

Gordon's Classification of Anoxia

According to Gordon,[3-5] anoxia is classified as follows:

- *Anoxic Anoxia*: Here oxygen cannot gain entry into the blood, e.g. hanging with obstruction of trachea—here no oxygen enters the pulmonary circulation, hence, there is no oxygen in the blood.
- *Anemic Anoxia*: Here oxygen can get into blood, but the blood is incapable of carrying it, e.g. carbon monoxide poisoning, wherein the carboxyhemoglobin formed prevents blood oxygenation.
- *Stagnant Anoxia*: Here oxygen can get into the blood, blood is capable of carrying it, but circulation is at failure, e.g. congestive cardiac failure.
- *Histotoxic Anoxia*: Here oxygen can get into the blood, blood is capable of carrying it, and the blood circulation is perfectly normal, but the cell cannot utilize the oxygen available in blood, e.g. cyanide poisoning, here the cyanides destroy the cellular oxygen enzyme system cytochrome *oxidases*. Histotoxic anoxia can be further classified as follows:

  Extracellular histotoxic anoxia – Here oxygen cannot be taken up due to the failure of tissue enzyme system by poisoning, e.g. cyanide, overdose of hypnotics or anaesthetics.

  Pericellular histotoxic anoxia – Here oxygen cannot be taken up due to reduced permeability of cell membrane, e.g. lipoid soluble anaesthetic agents such as chloroform, halothane, etc.

  Substrate histotoxic anoxia – Here oxygen cannot be taken up due to failure of efficient cell metabolism, e.g. hypogylcaemia, etc.

  Metabolite histotoxic anoxia – Here oxygen cannot be taken up due to the accumulation of *end products* of cell respiration, e.g. carbon dioxide poisoning, uremia, etc.

CLINICAL EFFECTS OF ASPHYXIA

A schematic representation of clinical effects of asphyxia may be presented as in Figure 15.2A.

CLASSIFICATION OF MECHANICAL/ VIOLENT ASPHYXIA

Interference of respiration by mechanical means constitutes mechanical or violent asphyxia. Mechanical asphyxia could be of following types depending upon the respiratory block (Fig. 15.2B).

1. *Compression/ constriction of the neck*, e.g. hanging, strangulation, throttling, etc.
2. *Blocking external orifices of respiration, i.e. mouth and/or nostrils,* e.g. smothering, overlying, suffocation, gagging, etc.
3. *Impaction of foreign bodies* in respiratory tract, e.g. choking
4. *Compression and mechanical fixation of the chest and abdomen* preventing the respiratory movements, e.g. traumatic asphyxia/crush asphyxia.
5. *Inhalation of fluid into the respiratory tract*, e.g. drowning.

HANGING

Definition

Hanging is defined as complete or partial suspension of the body by a ligature tied around the neck and force of constriction on the neck being applied by the weight of the body hanged.[3-6]

Causes of Death

In hanging, death is usually due to asphyxia or cerebral anoxia or vagal inhibition leading to cardiac arrest or injury to the spinal cord as observed in *judicial hanging* wherein death is due to fracture-dislocation of the $C_2C_3C_4$ vertebrae.[1,4,6]

Classification of Hanging[1-6]

- Complete hanging—both feet are not touching the ground (Fig. 15.3A and C).
- Partial hanging—both feet or any other parts of the body are touching the ground. Thus, it may be also induced in sitting, stooping, kneeling, lying prone or supine positions (Figs 15.3B and D to F).
- Typical hanging—knot of ligature is on the backside of the neck (Figs 15.4A and C).
- Atypical hanging—knot of the ligature is anywhere other than on the backside of the neck (Figs 15.4B and D to F). Usually location for the knot is near the mastoid process or angle of mandible. Occasionally, it may be under the chin (Fig. 15.4G).

Mechanism of Hanging

It has been scientifically accepted that pressure on the neck can result in occlusion of neck structures for respiratory functioning,

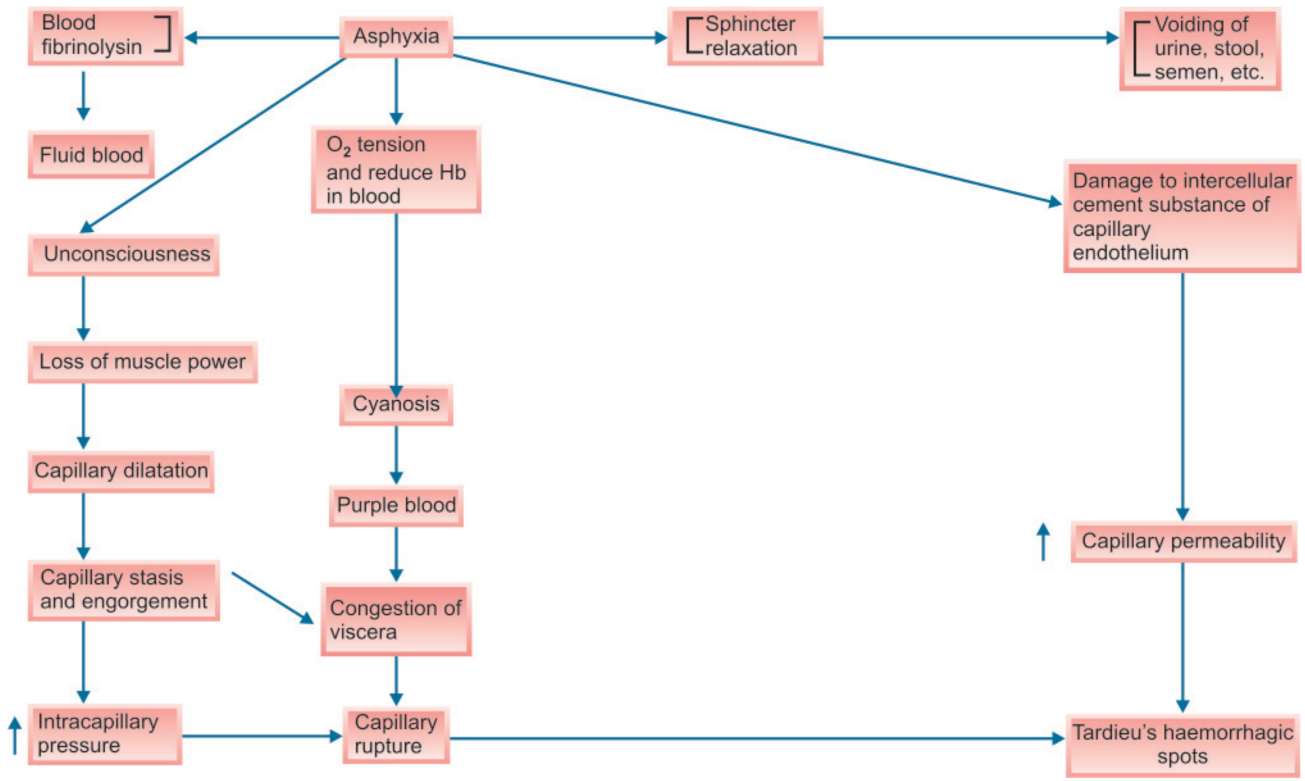

Blood fibrinolysin
Asphyxia
Sphincter relaxation
Voiding of urine, stool, semen, etc.
Fluid blood
O_2 tension and reduce Hb in blood
Damage to intercellular cement substance of capillary endothelium
Unconsciousness
Loss of muscle power
Cyanosis
Capillary dilatation
Purple blood
↑ Capillary permeability
Capillary stasis and engorgement
Congestion of viscera
↑ Intracapillary pressure
Capillary rupture
Tardieu's haemorrhagic spots

Fig. 15.2A: Clinical effects of asphyxia

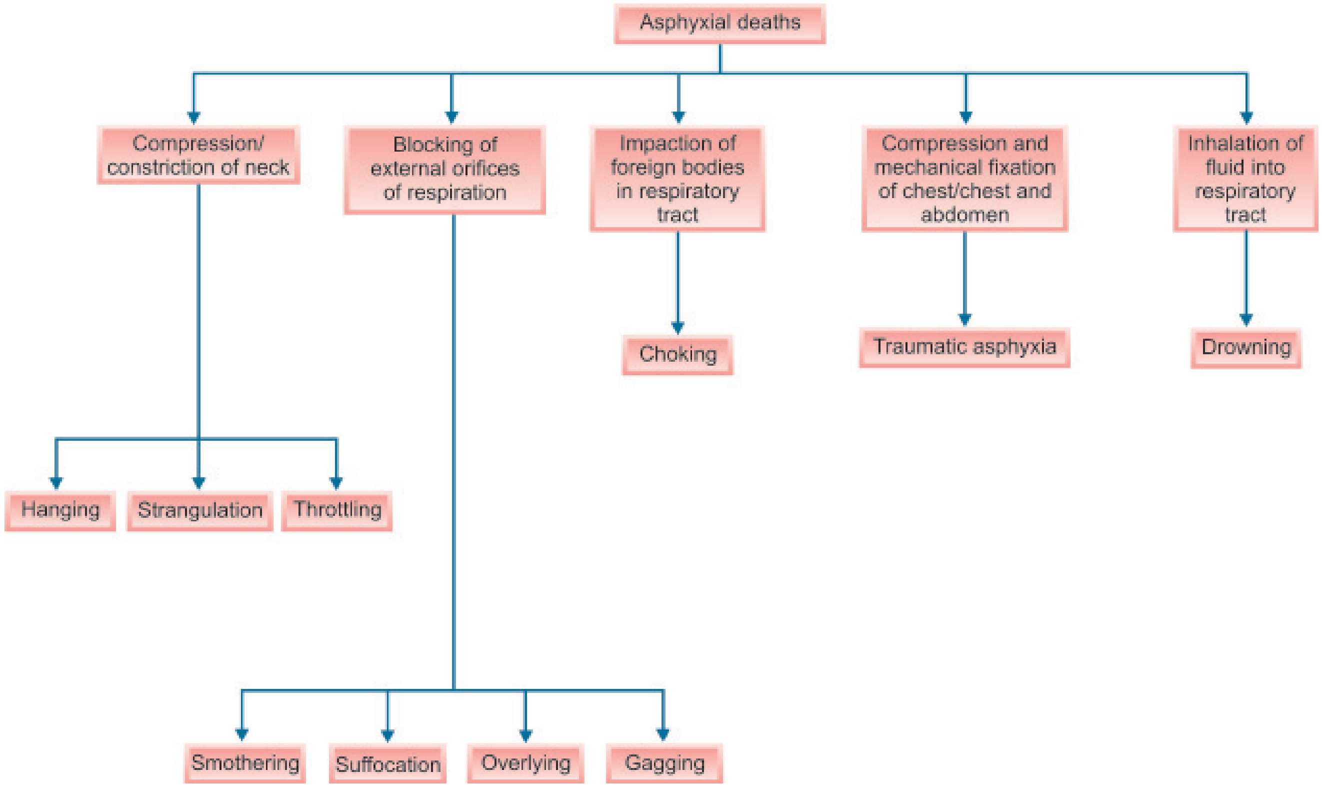

Asphyxial deaths
Compression/ constriction of neck
Blocking of external orifices of respiration
Impaction of foreign bodies in respiratory tract
Compression and mechanical fixation of chest/chest and abdomen
Inhalation of fluid into respiratory tract
Choking
Traumatic asphyxia
Drowning
Hanging
Strangulation
Throttling
Smothering
Suffocation
Overlying
Gagging

Fig. 15.2B: Types of mechanical asphyxial deaths

developing asphyxia. Experimentally, it has been proven that pressure/force of 2 kg (4.4 lbs) and 4-5 kg (8.8-11 lbs) on the neck can occlude jugular vein and carotid arteries respectively, 15 kg (33 lbs) can occlude trachea and 30 kg (66 lbs) can occlude vertebral arteries.[4,8] All these can bring about gross decrease in cerebral blood flow leading to cerebral anoxia, asphyxia and death.

AUTOPSY FINDINGS

Autopsy findings are discussed under two heads; *external* and *internal* findings.

EXTERNAL

All external findings are better sedlt under three heads, namely:

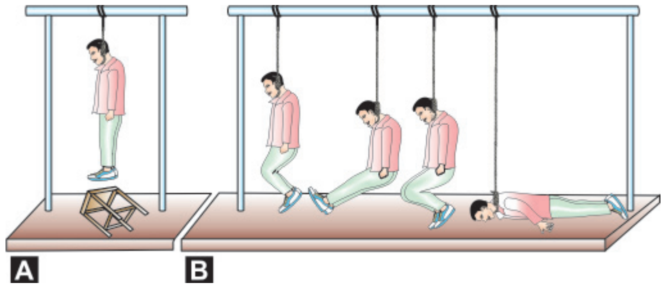
I. Findings in the face

II. Findings in the neck
III. Findings in other parts.

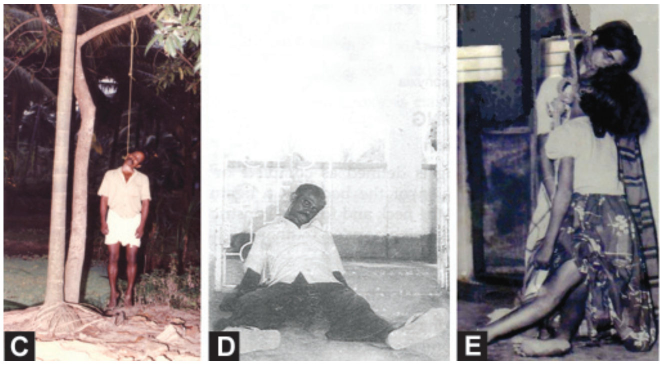
Each one of these is discussed separately with suitable illustrations.

I. Findings in the Face

Findings suggestive of *antemortem hanging* are illustrated in Figures 15.5A to E and discussed below:

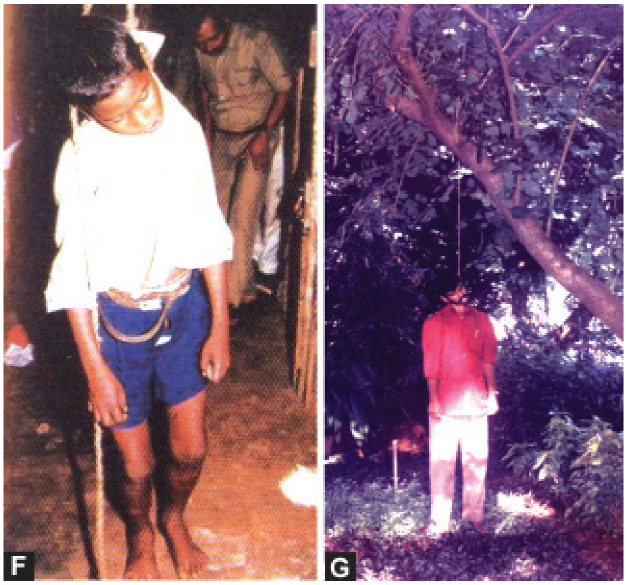


Figs 15.3A and B: *Types of hanging:* (A) Complete hanging; (B) Partial hanging

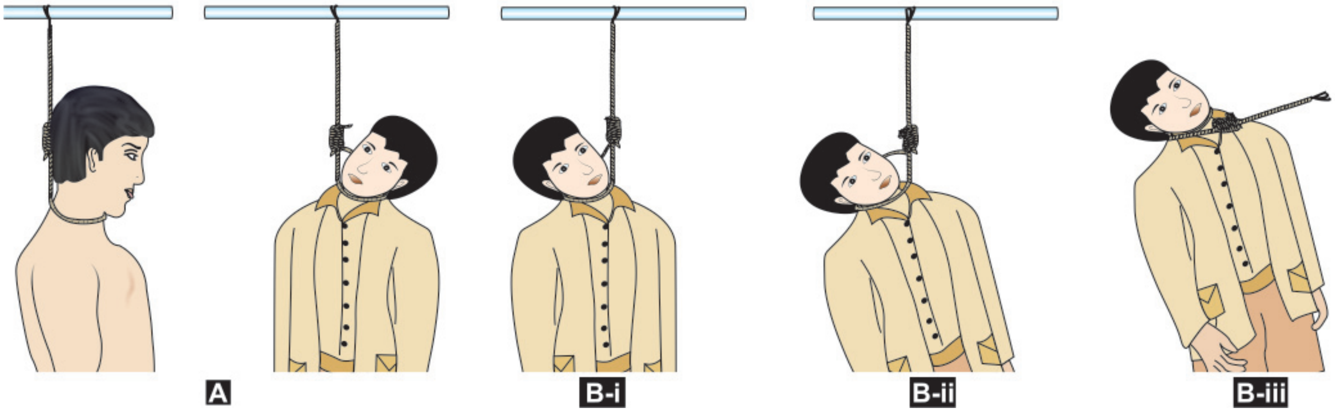


Figs 15.3C to E: *Types of hanging:* (C) Complete hanging: *(Atypical)* (*Courtesy:* Dr NG Revi, Professor & HOD, Police Surgeon, Dept of Forensic Medicine, Medical College, Trichur, Kerala; (D) Partial hanging in sitting position (*Courtesy:* Dr. SC Mestri, Professor and HOD, Dept of Forensic Medicine, JSS Medical College, Mysore, Karnataka); (E) Double suicidal hanging (mother and daughter): Partial hanging (*Courtesy:* Dr B Chandre Gowda, Formerly Professor and HOD, with permission of Dr Kiran J Head, Dept of Forensic Medicine, SDU Medical College, Kolara, Tamaka, Karnataka)

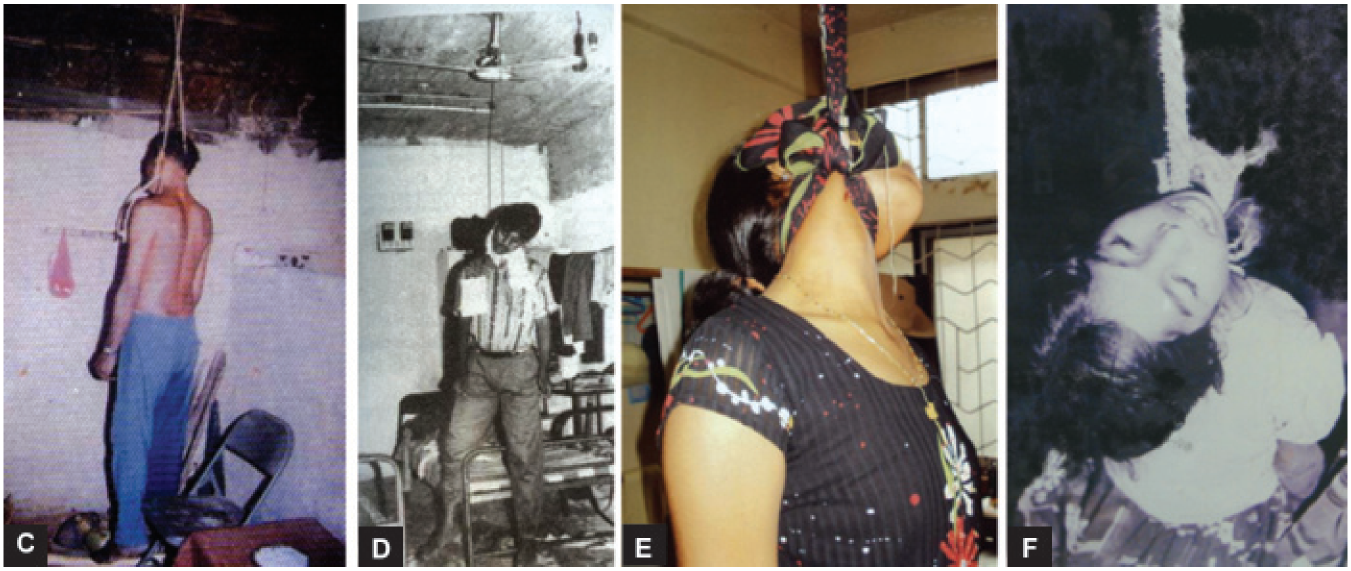
- ***Pallor/congestion*** – Usually in most hangings, the face is pale.[1] However it may be swollen and congested (Fig. 15.5B), with petechiae in partially suspended individuals, where the noose is tightened only by the weight of the head or the torso. In these instances while the carotid arteries and venous drainage are completely occluded, vertebral arteries still continue to supply blood to the head, producing accumulation of blood.[1,4,6] With time dependent accumulation of blood, punctuate hemorrhages and *Tarideu spots*, caused by hydrostatic rupture of vessels, will be seen on the forehead, over the eyelids, under the conjunctiva and sometimes near the temple (Fig. 15.5A).[1,9]
- ***Lips*** – may show cyanotic tinge (Fig. 15.5C).
- ***Changes in the eyes*** – Eyes may show following findings:
  - *Prominent eyeballs* – Eyeballs may look prominent due to increased pressure as a consequence of impaired circulation and venous return resulting in *passive accumulation of blood* (Fig. 15.5D).
  - *La-facie sympathique* – Etienne Martin (1950) described this peculiar change wherein right eye remaining open with dilated pupil and the left eye closed with small pupil.[8]



Figs 15.3F and G: *Types of hanging:* Suicidal hanging and partial hanging with feet on the ground (*Courtesy:* Manipal Police Station, Manipal)



Figs 15.4A and B: *Types of hanging:* (A) Typical and atypical hanging. (B) Possible ligature mark positions in the neck in hanging: (i) More common/usual position with fixed noose and high suspension. Mark rises high and may show a gap; (ii) When a slip knot is used it results into a smallest loop, that tightens maximum resulting into a deep ligature mark in the neck, could be low and horizontal; (iii). When the suspension point is low and victim leans away and mark will be horizontal, misleading for strangulation



Figs 15.4C to F: *Suicidal hangings:* (C) Typical hanging – Knot on the back side but high above; (D) Atypical hanging with suicidal note affixed on front of the shirt (*Courtesy:* Dr. Sreemathi Rajagopalan, St. John's Medical College, Bengaluru, Karnataka); (E) Atypical hanging (*Courtesy:* Dr Ritesh G Menezes, Assoc. Prof. KMC, Mangalore, Karnataka); (F) Atypical hanging – knot in the front (Dr B Chandre Gowda Formerly HOD, with permission of Dr Kiran J Head, Dept of Forensic Medicine, SDU Medical College, Kolara, Tamaka, Karnataka)

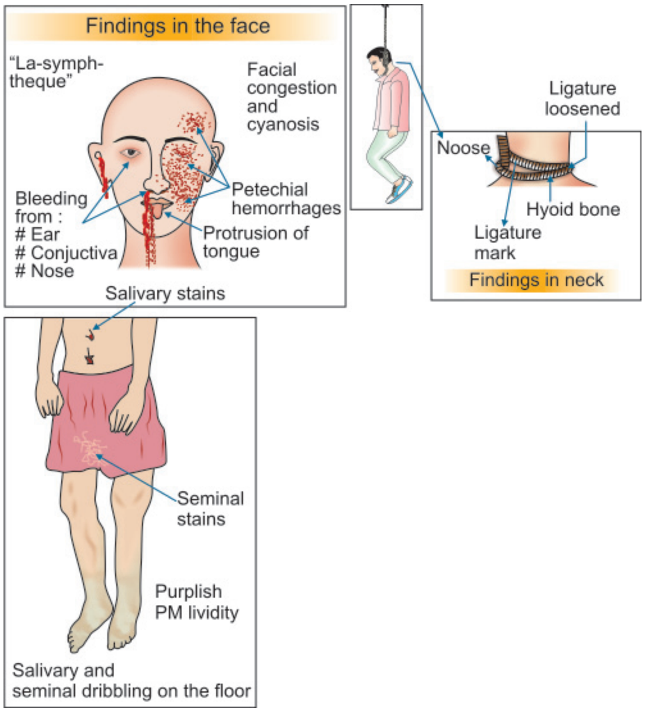
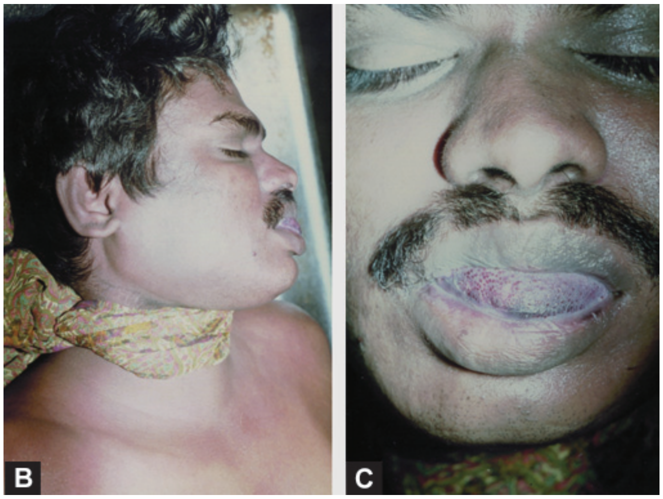

Findings in the face
"La-symph-theque"
Facial congestion and cyanosis
Bleeding from :
# Ear
# Conjuctiva
# Nose
Petechial hemorrhages
Protrusion of tongue
Salivary stains
Ligature loosened
Noose
Hyoid bone
Ligature mark
Findings in neck
Seminal stains
Purplish PM lividity
Salivary and seminal dribbling on the floor

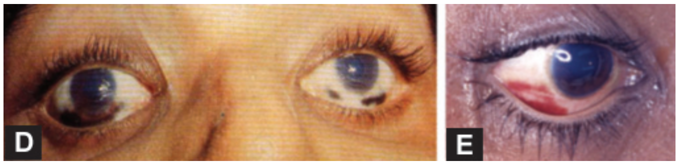
Fig. 15.5A: External autopsy findings in hanging

Thus, in the eyes of victim of hanging, wherein one of the eyes may be opened with dilated pupil, while the other eye may be closed. This occurs due to the pressure of the ligature/knot on the cervical sympathetic ganglia in the neck. Thus, the side on which the ligature/knot presses the cervical sympathetic ganglia, the eye remains open with dilatation of its pupil.[8]

– Subconjunctival hemorrhages (Figs 15.5D and E). Petechial haemorrhagic spots may be noticed with subconjunctival tissues and inner aspect of the eyelids.[7]



Figs 15.5B and C: *External autopsy findings in hanging:* (B) Swollen, cyanosed, face; (C) Protrusion of tongue (*Courtesy:* Capt. Dr. Santha Kumar, Professor & HOD, Forensic Medicine, Govt. Kilpauk Medical College, Chennai, Tamil Nadu)



Figs 15.5D and E: Hanging – Eye findings: Prominent eyes with subconjunctival haemorrhages. (*Courtesy:* Dr. B. Suresh Kumar Shetty, Asst. Professor, Dept of Forensic Medicine, KMC, Mangalore, Karnataka)

- ***Changes in the tongue*** – Tongue may be protruded out (Fig. 15.5C) due to pressure on the floor of the mouth by the ligature material from below pushing the tongue out of the oral cavity. Protruded portion of tongue often turns *black* due to drying.[1]

- ***Bleeding from nose/ears*** **–** Due to the impaired venous return and increase in pressure within, resulting in rupture of vessels and passive flow of the blood from nostrils and ears.
- ***Salivary dribbling marks*** **–** It is a constant and important finding in case of death due to hanging. Normally salivary secretion is a constant phenomenon in life, however, it is being swallowed continuously. When death takes place the deglutition also stops, while the local pressure and irritation exerted by the ligature on submandibular salivary glands continues and this results in secretion of saliva. This can flow passively through the lower angle or dependent parts of the mouth, and is suggestive of *antemortem hanging*[3-6,9] (Fig. 15.5F). Dribbling occurs from the angle of mouth whichever is at lower level, i.e. from the angle opposite to the side of knot. When the knot is on the nape of neck it occurs from middle of the lower lip. When the knot is under the chin, then it occurs through either or both of the angles of the mouth.[9] The saliva that dribbled out drops down in front of the chest when body is bare or it stains the clothes in front, when the victim is dressed. When dried or partly dried, it becomes quite fixed and cannot be easily removed or rubbed off. However, salivation can be also due to asphyxia and congestive hypoxia. Salivation may not occur, when death is due to vagal inhibition. Evidance of salivation should always be looked for testing the presence of mucin in it by turning blue if treated with iodine, during autopsy.[3] However, rough handling of the dead body kept in the cold chamber might remove these stains.[4-6]

II. Findings in the Neck

Findings in the neck are basically two only and they are:

1. ***Distended neck veins*** – *Prominently distended neck veins, above the ligature.*
2. ***Ligature mark of hanging*** **–** A detailed knowledge about the ligature mark of hanging essential for an autopsy surgeon, is highlighted precisely in Figures 15.5G to K, and described below:

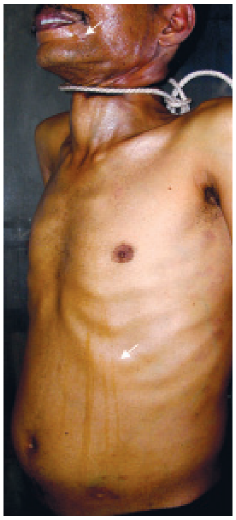
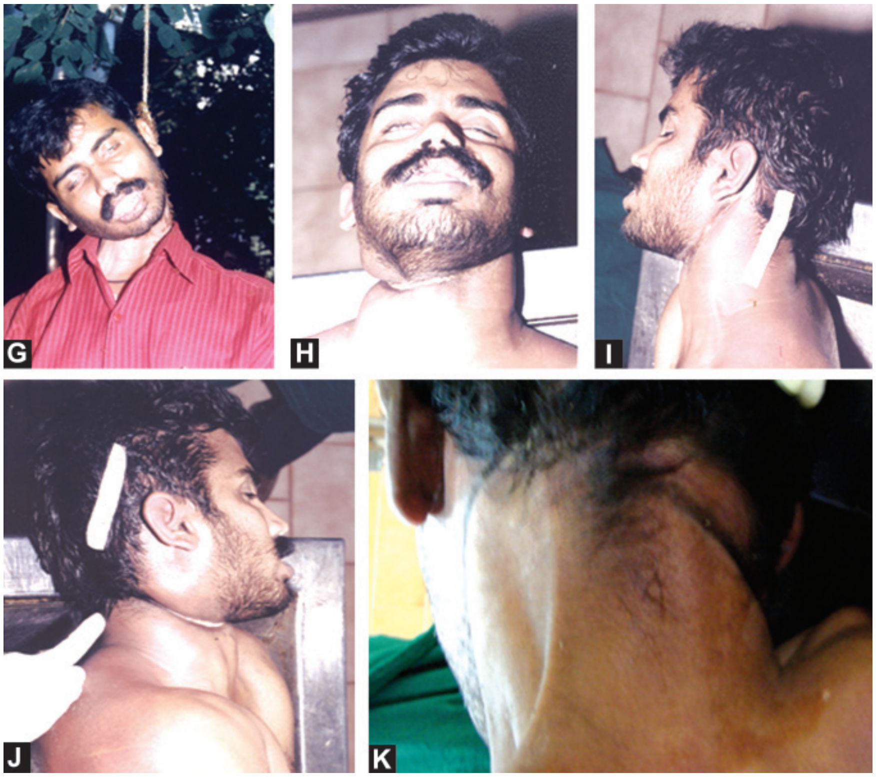


Fig. 15.5F: Hanging case with salivary dribbling marks on front of the face at and below the left side angle of the mouth and front and left side of the chest and abdomen



Figs 15.5G to K: *Ligature mark of antemortem hanging:* (G) Hanging *in situ*; (H) front view – mark running horizontally upwards towards point of suspension; (I) Left lateral view-running behind left ear over mastoid process; (J) Right lateral view – mark is at lower level than on left side, but oblique and moves upwards towards point of suspension; (K) Back view—noncontinuous due to the hairs intervene. *Note:* Inverted ‘V’ shape

- ***Site*** – seen round the neck; usually situated *above* the thyroid cartilage (Adam's apple) in about 75 per cent of cases of hanging, at the level of thyroid cartilage in another 15 per cent and below the thyroid cartilage in remaining 10 per cent cases.[4,7,8]
- ***Size/shape*** – depends on the *type of material used.*
- ***Cause*** – *weight of the body* tightening the ligature material around the neck.
- ***Appearance*** – A typical ligature mark is usually seen in *antemortem hanging.* It is also reported to appear if *hanged immediately after death* or within a period of 2 hours of death (*vide infra*).[11] However, ligature mark produced so will not be as prominent as in antemortem hanging.

Typical Ligature Mark of Antemortem Hanging

It has following characteristics:

1. ***Distribution of the mark:*** It is non-continuous, placed high up in the neck, above the level of thyroid cartilage (as stated above) and runs *obliquely, backwards* and *upwards* towards the point of suspension (Figs 15.5G to K). The ligature mark is non-continuous in the nape of the neck where the hairs intervene between the ligature material and skin underneath (Fig. 15.5K). Ligature mark may also be non-continuous at the site of knot due to some gap, produced by the pull on the knot from the point of suspension above. When the knot is in contact with the skin it is usually inverted "V" shaped (Fig. 15.5K), due to extension of ligature material downward on either sides from the knot above.[9] Ligature may be continuous in partial hanging.
2. ***Skin at the site***: It is usually depressed, pale, dry, and hard and may be with small abrasions at its edges, corresponding to the thickness and edges of the rope used to hang. These abrasions are also known as *rope burns and they are due to frictional force.*[18]
3. ***The pattern of the ligature:*** Often the pattern of ligature used for hanging gets imprinted on the skin as a *pressure abrasion.* If the ligature material used is tough and narrow like a plastic wire or electrical cord, then the ligature mark is deep and prominent. However, if the ligature material is soft and broad, the ligature mark is less prominent and less deep. It *may not be visible,* if a light and thin cloth material like nylon *sari* is used as ligature material. There may not be any ligature mark if the body has been released from hanging swiftly after suspension. It may not be seen if anything intervenes between the skin and ligature material such as long beard or clothing, etc. Very low point of suspension, old and cachectic body, partial hanging could be the other causes of not vissible ligature mark.
4. ***Postmortem staining:*** The upper margin of the ligature mark has a line of postmortem staining, all around above the ligature mark, due to pooling and settling of blood from head and neck above the level of constriction, which becomes the most dependent part vertically.[1]
5. ***Double ligature mark:*** At times there could be a *double ligature mark observed.* This may occur under following circumstances:
   - A *cloth material* like a *sari* or *dhoti (Lungi)* is used as ligature material, which might have 2 folds (thin) one above the other with thick bands in between the cloth.
   - A ligature which is fastened at times at a lower level on the neck may move upwards due to *slipping* of the body producing *two ligature marks*. In such cases a wider abraded area may be noticed due to frictional displacement of the ligature material. When ligature is applied in multiple turns, multiple, parallel, grooved ligature marks may be seen.
6. ***Microscopy of ligature mark:*** it shows usual characteristics of an abrasion, with desquamation and flattening of cells of the epidermis. There may not be findings of any vital reaction if the death occurred immediately after asphyxiation.
7. ***Other factors influencing appearance of ligature mark:*** Other factors which influence the appearance of ligature mark are several as noticed and enumerated below:
   - *Period of suspension* – more the period of suspension, more prominent will be the ligature mark.
   - *Degree of suspension* – total suspension routinely presents with a prominent ligature mark compared to partial suspension.
   - *Weight of the body hanged* – heavier is the person hanged, deeper and prominent will be the ligature mark.
   - *Tightness of the ligature* – more is the tightness of the ligature, deeper will be the ligature mark.
   - *Any intervening material* – Any intervening material such as shirt collar, cushioning by a soft cloth pad/handkerchief, etc. (as seen with *auto-erotic,* masochistic type of sexual asphyxia cases) between the ligature material and skin, can make the ligature marks less prominent.
   - *Lengthening and bending of neck* – Upon prolonged suspension, neck can become slender and increase in length. However, this is not observed in hanging for short time. The neck may be tilted to opposite side of the knot. This may continue until onset of rigor mortis.
8. ***The knot*** – Among most of the right handed suicide victims, knot is usually seen on the right side of the neck as it is easier for the victim to tie himself/herself. Knot may be a slip knot or granny knot (Fig. 15.5L). Common site for the knot is either right side or left side of the neck. It could be also on the occiput. Occasionally it is seen below chin also. Ligature marks tend to be deeper opposite to the position of knot. When hanging is from a low point of suspension or partial hanging, the mark may be horizontal and could resemble strangulation. However in most cases of hanging it is above the level of thyroid cartilage.
9. ***Availability of ligature material used for hanging with cadaver*** – This may be supplied by the police intact in-situ around the neck or may/may not be sent separately with the dead body.

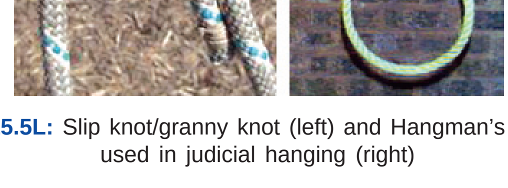


Fig. 15.5L: Slip knot/granny knot (left) and Hangman's noose used in judicial hanging (right)

10. ***Removal and preserving of the ligature and noose*** – Certain types of knots and nooses may suggest involvement of another party in cases which initially appear to be a suicide. In order to enable subsequent examination of a given noose, the knot should not be undone.[10,18] Also prior to removal of the noose from the neck, its nature and composition, width, mode of application, location and type of knot should be described in detail. After noting these observations, removal of the noose is done by cutting it away from the knot and tying the cut ends with a string or a wire.[10,18] This will enable adequate preservation (Fig. 15.5M).
11. ***Scratch marks on the neck*** – Rarely, scratch marks may be seen on the neck above or below the ligature mark of hanging, where the victim tried to undo the noose.[1] These are often refered to as 'periligature marks/injuries' and are always antemortem in nature.[6,7]
12. ***Ligature mark and putrefaction*** – Ligature marks being avascular and dry are found to resists putrefaction.[18] Hence, it can be easily noticed in decomposed body even up to the 5th day after death (Fig. 15.5M_1 to M_3).

III. Findings in Other Parts of the Body

- ***Hands*** – Hands are usually clenched. May be at times the hands may show the jute fibres, such as that of jute fibre when jute rope is used as the ligature material. Hands may also show bluish discolouration of fingertips and nail beds due to *cyanosis*.
- ***Postmortem hypostasis*** – Purple coloured *postmortem hypostasis* is usually in a peculiar distribution in the lower limbs and lower regions of upper limbs *(hands/forearm)* (Fig. 15.5N) and at the upper margin of the ligature mark, which are actually lower parts of different body segments in this posture. *This peculiar distribution* of the postmortem staining is however, though *not suggestive of antemortem hanging,* speaks of the fact that body was in *a state of suspension in an upright position for a considerable period after death.* It may also be noticed here that such hypostasis could be developed even if a *dead body is hanged immediately after death (perimortem hanging).*
- ***Involuntary voiding of urine and /fecal matter***– stains of which may be seen on the under garments/on the floor below.
- ***Abrasions or contusions on lateral aspect of shoulder, feet, toes*** – Abrasions or contusions on lateral aspect of shoulder, feet, toes, etc due to friction with a wall or a pillar during suspension, in the last phase of life when there are convulsions. Injuring parts of the feet or toes may also be noticed, is again common while making an attempt to jump from a support such as a wooden stool or a steel chair or a tree branch at a height, or such other things in the process of inducing suicidal hanging. Otherwise there shall be no other injuries seen.
- ***In male victims:***
  - *Penile turgidity—penis may be found turgid and erect*—no definite causes are described up to date. However this is usually not seen. Several possibilities are presented. Accordingly one of the explanations is that, it could be due to *pooling of blood* after death into the vascular spaces within the penile musculature due to *gravity shifting.*
  - *Seminal voiding*—seminal emission may be noticed at the tip of the glans penis over the thighs (Fig. 15.5O) or seminal stains may be seen on inner garments, etc. It may also be seen on the floor immediately below the site of hanging. This is explained to be due to *sphincter relaxation* due to *asphyxia of higher centres of the brain* controlling these muscles. However, this may also be seen in death due to natural causes.
  - Voiding of urine and stools – may also be seen due to relaxation of the sphincters.
- ***In female victims:*** Apart from the voiding of urine and stools, turgescence of vagina with blood-stained discharge may also be noticed.[4]

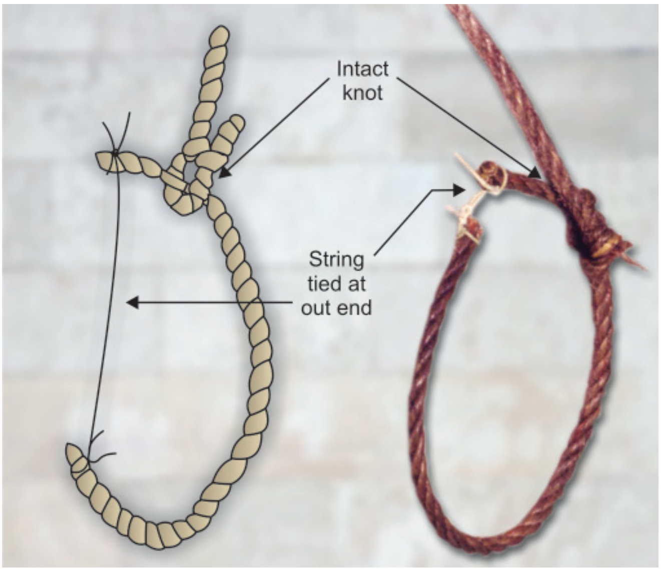
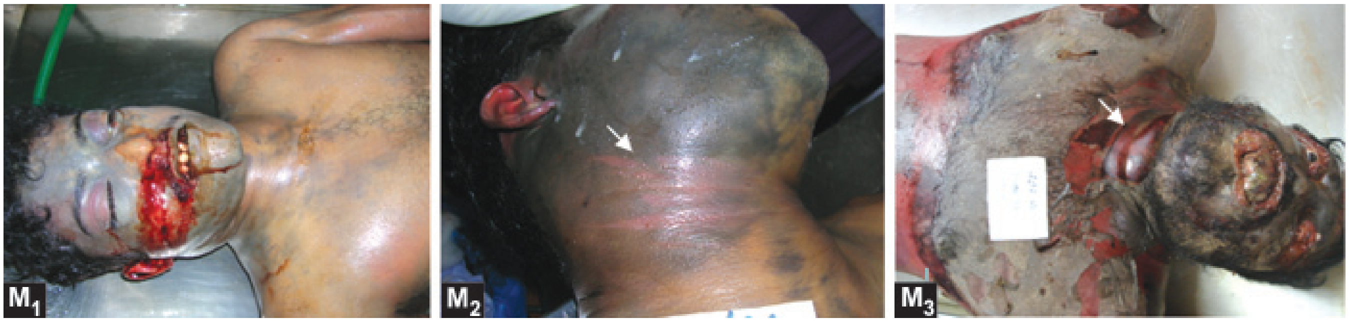


Fig. 15.5M: The method of removing the noose with intact knot for preserving



Figs 15.5M_1 to M_3: (M_1) Both the cadavers M_1 and M_3 were recovered in hanging position with decomposition changes. (M_2 and M_3) Ligature marks noticed on the neck

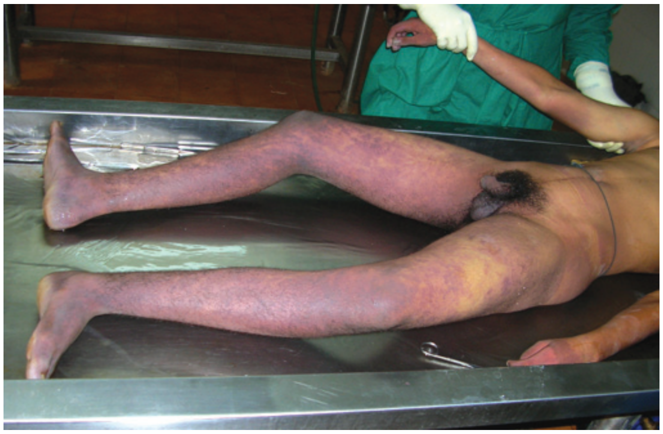


Fig. 15.5N: Purplish postmortem lividity over the lower extremities and forearms. *Note:* Distribution of lividity is corresponding to dependent parts of the body position which is invariably vertical in hanging

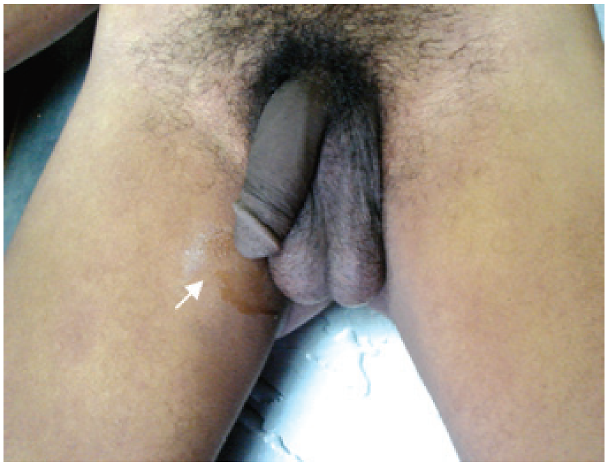


Fig. 15.5O: Voiding of semen (Arrow)—Seminal stain on inner thighs in a victim of antemortem hanging

External Autopsy Findings in Cases of Vagal Inhibition Death

Here death is instantaneous and hence none of the usual asphyxial findings are seen.

INTERNAL

Internal autopsy findings include findings in the *neck, lungs, heart, brain, other abdominal viscera and blood.* Of all these, neck findings are more important and will be discussed later.

- ***Findings in the lungs*** – Lungs will be congested, distended and emphysematous with plenty of hemorrhagic spots *(Tardieu's spots)* subpleurally. Cut section usually exudes frothy fluid blood, if constriction occurs at the end of exspiration. It would be pale with little or no congetion, if constriction occurs at the end of inspiration.
- ***Findings in the heart*** – Heart is congested and shows Tardieu's haemorrhagic spots over the pericardium (Fig. 15.5P). Right side of the heart, pulmonary artery and vena cava are usually found full with dark fluid blood, while left side empty.[7]
- ***Findings in the brain*** – Brain and its membrane will be congested and with or without scattered petechiae on its surface and substance.

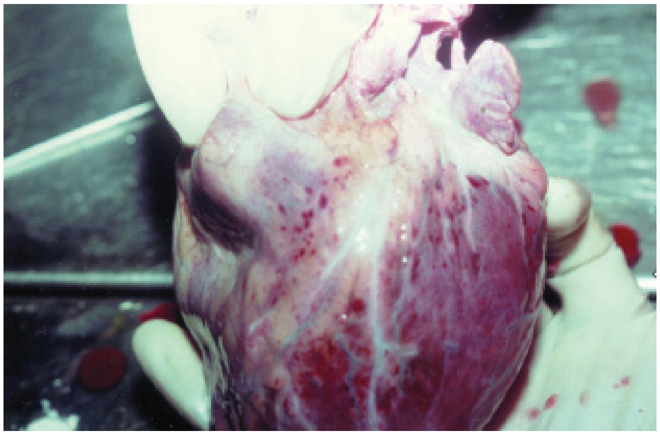


Fig. 15.5P: Heart in a case of hanging. *Note:* Tardieu's haemorrhagic spots on the pericardium

- ***Findings in abdominal viscera*** – All the abdominal viscera are *congested*.
- ***Blood changes*** – Blood will be fluid and purplish in colour.
- ***Neck findings*** – In every case of death by hanging, a careful dissection of the neck is done, layer by layer, so as to not to miss any of the internal findings, *confirming* hanging as the cause of death. This is possible only if the dissection area is maintained *clean free from bleeding,* which can be only achieved by a special technique called *bloodless dissection of neck*.

Bloodless Dissection of Neck

This simply means conducting the neck dissection at the end of autopsy examination, after performing dissection of cranium above and thorax and abdomen below, with removal of every other viscera except in the neck. Majority of cases according to the author's experience, show no internal injuries in the neck. DiMaio reports of only 50 per cent of cases presenting with internal neck injuries in hanging victims.[1] Probable neck findings suggestive of antemortem *hanging* are:

- Subcutaneous tissue underneath the ligature mark is dry, whitish, firm and glistening.
- The platysma and the sternomastoid muscles may show petechial haemorrhages and may be ruptured occasionally.
- There may be bruising in the subcutaneous tissue and the muscles, deeper to the mark.
- Rupture or tears of intima of carotid arteries above its point of bifurcation, around the sinuses with extravasation of blood in their walls, especially in cases of a long drop, may be observed (Fig. 15.5Q).
- Trauma to hyoid bone and larynx—a brief discussion on forensic anatomy of hyoid bone and larynx is appropriate at this juncture (Fig. 15.5R).

Forensic Anatomy of Hyoid Bone and Larynx

Hyoid Bone

This is a bone described is have a 'U' shape, with a central horizontal body; to which 'greater horns' are attached on either side by a natural joint (Figs 15.6A and B). The greater horns lie behind the sternomastoid muscle at its front part, 3 cm below angle of mandible and 1.5 cm away from the midline. Usually, the connection between greater horn and hyoid bone body is cartilaginous in early life, while after middle age it turns bony.

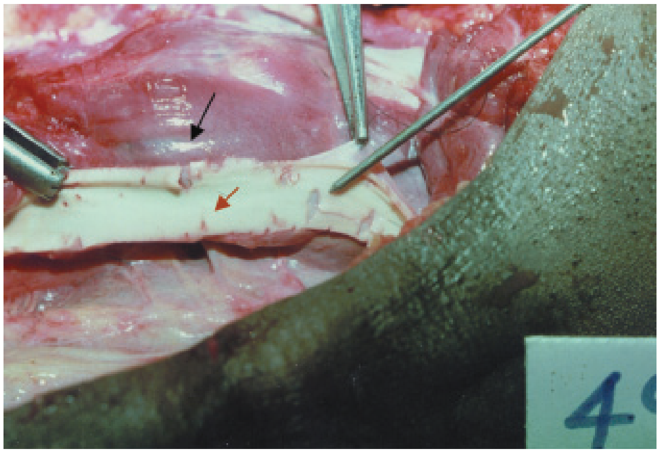


Fig. 15.5Q: Demonstrating bloodless dissection of neck in a case of hanging. *Note:* Dissection of the carotid artery with revealing multiple intimal tears (Arrows) suggestive of antemortem hanging

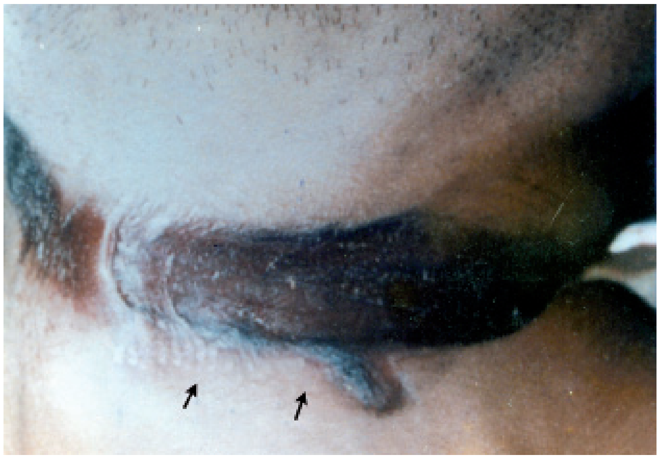


Fig. 15.5R: *Rope burns:* A pressure abrasion due to frictional irritation (Antemortem sign) (*Courtesy:* Dr SC Mestri, Professor and HOD, Dept of Forensic Medicine, JSS Medical College, Mysore, Karnataka)

However, there are lot of controversies regarding this.[7] Hyoid bone has two 'lessor horns' located close to the greater horns, but has no medicolegal significances.

Larynx

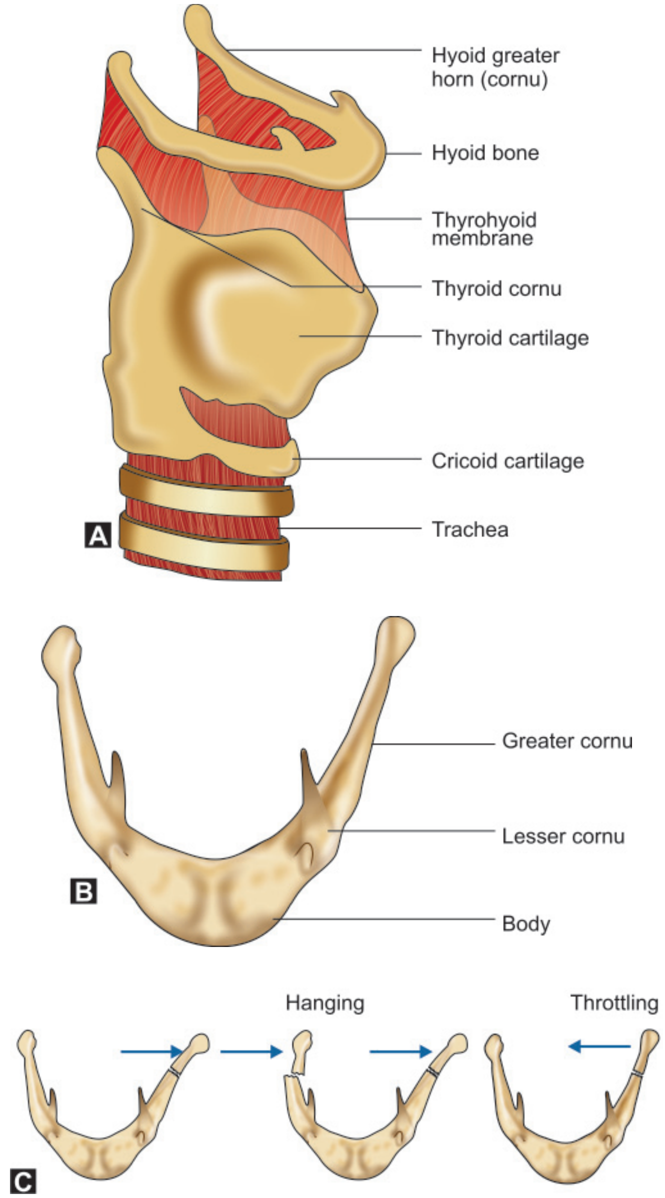
Larynx is a neck structure in the mid-line anteriorly. However, its exact location varies with sex and age. In an adult male, it lies opposite 3rd-6th cervical vertebrae, while in a female, it is at a higher position slightly. It comprised of nine cartilages; namely, thyroid, cricoid, epiglottis and smaller pairs of cuneiform, corniculate and arytenoids. Thyroid and cricoid cartilages are more important.

Cricoid Cartilage

Cricoid is of signet ring shape with signet part situated posteriorly. It is rarely injured in hanging deaths because of its position and cartilaginous structure.

Thyroid Cartilage

Thyroid cartilage is made up of a shield shaped body in the middle. It is angled at about 90° in a male and 120° in a female. Its location is being covered merely by fascia and skin, it is highly vulnerable to injury. At its backside, superior and inferior horns are attached to the body. The superior horns are firmly attached to the hyoid bone by thyrohyoid ligament. It consists of hyaline cartilage and gets ossified earlier in men than women; as the age advances, begining at about 25 years. However, it may not ossify even at advanced old age.



Figs 15.6A to C: (A) Anatomy of the larynx, (B) Hyoid bone (Normal morphology), (C) Fracture of hyoid bone in hanging and throttling (probable mechanisms)

Mechanism of Fracture and Fracture Dislocation of Hyoid Bone

- Hyoid bone—usually reported to remain intact (90-95%). However, if fractured it is usually seen in age group of 40 years and above, at the greater cornu at the junction of inner two-thirds with outer one-third (Figs 15.6C and D).
  These fractures usually fall under any one of the following three types:
  - *Antero-posterior compression fracture* – Here the distal fragment gets desplaced outwards and periosteum may be torn on the inner aspect.
  - *Side-wise compression fracture* – Here the distal fragment will be bent inwards and the periosteum may be torn

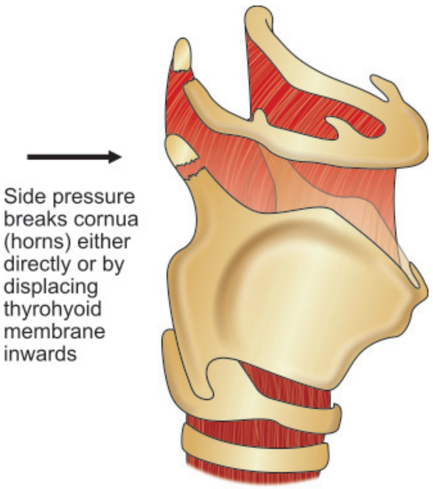


Fig. 15.6D: Mechanism of hyoid and thyroid cartilage fracture in strangulation

on the outer aspect. It is possible that one or both the horns may be fractured due to compression on one or both sides, with one horn fractured on to innerside and the other on to the outer side (Fig. 15.6B).

- *Traction/Avulsion/Tug fracture* – Here due to the powerful muscles attached to the upper and anterior surface of hyoid bone, it is drawn up and held rigidly (Fig. 15.6D). Violent lateral or downward movements of thyroid cartilage or pressure between the cartilage and hyoid bone can bring about the traction through thyrohyoid ligaments resulting into this fracture of the hyoid bone.

These fractures are usually with extravasation or blood in soft tissues around; need not be seen in all cases of antimortem hanging. It is said that circulation gets compromised during hanging and hence no bleeding is seen even if the hanging is antemortem.[4,6]

Fracture of thyroid cartilage – Fracture of superior horns of thyroid cartilage is common like greater horns in hyoid bone and this usually depends on state of ossification of these structures.[4] According to Polson, the incidence is 50 per cent of all handing cases they studied.[7] However, common neck injuries are tabulated in Table 15.1.

MEDICOLEGAL ASPECTS OF HANGING

Postmortem Hanging (Postmortem Suspension)

Hanging could be a *postmortem hanging* also, in order to conceal and mislead a case of homicide as suicidal hanging. Differences between antemortem and postmortem hanging is shown in Table 15.2.

Suicidal Hanging

In India, hanging is a common mode of committing *suicide* among *men*. Table 15.2, highlights the differences between antemortem and postmortem hanging (postmortem suspension). Age is no bar for suicide by hanging. Author held an antopsy on a boy of 12 years who committed suicide for not able to get admission in a private school due to high fee. Partial hanging is almost diagnostic of suicidal hanging.[4] Physically disabled, blind person, lame, amputated arms, or forearms – all have successfully committed suicide by hanging.[4]

Homicidal Hanging

This is very rare. It is virtually impossible to hang a healthy adult unless beaten to unconsciousness or rendered helpless by alcohol or drugs.[1] Firstly, one may suspect homicide by virtue of the injuries on the victim. Secondly, there may not be any struggle marks on the body except noose mark or possible contusions of the arms, inflicted while being forcibly restrained to hang. This may also be a fact when there is disproportion between victim and assailant, such as an adult and a child or a huge man and a small woman etc. to rule out any violence that may not be visible and to make sure that the individual was not drugged. In every alleged case of suicidal hanging, a complete toxicological analysis must be performed.[1] Homicidal hanging may be suspected when following findings are observed in the body hanged:

- Knot of the ligature is exactly on the back of the neck
- Mouth is gagged with cloth/paper
- Hands are tied on the backside of body
- Several injuries on the parts of body

All these are suggestive of *struggle* by the victim, being attacked before hanged. However, tying of hands and feet may also be observed among victims of sexual asphyxial practices (refer below). Gagging, self suffocation, cutting of the wrists,

Table 15.1: Common neck injuries in hanging deaths

Neck structures injured	*Type of injury*	*Probable courses*	*Frequency*
Muscles of neck	Haemorrhages bruising rupture (rarely)	Direct pressure indirect stretching	More common
Blood vessels on ipsilateral side to the location of knot	Transeverse tear of intima with haemorrhage	Traction on the blood vessel	Rare
Hyoid bone	Fracture	Point of suspension and width of ligature	Common in age >30 years
Thyroid cartilage, sup. horns	Fracture	Location of knot Locating ligature – Length of ligature – Possible swing – Indirect force by stretching of thyroid ligament and thyroid membrane	More frequent common in age >30 years

Table 15.2: Differences between antemortem and postmortem hanging (postmortem suspension)

Characteristics	*Antemortem*	*Postmortem*
Salivary dribbling mark at the angle of mouth, opposite side of knot	+ve	–ve
Fecal/urinary discharge	not very common	–ve
Ligature mark		
Direction	oblique	Circular
Continuous/not	noncontinuous	Continuous
Level in the neck	high up above the level of thyroid cartilage.	Low, below below the level thyroid cartilage.
Parchmentisation	+ve	–ve
Vital reaction	+ve	–ve
Knot	Single, simple, on one of the side of neck/ rarely on chin or occiput	More than one, granny or reef type on the occiput/chin
Tongue may or may not be protruded out, bitten at the tip	+ve	–ve
Injuries		
Imprint abrasion/ Bruise	Always +ve and with vital reaction	+ve/ -ve and if +ve no vital reaction
Elongation of neck	+ve	–ve
PM staining		
Above ligature mark	+ve	–ve
in lower limbs	+ve	+ve
Glove-stocking like distribution	+ve	In dependent parts of body at the time of death and position at death
Lung Surface		
Emphysematous bulla	–ve	+ve
Evidence of Injury		
Self-infliction	+ve (compatible)	–ve
Struggle evidence	–ve	+ve
Tear of carotid artery intima	+ve	–ve
Fracture of hyoid	+ ve (in long drop)	
Cyanosis	Deeply +ve	–ve/ faintly +ve
Point of suspension	Compatible with antemortem self-suspension	Not so

stabbing chest, self shooting, etc. may be noticed in the dead body of hanging, as hanging is often opted by these victims as last measures who fail to die with all these techniques, may mislead the case as homicidal hanging.

Accidental Hanging

Though the incidence of accidental hanging is not very common; it may be reported among infants, children, adolescent and adults.

- ***Among infants***—e.g. umbilical cord *around the neck* – this is also known as *twisting of cord around the neck*. This can cause the death of the fetus during birth or occasionally, immediately after birth from asphyxia due to accidental strangulation.[9] An infant may accidentally slip down in between the side grills of a poorly designed crib, may die either because of constriction of neck with the body being in suspended position[9] or because of smothering when trapped between a small mattress and the frame of the crib with face wedged against the mattress.[1] Rarely, infants may get hanged accidentally from a pacifier suspended around their neck on a cord or get entangled in a toy or mobile laced above their crib.[12]
- ***Among children***—e.g. while playing *Lasso*. Here the neck of the child may get entangled in a ropes resulting in death due to asphyxia. Children while playfully acting hanging (especially judicial hanging scene), may actually be hanged to death.[1,9]
- ***Among adolescent/adults***—e.g. *Sexual asphyxia*, popularly known also as "*sex associated asphyxia, autoerotic hanging, asphyxiophilia, hypoxiphilia, Kotzwainism, masochistic phenomenon*", etc. These are asphyxial deaths in principle, caused by hanging; in which transitory anoxia is intentionally induced to enhance sexual arousal produced by *masturbation*.[1,16] Within the victim's view may be found scattered pornographic literature supportive of deviant practice. Usually this is practiced in secluded, private spots and in any accidents resulting in continuation of asphyxiation, death is inevitable. This is more commonly practiced among the adolescent boys in comparison to old aged men. Literature though reports few such incidences among

female,[16-19] it is extremely rare. Individual may often be dressed in female attire, particularly underwear. Neck is usually found to be protected by soft cloth pads between skin and the noose preventing any visible ligature mark. Some element of *bondage* or *tying* may also be seen in these victims of auto-erotism[20] which may mislead the case as homicidal hanging unless the investigating officer is aware about the fact. Victim of sexual asphyxiation may also be found with other methods of asphyxiation such as strangulation, or with plastic bag covering the head and face, etc.[18]

Judicial Hanging

Cause of death in judicial hanging is due to injury to spinal cord in the neck because of the fracture and dislocation of upper cervical vertebra $C_{1,2 \text{ or } 3}$ and contusion or transaction of the underlying cord. The knot is usually placed under *left ear* or less commonly, under the *chin*. A trap door on which he is standing is sprung suddenly and the prisoner falls into the pit below, a specific distance or depth here being determined by his height and weight.

In perfectly performed judicial hanging, the victim abruptly stops at the end of his fall when his head is jerked suddenly and violently backwards, fracturing his spine, known as *hangman's fracture,* a fracture through the pedicles of C_2 with the posterior arch remaining fixed to C_3, C_1, the *odontoid process* and *anterior arch* of C_2 remaining in articulation with base of the skull. This injury pattern is caused by *hyperextension* and *distraction* and results in injury of the cord at $C_{2\text{-}3}$. This classic fracture does not occur always in hanging.

According to Spence et all, in six cases of judicial hanging studied by them, they observed fracture of the hyoid, the styloid process, occipital bones, cervical body of C_2 and transverse process of $C_{1\text{-}3}$ and C_5.[13,14]

Lynching

Lynching is hanging of a victim who may be criminal by a mob. Thus it is homicidal hanging. The people who get angry by the offence committed by the accused, kill him publicly by hanging to demonstrate the punishment given to him. This was common in South America, where black rapists were killed by angry white mob.[9] The terminology lynching is more often used presently for any type of killing of a social offender, publicly by a mob.

Difficulties in Diagnosis of Death from Hanging

Following may be difficult to explain whenever encountered in a body found hanged:

- Ligature running around the neck —victim might be killed first by strangulation and then hanged to simulate suicidal hanging.
- Presence of two-ligature marks—it might be an antemortem hanging case, but after few minutes of hanging, it might have slipped further down producing second ligature mark.
- Nail marks on the neck—it might look like manual throttling. But possibilities are that, the victim after getting hanged, made efforts to release the ligature around neck with his or her fingers and while doing so his or her own fingernails might have produced nail marks.
- Faint ligature mark—may be seen in dark skin complexion person.
- Injuries on the body—victim while inducing hanging might have got injured him or herself (*wide supra*).

STRANGULATION

Definition

Strangulation is a form of violent asphyxia caused by constricting the neck by some means, the force of constriction being other than weight of victim's body.[1,3-7]

Explanation — here the application of pressure on the neck is either by bare hands, or by a ligature, or by any other material. In this, asphyxia is caused by constriction of the neck without suspending the body.

Neck structures involved in strangulation — Various neck structures involved during strangulation are presented diagrammatically in linedrawing Figure 15.7A.

Classification with explanation of each type, autopsy findings and medicolegal significance of strangulation are discussed below individually briefly.

Classification of Strangulation

Depending on the means used, strangulation could be of several types and they are - *ligature strangulation, manual strangulation (throttling), mugging, bhansdola, garroting and palmar strangulation*. Each one is explained below briefly along with autopsy findings separately. The medicolegal aspects of strangulation are also discussed in general.

Ligature Strangulation

When a ligature material is used to strangulate the victim it is called ligature strangulation. Here the pressure on the neck is applied by a constricting band that is tightened by a force other than the victims body weight.[1,6,9]

Postmortem Appearances

External

All findings are same as in hanging, except certain specific findings in the neck as enumerated below (Fig. 15.7C).

Ligature mark

- Seen all around the neck.
- Situated at the level of lower part of thyroid cartilage or below it.
- It will be running horizontally
- Few abrasions seen – finger nail marks when present are directed downwards and outwards.

Other parts: Strangulation being usually homicidal, injuries due to struggle, known as *struggle marks* are often observed, as the victim usually makes all the effort to resent prior to getting strangulated. These could be abrasions, scratches or abraded contusions over face, arms and other parts of the body.

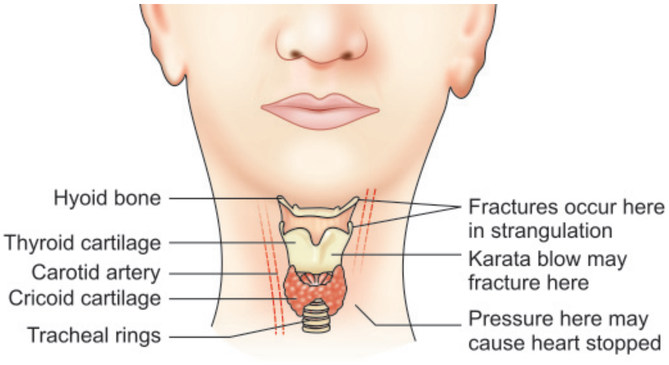

Hyoid bone
Thyroid cartilage
Carotid artery
Cricoid cartilage
Tracheal rings
Fractures occur here in strangulation
Karata blow may fracture here
Pressure here may cause heart stopped

Fig. 15.7A: Diagrammatic representation of neck structures involved in strangulation

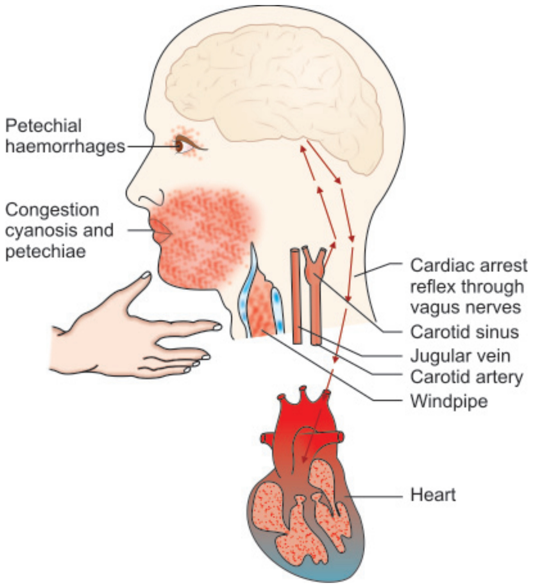

Petechial haemorrhages
Congestion cyanosis and petechiae
Cardiac arrest reflex through vagus nerves
Carotid sinus
Jugular vein
Carotid artery
Windpipe
Heart

Fig. 15.7B: Effects of pressure on the neck in manual strangulation: (1) Carotid sinus pressure – vagus nerve stimulation – cardiac arrest; (2) Carotid artery blockage – unconsciousness; (3) Jugular vein blockage – congestion and haemorrhages; (4) Airway blockage – oxygen lack

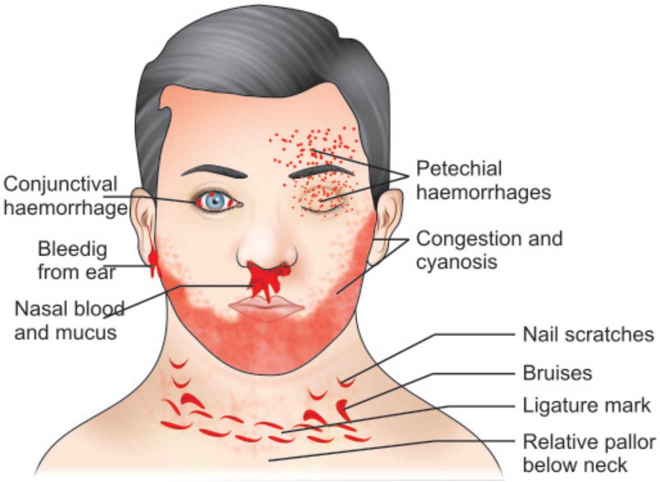

Conjunctival haemorrhage
Bleedig from ear
Nasal blood and mucus
Petechial haemorrhages
Congestion and cyanosis
Nail scratches
Bruises
Ligature mark
Relative pallor below neck

Fig. 15.7C: Classical features in strangulation

Internal

- Thyroid cartilage – usually fractured vertically in all strangulations.
- Hyoid bone – is usually not broken. However if seen, it is seen in the greater cornu at the junction of outer one-third with inner two-third, with the broken fragment displaced outwards (*see* Fig. 15.6C).
- Bruising of the soft tissue and muscles of the neck is more common in strangulation than in hanging. It is by the ligature, more so when a rough ligature is used, and there has been struggle and resistance. Bruising of the subcutaneous tissue may be present even when there is no external marks on the skin. However, brusing may be absent when the ligature has been tightly secured and not removed until circulation stops.[4]

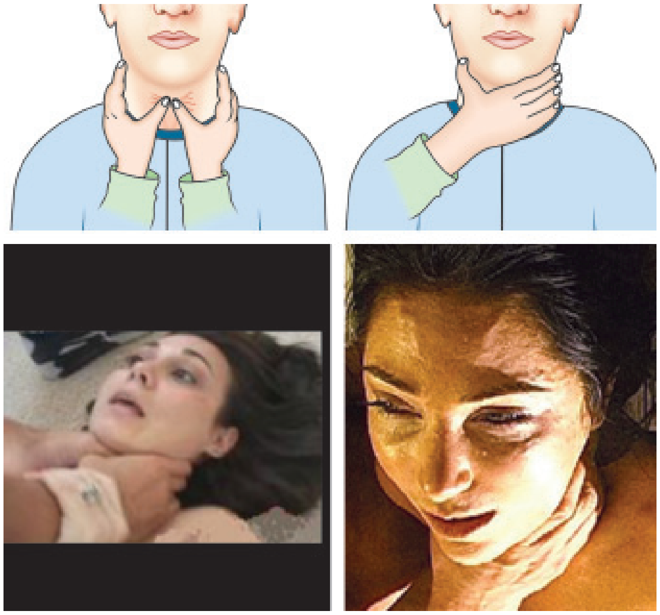


Fig. 15.7D: Mannual strangulation (throttling) using both hands (left) and one hand only (right)

Manual Strangulation (Throttling)

When bare hands are used to compress the neck and strangulate it is known as manual strangulation or throttling. The mechanism of death is by occlusion of the blood vessels supplying blood to the brain, i.e. the carotid arteries. Occlusion of the airway probably plays a minor role, if any, in causing death.[1] Carotid sinus pressure, may result in vagus nerve stimulation resulting in cardiac arrest (vagal inhibition) (Fig. 15.7B).

Autopsy Findings

Signs of asphyxia are present. In addition, following specific findings are obvious in the neck: (Figs 15.7E to G).

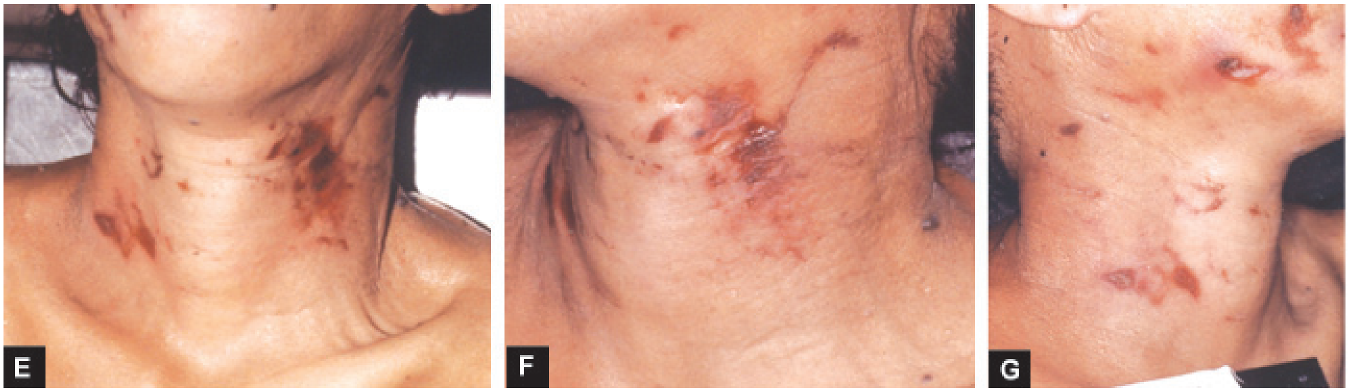
External

Bruising of the neck – Bruising of the neck is due to the grasping of neck by the assailant. These bruises are usually circular, dark red or purplish in colour, and are about 1-2 cm in size. If the gripping fingers slide over the skin, bruises take up an elongated shape (Figs 15.7E to G).

Abrasions on the neck – The neck examination will show fingernail marks, which could be from both assailant as well as victim. They can be curved, semilunar or linear. If curved they could be concave or convex due to the elasticity of skin and subcutaneous tissue. When one hand is only used, thumbnail impression is seen on one side of the neck, while other four-fingernail marks are seen on other side of the neck (Fig. 15.7D). When both hands are used, thumbnail impression of both hands will be in the middle with rest of the four fingers nail marks seen on either side of the neck (Fig. 15.7D). However, if the thumbs overlap then midline impressions would be absent. Thus, the finger nail marks, are helpful in interpretation of the case giving opinion.

Internal

If extensive pressure is used, it can lead to fracture of thyroid cartilage, and inward fracture of greater cornu of hyoid bone (*see* Figs 15.6C and D).



Figs 15.7E to G: Manual strangulation (throttling) findings in the neck. (E) Front view; (F) Left lateral view; (G) Right lateral view. *Note:* Bruises and abrasions of fingers and finger nails

Mugging (Arm-Locks)

Neck is compressed or squeezed by holding it between the crook of the *elbow* or *knee.* The attack is usually made from behind. Though a victim killed by this method shows signs of asphyxia, and other struggle marks, on examination neck will not show any evidence of violence, as the compression was affected by the soft muscles of the arm or leg.[6,9] It may also be because the death is due to vagal inhibition.[4]

Bhansdola

Neck is compressed between two *bamboo sticks* one held in front of the neck and the other behind it. This may also be induced by just one bamboo stick or a metal rod, compressing over the neck against the ground or wall. This may be even done by compressing the neck on the ground by a foot from the front.[5,6,9]

Garroting

In this method a thin string is thrown around the neck of an unaware victim, from his back. The ligature material is then tightened rapidly with the help of two sticks tied at the free ends of the string, so as to constrict the neck strongly.

Note: The method is often used by *Thagi's* in the past in India, to kill the travelers and rob their belongings in lonely places. In Spain, garroting was also said to be the official method of execution of criminals[9] (Fig. 15.7H).

Palmar Strangulation

Palmar strangulation is a combined from of violent asphyxia where in smothering and strangulation are performed together. In this, pressure on the neck is applied by both hands. When base of the palm of one hand is used to compress over the front of the neck, fingers of the same hand compress the mouth and nose, while the second hand is held over the fingers of the first hand, reinforcing the strength of compression.[4-6,9]

Medicolegal Aspects of Strangulation

- *Motive* – Virtually all strangulations whether ligature or manual are *homicidal*.[1] There will be marks of violence, on other parts of the body.
- *Sex incidence* – Though in ligature strangulation, females predominate as victims, it is not as much as reported in case of manual strangulation. According to the studies reported by DiMaio and DiMaio, the most common motive for manual strangulation was rape, followed by domestic violence.[1] Thus if the victim is female, carefully look for evidence of sexual assault, as it is common for an assailant to molest/ rape a girl and then strangle her. Detailed examination of external and internal genital organs, including vaginal washings for chemical and microscopic examination, is performed preferably.[18] Finger nail scrapings and pubic and scalp hair sample from the deceased should also be retained for possible examination later at forensic science laboratory.
- *Ligature material* – It is reported that in ligature strangulation anything which can be twisted may be used as a suitable ligature material. Ligatures used range from electrical cords, neckties, ropes, and telephone cords to sheets and hose. The knot could be half knot, a full knot, a double or tipple knot. Appearance of ligature mark on the neck is subject to considerable variation, depending on the nature of the ligature, amount of resistance offered by the victim, and the amount of force used by the assailant. It may be faint and barely visible, or absent in young children or incapacitated adults, especially if the ligature is soft, for example, a towel, and removed immediately after death. If a thin ligature is used, there will be a very prominent deep mark encircling the neck, which will be yellow parchment-like initially and turn brown later.

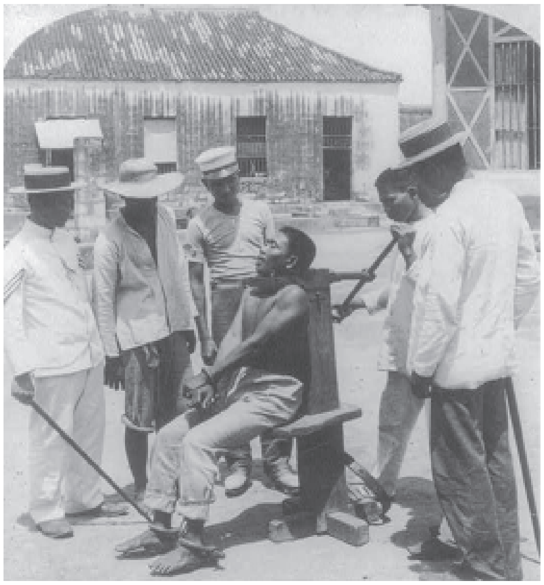


Fig. 15.7H: Execution of a criminal prisoner by garroting at Bilibid Prison, Manila

- *Suicidal Manual Strangulation* is never reported. Manual self-strangulation is impossible, as pressure on the neck depends on voluntary action, which is discontinued when the power of decision is obliterated with loss of consciousness.[4,6,18]
- *Suicidal Ligature Strangulation* is not rare, but in these cases the ligature is sustained by '*stay*' mechanisms such as overlapping loop. Some of the methods adopted for suicidal strangulation are described below:[1,9]
  - One may go on giving turns of ligature material around his neck until he becomes unconscious. The death ensues here prior to the turns of ligature getting untwisted to relax the constriction of the neck.
  - Another method is that after encircling the neck with a rope he may go on putting knots until he becomes unconscious and dies.
  - In this method one may lie down on the side of a cot and tie a rope round his neck loosely and at the other end of rope he will tie a weight. He then drops the weight down the cot. The length of the rope being shorter than height of the cot, he gets strangled due the weight of the stone hanged.

SUFFOCATION

Definition

Suffocation is a form of asphyxia caused by lack of oxygen in the atmospher or by mechanical obstruction to the air passages by mechanical means other than constriction of the neck and drowning.

Classification

There are several forms of suffocation in general and they are enumerated and discussed individually below:

- Entrapment
- Environmental suffocation
- Smothering
- Choking
- Traumatic asphyxia
- Burking
- Suffocating gases.

ENTRAPMENT

This is due to inadequate oxygen in the environment. These are exclusively accidental in nature. In entrapment, people find themselves trapped in an airtight or relatively airtight enclosure. Initially there is sufficient oxygen to breathe. However, as respiration continues, they exhaust it soon as time passes and then asphyxiate. E.g.: A child getting trapped in a *discarded refrigerator*. Fortunately, this specific form of death by entrapment is rare these days, as modern refrigerators do not have a latch system of locking and can be pushed open from within. Suicide and homicide by entrapment are rare, but do occur.[1,6,18]

ENVIRONMENTAL SUFFOCATION

Here the individual inadvertently enters an area where there is gross deficiency of oxygen, e.g. mountaineering – where people climb to reach high altitudes, certain profession demanding entering into the underground chambers, petrol or diesel tanks or tanker carriages, unused deep wells, underground sewage drain system, etc. for cleaning purpose, may get suffocated due to lack of oxygen or presence of irrespirable gases in it.[1,6,9,15]

In death due to entrapment and environmental suffocation, the cause of death cannot be determined by autopsy alone, as there are no specific findings. *All that one finds is nonspecific acute visceral congestion*. It is only by analysis of circumstances leading up to and exclusion of other causes; one can determine the cause of death.[1]

SMOTHERING

Definition

Smothering is a form of asphyxia caused by mechanical occlusion of external air passages, *viz.* the nose and mouth by *hand, cloth, plastic bag, duct tape* or such other material.

Autopsy Findings

Following specific findings are usually seen:

- Abrasions, bruises and lacerations - may be found around the mouth and nose in homicidal/accidental/suicidal smothering cases and they are:
  - When hands are used, these abrasions are usually crescent shaped nail scratches.
  - There may be *fracture* of the nasal cartilage with *bleeding* from nose/ mouth (occasionally)
  - Abrasions may not be seen if a soft material such as cloth, a pillow, etc. is used. However *depression* and *bleeding* from the nose may be seen in such cases.
  - At times due to the friction over teeth, there will be frenular tears, abrasions, contusions or lacerations seen on the inner aspect of lips. Teeth may also be loose and dislocated at times with tearing of gum tissue.
  - All above findings are more common and extensive when the face is pressed against a hard surface.
  - Body may show *struggle marks* in other parts of the body, more often noticed in homicidal smothering than in accidental or suicidal type.
    - In *accidental smothering* — by fall over heap of dust, flour, etc, stains of these materials may be present outside as well as inside the face, mouth and nose.
    - In case of *overlaying* by mother's breast — the delicate tissue of nose and face of the infant will be compressed and pale (refer below).
    - In cases of *sexual asphyxia* — the pornographic pictures around, exposed genitals or total nudity of the victim, wearing apparels or inner garments of opposite sex, methods of inducing asphyxia, private or isolated room, etc. act as evidence in favour.
    - In *suicidal smothering* there will be some of the pressure effects but no remarkable injuries detected.

Note: The above findings may be missed, unless looked for.

Medicolegal Importance

- These deaths are usually either *homicidal* or *suicidal*, but very *rarely accidental.*[1]

  Accidental smothering:
- It is common in alcoholics, intoxicated or unconscious person lying in prone position, or may fall or roll over and bury in bins of flour, heap of mud or such other material. Similar may be the case with epileptics, during the attack of fit.[9]
- After birth an infant may die from smothering if it is born with membranes covering the nose and mouth *(cul-de-sac)*.
- Children may get suffocated while playing with plastic bags over the face or head or on entire body *(rarely)*.
- Sexually perverted individuals may cover/envelop the head in *plastic bag* during the *autoerotic experiences* and get killed accidentally.

Overlaying

It is form of accidental smothering of an infant by the mother sharing the same bed with the child, may roll over it during sleep and occlude the air passages by her breast which the infant may be suckling, developing asphyxia and death. Overlaying, the accidental death by smothering caused by a larger individual sleeping on top of an infant is a cause of death that has been documented for centuries. The hazard of death has been reported to be greater in infants less than 5 months of age but may occur in children up to the age of 2 years. When an adult or older child rolls on top of an infant, mechanical asphyxia results. The face may be pressed into the mattress or into the body of the sleeping adult or older child. The infant's air may be expressed, and he or she is unable to cry due to pressure on the thorax and the inability to inhale. Some pathologists and investigators believe that the victims of overlaying have no pertinent physical findings at autopsy and that any injury is indicative of inflicted trauma. Others believe that one may see contusions and abrasions from overlaying in and of itself. Wedging is another form of accidental mechanical asphyxia that may have negative autopsy findings. The prevalence of bruising, contusions, or facial and ocular petechiae is not clear.[9,10]

Medicolegal importance—though overlaying is not a common condition, these cases are likely to be referred as victim of *sudden infant death syndrome (SIDS)*. It may also be a case of *infanticide*. The condition may also be reported when a child and adult are sharing same bed.[9]

Homicidal Smothering

- Homicidal smothering of adults is very rare and also difficult, unless the victim is weak, or stupefied by drugs or drinks like alcohol as seen in *burking*. Some of the methods of homicidal smothering are by:
  - Pressing the face and the nose by hand, towel, pillow, etc.
  - Tying plastic bag or such other materials around the mouth and nose.
  - Pressing the face of the victim over ground.

Suicidal Smothering

- Suicidal smothering by one's own hands on him or her is impossible. However some of the methods attempted are by:
  - Tying a *plastic bag* around the head covering the mouth and nose and tightening it by applying multiple knots until the person loses consciousness.
  - Tying a *pillow* in front of the face with application of several knots until loses consciousness.

CHOKING

Definition

Choking is a form of asphyxia caused by mechanical occlusion within various sites of upper air passages (Fig. 15.8) by foreign object.

These objects may be:

- A solid object like a metallic coin, edible fruit seed, toffees, candies, spherical solid sweets, etc.
- A food particle like bolus of rice particles, fish and any other animal bone, etc.
- Piece of cloth like handkerchief, *sari* or *dhoti* material stuffed into the mouth tightly occluding the air passages.
- Tongue of the person who is in an epileptic attack may fall back onto the posterior pharyngeal wall.
- Sucking of a piece of balloon to puff it or inflate it inside the mouth may result in occlusion of larynx when sucked into it accidentally when the grip on the balloon is not firm.[9]

Mechanism of Choking

Usually any object in the air passages excite violent coughing out, but if this is not successful in expelling the object out, choking results.

- *Choking and size of the object*—in inducing choking size of the object is not important. Even an object smaller than the lumen of respiratory tract can bring about reflux spasm of air passages with fatal consequences.
- *Choking in an epileptic*, during the attack, with the tongue falling against the posterior pharyngeal wall, blocks the entry of air and results in choking.

Causes of death in choking Death is usually due to any one of the following:

- Asphyxia
- Vagal inhibition (occasionally)
- Laryngeal spasm

Autopsy findings Apart from routine autopsy findings of asphyxial death, following specific findings are observed:

- Presence of the foreign object that produced choking will be found in the respiratory tract, when dissected properly.
- If the victim was an epileptic, the tongue must be specially examined for its position and the presence or absence of bruising or bite marks. It is usually found on posterior pharyngeal wall with injuries.

Medicolegal Importance

1. Choking is usually *accidental*, more common in children, who play, laugh, talk, etc. while eating.
2. ***Cafe coronary:*** This is a condition of accidental choking where a bolus of food produces complete obstruction of the larynx. It is called so because it *mimics heart attack*.

   Causes: It could be any one of the two causes enumerated below:
   - Suppressed gag reflex due to gross intoxication with alcohol or large doses of tranquilizers in mental institutions or hospitals.
   - Reflex cardiac arrest as a consequence of stimulation of laryngeal nerve endings.

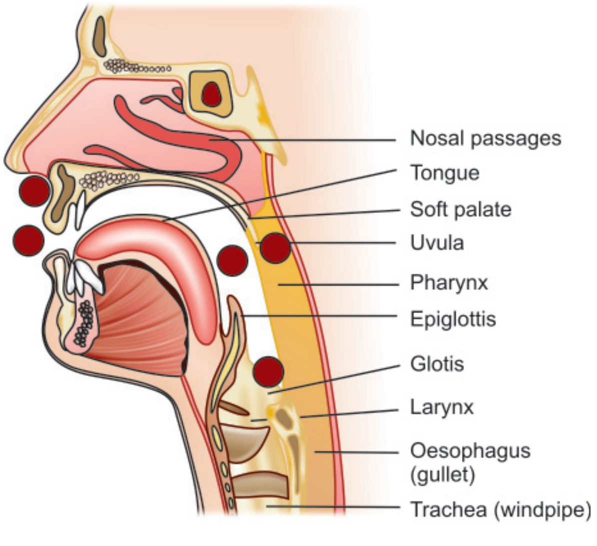


Fig. 15.8: Sites of obstruction to upper air passages

Clinical findings: The victim who was healthy apparently collapses suddenly turning blue while eating at a dining table.

- Autopsy findings: Bolus of unchewed food or such other material is found impacted in larynx.

GAGGING

It is a means to affect choking by preventing the air entry into the respiratory tract by *stuffing gag material (clothes, paper balls, etc.)* into the mouth

Causes

The gag material is usually composed of cloth, paper, etc. being stuffed into the position tightly, filling the mouth and obstructing breathing through the back of the throat.

Mechanism

When a gag material is tightly packed, choking occurs as soon as saliva, mucus, or oedema fluid moistens and the '*air pockets*' in the cloth or paper collapses.

Autopsy Findings

These are same as choking, with presence of material used for choking in the mouth blocking the air passages from within.

TRAUMATIC ASPHYXIA

Definition

Traumatic asphyxia is defined as a form of asphyxia resulting from trauma of the chest leading to forceful compression of the chest preventing respiratory movements.

Causes

Various reported causes are:

- Penetrating trauma like stab injury, resulting in pneumothorax leading to collapse *(atelectasis)* of the lungs.
- Non-penetrating trauma due to run-over motor vehicle accident, or steering wheel impact injury on chest, or in a collapse of a mine or a building, fixing the chest of the victim under a collapsed beam or any masonry work.
- Indirect compression while victim's thighs and knees are drive against the chest, resulting in so-called, 'Jack-knife' position.
- In a stampede by a crowd, running in panic without any sense, as it happens during outbreak of fire in a movie theatre, in a temple during festive occasions with lot of people participating in it, etc. Pressure over the chest from unconcerned movements of persons in a crowd fixing or arresting the normal chest movements.

Cause of Death

Usually death is due to asphyxia.

Autopsy Findings

Findings of asphyxial death are predominantly seen above the level of obstruction to chest, however, with following specific findings:

- Face is congested and livid
- Specific cause producing traumatic asphyxia may be noticed evidently.

Medicolegal Importance

- Traumatic asphyxia is usually accidental.
- Could be homicidal also as described in *burking* (*refer below*).
- It can be rarely suicidal also, when the victim by himself or herself arranges a heavy object to fall on his or her chest.

POSITIONAL ASPHYXIA

This is virtually always accidental and is associated with alcohol or drug intoxication.[1] The victim here gets trapped in restricted spaces, where because of the position of the body they cannot move out of that area or position. As a consequence the breathing is restricted; asphyxia sets in, followed by death.

Example: Fall into a narrow well and wedged between the walls. Every time they exhale, they slip farther down the well, further preventing inhalation.

Autopsy findings show marked congestion, cyanosis and petechiae.

RIOT-CRUSH

As the name implies, this occurs in riots, when the thoracic wall is compressed by stampeding people piling on top of each other. Respiratory movement is thus prohibited by this human piling up, resulting in asphyxia.[1]

BURKING

Burking is a combination form of smothering/palmar strangulation and traumatic asphyxia, resulting in death. This method was apparently invented by two criminals, namely William Burke and William Hare, introducing the terminology after the name of first one, who had planned the method, for killing selected victims and then sold their dead bodies to the Anatomy department of Edinburgh Medical School in early 19th century.

According to the method the victim was first intoxicated with alcohol and when inebriated, pinned to the ground first by Burke who used to sit on the chest of the victim and cover his mouth and compress the neck with one hand, while the other partner Hare pulled the body of the victim round the room by feet.[4-6,9]

Medicolegal Importance

The method is a combined form of *smothering* and *traumatic asphyxia*.

DROWNING

Definition

Drowning is a form of violent asphyxial death, wherein the entry of air into the lungs is prevented by water or other fluids due to the submersion of mouth and nostril *(complete submersion of whole body is not necessary)*.

Incidence

It is quite common to encounter cases of drowning in India and Table 15.3, below narrates the incidences.

Table 15.3: Incidence of drowning in India

Accidental	*Suicidal*	*Homicidal*
Is the commonest form seen among *fishermen, bathers, dock workers* and *intoxicated* and *epileptics*.	Is also fairly common among *women*. A determined suicider will tie the limbs or attach weights to the body.	It is the rarest form of death.

Mechanism and Causes of Death in Drowning

The mechanism of death in acute drowning is irreversible cerebral anoxia.[1,21-23] Original concept of death in drowning was due to asphyxia with water occluding the airways.[21-23] However, experiments and experiences suggested various causes of death among the drowning victims[1,21-25] and they are:

- Electrolyte imbalance (due to fresh/salt water drowning)
- Cardiac arrhythmias
- Vagal inhibition
- Laryngeal spasm
- Head injury during fall in water
- Apoplexy
- Exhaustion
- Infection.

Mechanism of Drowning

- When a conscious non-swimmer falls into the water he sinks initially into the depth of water column owing to factors such as height of fall, depth of water column, and body weight.
- However, the factors such as – struggle movements, *natural buoyancy* of the body and air trapped under the clothing's worn by the victim will help him to rise up to the surface.
- Being a non-swimmer the victim on reaching the surface will make all efforts not only to breathe air but also to shout and attract people for help. In this process air and water are simultaneously inhaled, resulting in involuntary coughing. This in turn diminishes air reserve in the lungs drawing water in its place.
- With the entry of more water into the body (*inhaled* and *swallowed*) there will be increase in *body weight* and *specific gravity (than that of water)* of the victim, consequence of which will be re-sinking of the victim, only to surface again due to all factors mentioned already.
- This will go on repeatedly till victim sinks finally to the bottom, and dies due to exhaustion, loss of consciousness, and the effects of onset of vicious cycle of asphyxia as a consequence of formation of fine froth, in the respiratory tract.
- *Formation of froth* – Froth in the respiratory tract is due to *churning-like mechanism* of air, water and respiratory mucous secretions (*surfactants*), which prevent entry of air into the respiratory passages,[3-6,9] resulting in onset of asphyxia.
- It is claimed that the victim will die within 2 to 5 minutes of complete submersion as asphyxia supervenes within 2 minutes and heart function stops in next 2 to 5 minutes.
- Figure 15.9 illustrates the mechanisms briefly.

Pathophysiology of Drowning

Recent Advances

Death can occur in drowning in an ocean or in water as shallow as 15 cm in case of alcoholic stupor, epileptics, or infants.[1] Mechanism of death in acute drowning is irreversible cerebral anoxia. The original concept of drowning deaths was that they were asphyxial in nature with water occluding the airways. Experiments in the late 1940s and early 1950s suggested that death was due to electrolyte disturbances and/or cardiac arrhythmias produced by large volumes of water entering circulation through on reaching surface lungs.[24,25] Present thought however, is that the most important physiological consequence of drowning is hypoxemia.[21]

When people sink beneath the surface of water, their initial reaction is to hold their breath. This continues till reaching of

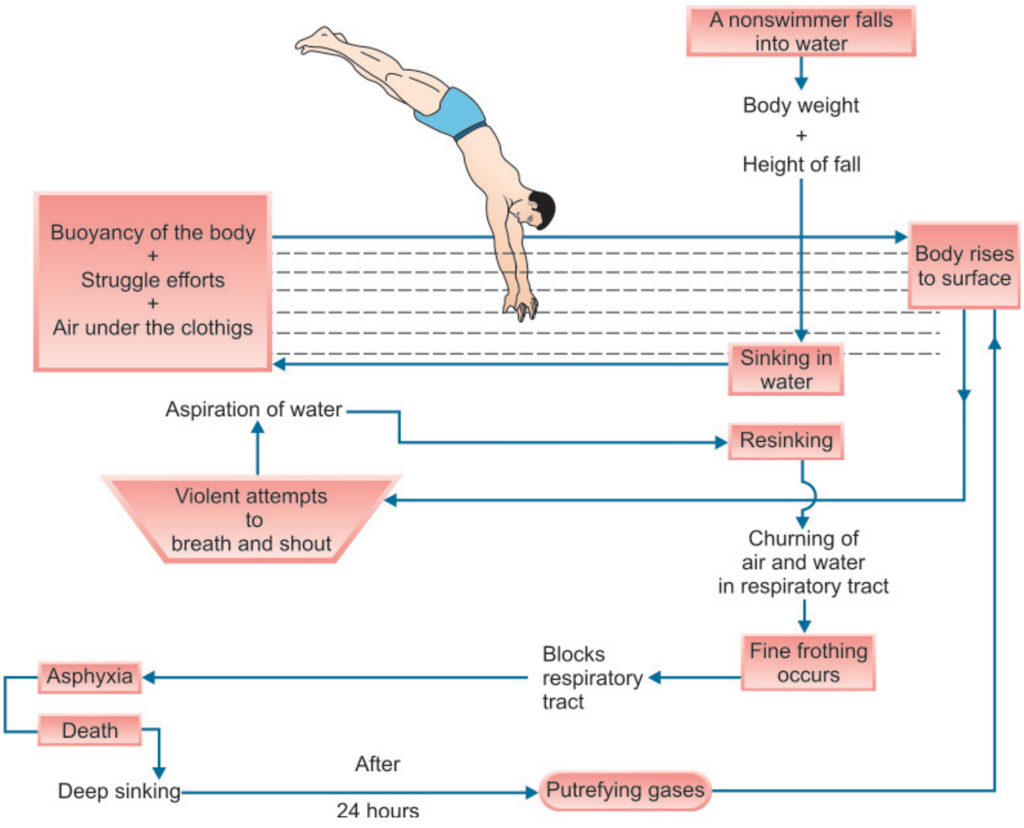

A nonswimmer falls into water
Body weight
+
Height of fall
Buoyancy of the body
+
Struggle efforts
+
Air under the clothigs
Body rises to surface
Sinking in water
Aspiration of water
Resinking
Violent attempts to breath and shout
Churning of air and water in respiratory tract
Fine frothing occurs
Blocks respiratory tract
Asphyxia
Death
Deep sinking
After
24 hours
Putrefying gases

Fig. 15.9: Various events of mechanism of drowning

breaking point at which time the individual have to take a breath. Breaking point is determined by a combination of high levels of CO_2 and low levels of O_2 concentrations. According to Pearn,[21] the breaking point occurs at PCO_2 levels below 55 mm Hg when there is associated hypoxia and at PAO_2 levels below 100 mm Hg when the PCO_2 is high.[1]

Upon reaching the breaking point, the individual involuntarily inhales, taking in large amount of water. Some water is also swallowed and will be found in the stomach. During this phase of submersed breathing, the patient may also vomit and aspirate some of the gastric contents. The involuntary gasping for air under water will continue for several minutes. The developing cerebral hypoxia will continue until it is irreversible and death occurs. This is, however is dependent on age of the individual and temperature of water.

With warm water, this is about 3-10 min,[26] while in cold or icy water among child victim, it may be as long as 66 minutes with fairly good opportunity for resuscitation.[21] The traditional explanation to this longer survival in cold water is that the immature brains (children) are more resistant to anoxia. It also has been recently explained on the basis of '*diving reflex*'. This refers to vasoconstriction in the vascular beds (except for heart and brain), shunting of blood to the brain and heart and bradycardia, all triggered by immersion of face in cold water. Another thought is that children survive due to development of *hypothermia.*[1,27] It is said that because of large body surface area and lack of adequate insulation in children, the body cools rapidly.

The sequence of events is thus summarised below:

Breath holding
↓
Involuntary inspiration and gasping for air at the breaking point
↓
Loss of consciousness
↓
Death

The sequence may be altered if the individual hyperventilates prior to sinking under water. This can cause significant decrease in the CO_2 levels. Thus, cerebral hypoxia due to low blood PO_2, with development of unconsciousness, might occur before the breaking point is reached. In this case, the sequence will be:

Voluntarily holding of breath
↓
Unconsciousness
↓
Aspiration of water

Effect of Type of Water Inhaled

Perhaps the former concept of fresh water and salt water drowning is now to be forgotten as it will have very little effect on survival of the individual.

Current Concept

In fresh water, as noted earlier, large volumes of water can pass through the alveolar capillary membranes. It can rather alter or denature the pulmonary surfactant, while sea water may dilute it or wash it away.[1,21] Presence of *chlorine* or *soap* in water apparently has no effect on this property.[21] Denaturisation is said to continue even after a person is successfully resuscitated. Loss or inactivation of pulmonary surfactant and alveolar collapse decreases lung compliance, resulting in profound ventilation perfusion mismatch with up to 75 per cent of the blood perusing non-ventilated areas.[1] When water is inhaled, vagal reflex cause increased peripheral resistance, with pulmonary vasoconstriction, resulting in pulmonary hypertension, leading to decreased lung compliance, and fall of ventilation perfusion ratio.[1] This can cause delay in return of and redistribution of blood perfusion to fully normal status even among successfully resuscitated and apparently healthy victims.

Older Concepts

Inhalation of water in drowning is followed by its absorption into circulation through semi permeable alveolar membrane leading to electrolyte imbalance with other consequences within few minutes of submersion. The extent and direction of exchange of water into blood through alveolar membrane will depend on osmotic gradient between blood and water, which usually depend on type of water in which drowning has taken place, i.e. fresh water or salt water. Pathophysiology of death due to drowning in *fresh water* and *salt water* is discussed below individually.

Fresh Water Drowning

Drowning in fresh water (no sodium chloride) or brackish water (with 0.5% sodium chloride) is characterised by rapid absorption of water into the blood stream because of descent or low salt in these waters. Thus, a large quantity of water crosses the alveolar membrane and enters into the circulation, causing in *hemodilution* and overloading of heart, hemolysis and release of potassium ions resulting in hyperkalemia. Both these factors can bring about *ventricular fibrillation* and *death* within 4 to 5 minutes due to heart failure**.** Figures 15.10 and 15.11 give the schematic illustrations and the actual mechanisms involved respectively.

Salt Water Drowning

Salt Here water that has entered the lungs, being hypertonic draws the water from the blood in pulmonary circulation due to the osmotic pressure effect. This results in *fulminating oedema* of the lungs, hemoconcentration, hypovolemia of blood, leading to asphyxia and circulatory shock, followed by cardiac standstill/ systole leading to death. Figures 15.12 and 15.13 give the schematic illustrations and the actual mechanisms involved respectively.

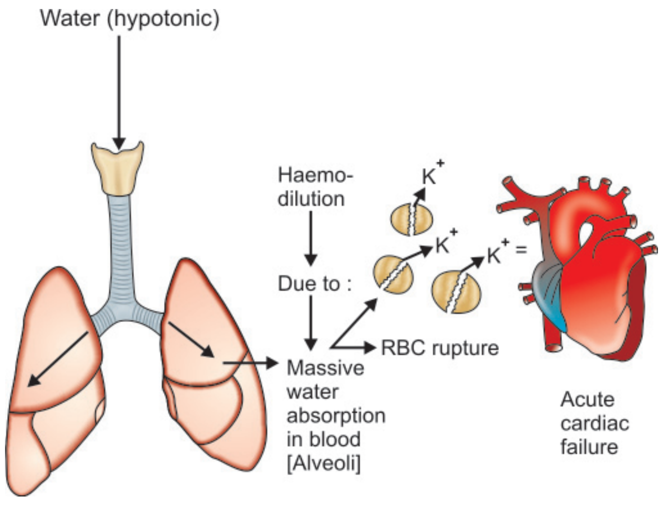


Fig. 15.10: Pathophysiology of death in fresh water drowning

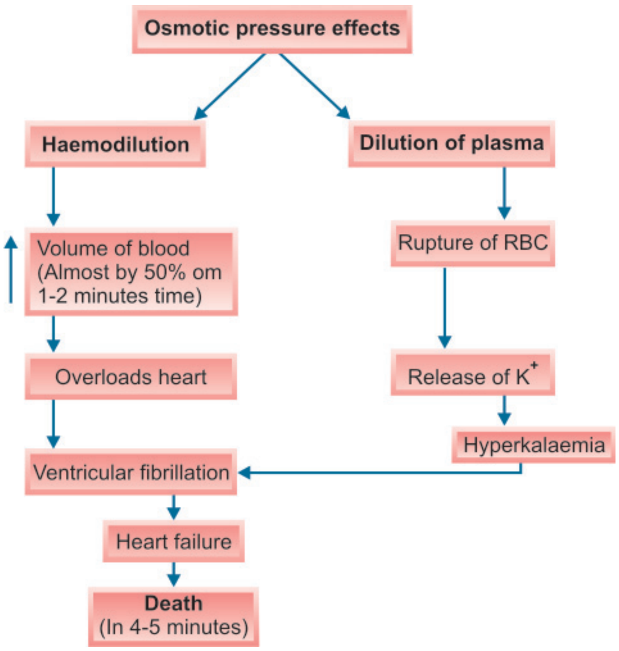

Osmotic pressure effects
Haemodilution
Dilution of plasma
Volume of blood (Almost by 50% om 1-2 minutes time)
Rupture of RBC
Overloads heart
Release of K+
Hyperkalaemia
Ventricular fibrillation
Heart failure
Death (In 4-5 minutes)

Fig. 15.11: Pathophysiology of death due to fresh water drowning

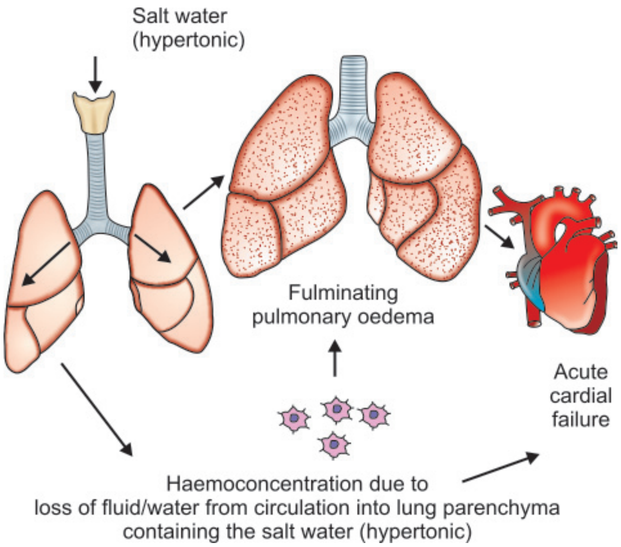

Salt water (hypertonic)
Fulminating pulmonary oedema
Acute cardial failure
Haemoconcentration due to loss of fluid/water from circulation into lung parenchyma containing the salt water (hypertonic)

Fig. 15.12: Pathophysiology of death in salt water drowning

Classification of Drowning

Drowning is classified into two major types: *Typical drowning* and *atypical drowning*.

i. Typical Drowning

This is also known as '*Wet Drowning*'. Here there is actual obstruction of the air passages by the fluid or water column entering into it.

ii. Atypical Drowning

Here there is very little or no fluid or water which is inhaled into the air passages. It includes four subtypes:

- *Dry drowning*
- *Immersion syndrome*

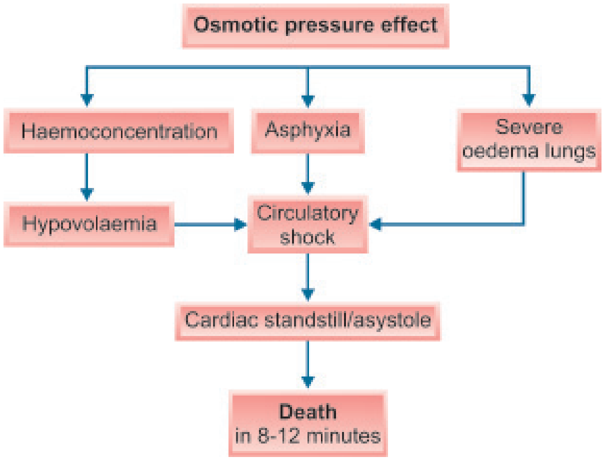

Osmotic pressure effect
Haemoconcentration
Asphyxia
Severe oedema lungs
Hypovolaemia
Circulatory shock
Cardiac standstill/asystole
Death in 8-12 minutes

Fig. 15.13: Pathophysiology of death due to salt water drowning

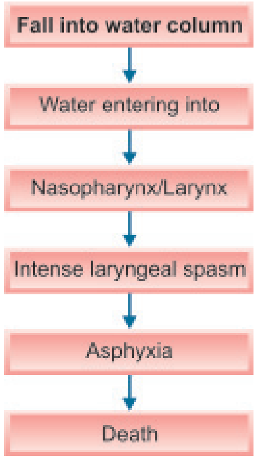

Fall into water column
Water entering into
Nasopharynx/Larynx
Intense laryngeal spasm
Asphyxia
Death

Fig. 15.14: Mechanism of death due to dry drowning

- *Submersion of unconscious*
- *Secondary drowning (Near drowning syndrome).*

Dry Drowning

Though this is a rarer entity; it is reported to account for 10-15 per cent of all drowning cases in west,[1] and the corresponding figure in India is 20 per cent.[9] Here no water is detected in the lungs during autopsy examination. Lungs remain dry and water-free.

Mechanism of Death

This condition is only a hypothesis and is yet to be proven.[1] Death in these cases is due to intense *laryngeal spasm* mediated as vagal reflex. Thick mucous, foam, and froth may develop, producing an actual *physical plug* at this point. Thus water never enters the lungs. During autopsy neither "*mucous plug*" nor the "*laryngospasm*" can be demonstrated, as death usually causes relaxation of musculature. However, according to DiMaio and DiMaio this could be one end of the spectrum of changes seen in the lung produced by occlusion of airway by water, with other end the heavy, boggy, lung containing a massive amount of oedema fluid.[1] Figure 15.14 illustrates the probable mechanism of death in dry drowning.

Immersion Syndrome (Hydrocution, Vagal Inhibition)

This also occurs rarely. It is usually found in temperate or cold zones. Where usually young swimmers are the victims. When they dive in very cold water, they may suffer from vagal inhibition of the heart and die sudden death in water, *even* though they may be good swimmers.[9] Mechanism of death involved in this type of atypical drowning is enumerated in the Figure 15.15.

Submersion of the Unconscious

This is often reported among those who suffer from conditions like epilepsy, cardiac diseases, etc. or those who are intoxicated, sustain head injury during fall into a water column, etc.

Mechanism of death: In all these conditions, there will be no efforts made by the victim to breathe/come out of water, as the person is unconscious. However, death is due to asphyxia. Figure 15.16 summarises the mechanism of the death due to submersion of unconscious.

Secondary Drowning (Near Drowning Syndrome)

This is occasionally encountered and refers to a submersion victim who arrives at an emergency facility and usually survives for 24 h.[29] However, here death may also occur in 30 minutes to several days of *being saved* from drowning. It is in these cases of near drowning that the physicians have been able to observe electrolyte changes. They have also reported that the electrolyte disturbances and hemoglobinemia are mild, if present at all, and rarely have any clinical significance.[21,30] It is common in children, elderly or sickly victims of drowning, who ultimately die because of the causes such as:

- Septic pneumonia
- Pulmonary oedema
- Chemical pneumonitis
- Metabolic acidosis.

In all cases of atypical drowning deaths, examination of the body discloses none of the usual signs of drowning.

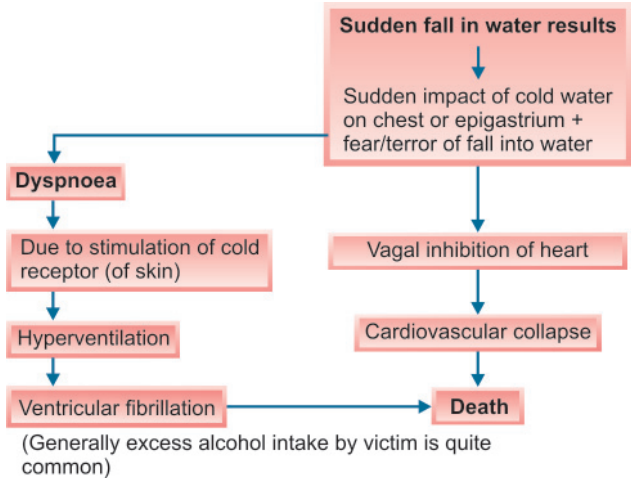

Sudden fall in water results
Sudden impact of cold water on chest or epigastrium + fear/terror of fall into water
Dyspnoea
Due to stimulation of cold receptor (of skin)
Hyperventilation
Ventricular fibrillation
Vagal inhibition of heart
Cardiovascular collapse
Death
(Generally excess alcohol intake by victim is quite common)

Fig. 15.15: Mechanism of death in immersion syndrome

POSTMORTEM FINDINGS OF DROWNING

Postmortem findings are described under *external* and *internal* findings (Figs 15.17 to 15.24).

External

Includes findings in—face, skin, hands and feet.

I. Findings in the Face

These are:

- Face is congested and livid
- Eyes
  - i. Palpebral fissure—open/closed (half)
  - ii. Conjunctivae—congested
  - iii. Pupils—dilated.
- Tongue - swollen and protruded out
- Mouth and nostrils - show *fine froth* collected around, which may be whitish, shaving cream lather like (Fig. 15.17). It may be blood stained also. The various events on formation of froth in the respiratory tract are described in the Figure 15.18.

Note: This froth being very light rises up in the respiratory passages and flows out of the mouth and nostrils–passively in a cadaver.

II. Findings in the Skin

The skin changes in general include *cutis anserine* and *postmortem lividity.*

Cutis anserine *(goose skin appearance):* Here the skin appears like an orange peel. This is due to the rigor mortis of erector pilorum muscles of hair follicles in the skin and subcutaneous tissues.

Postmortem lividity: It would be seen on dependent parts of the body. This solely depends on the position in which the body was floating.

III. Findings in the Hands

Common findings observed in the hands include *cadaveric spasm* and *washerwoman's hands.*

- ***Cadaveric spasm*** *(instantaneous rigor)*— Here the hands of the deceased would be clenched and on opening may show water, plants, seaweeds, etc. This is due to the struggle efforts finally made by the drowning victim to catch hold of whatever available in water and try to surface up. Thus, if a dead body removed from water presents the feature of *cadaveric spasm*, it is a finding in favour of antemortem drowning (Figs 15.19A and B).

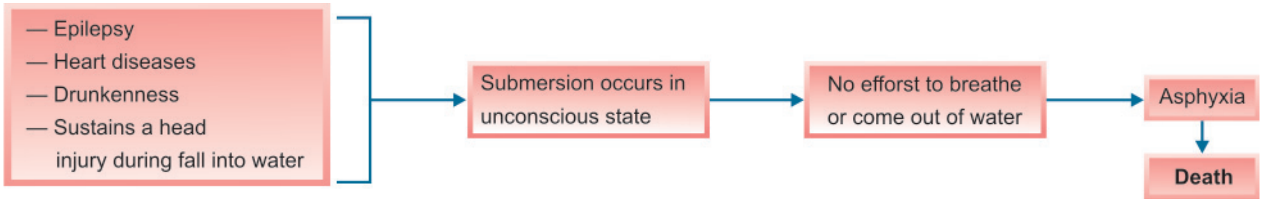

— Epilepsy
— Heart diseases
— Drunkenness
— Sustains a head injury during fall into water
Submersion occurs in unconscious state
No effort to breathe or come out of water
Asphyxia
Death

Fig. 15.16: Mechanism of death due to submersion of unconscious

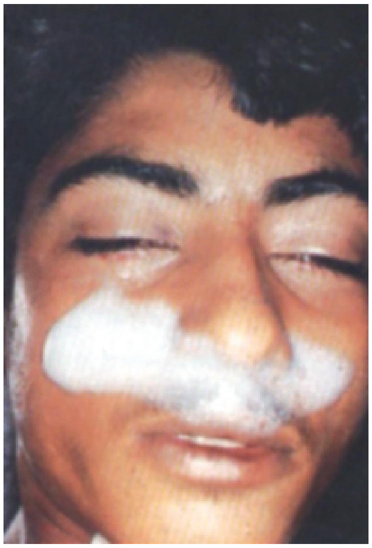


Fig. 15.17: Signs of antemortem drowning: Froth around nostrils

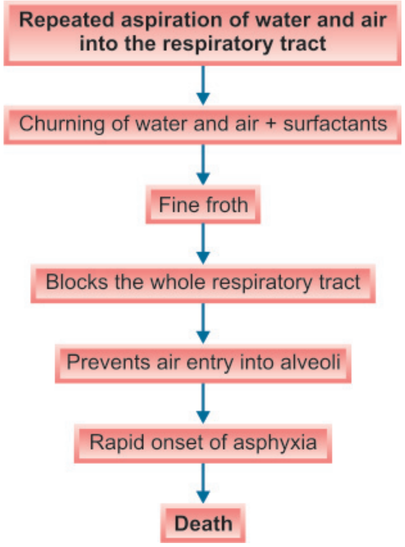
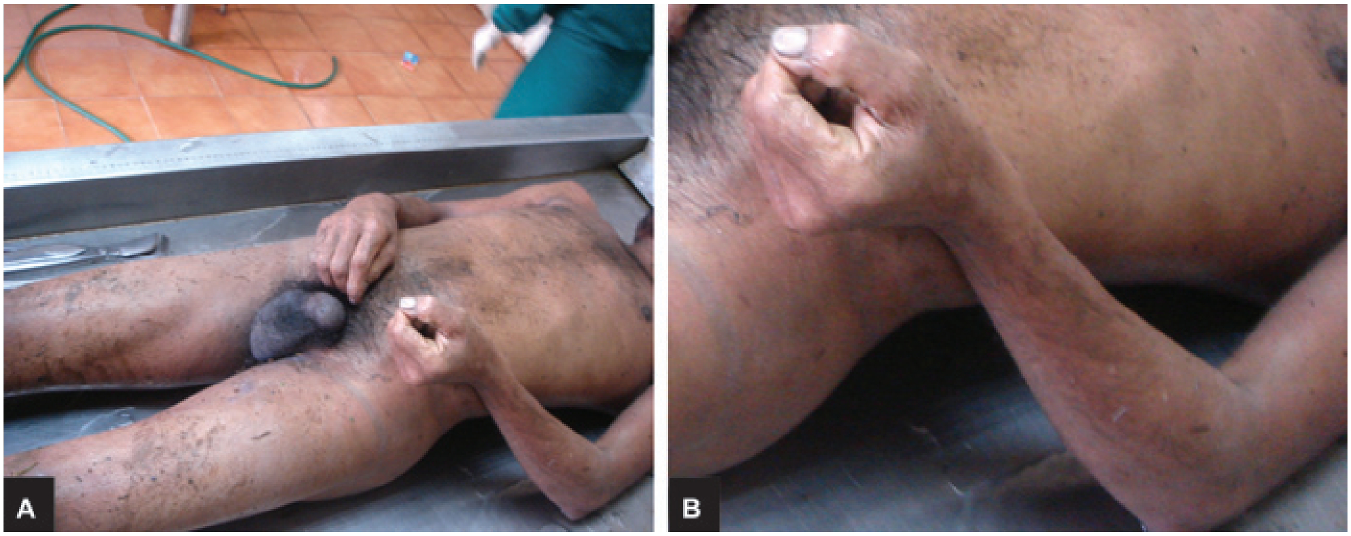

Repeated aspiration of water and air into the respiratory tract
Churning of water and air + surfactants
Fine froth
Blocks the whole respiratory tract
Prevents air entry into alveoli
Rapid onset of asphyxia
Death

Fig. 15.18: Mechanism of froth in the respiratory tract in an antemortem drowning case



Figs 15.19A and B: Cadaveric spasm in the left hand in a case of death due to drowning. (A) Vegetations on the body suggestive of removing from water. (B) Magnified view of cadaveric spasm (*Courtesy:* Dr Tanuj K, Asst Professor of Forensic Medicine, KMC, Mangalore)

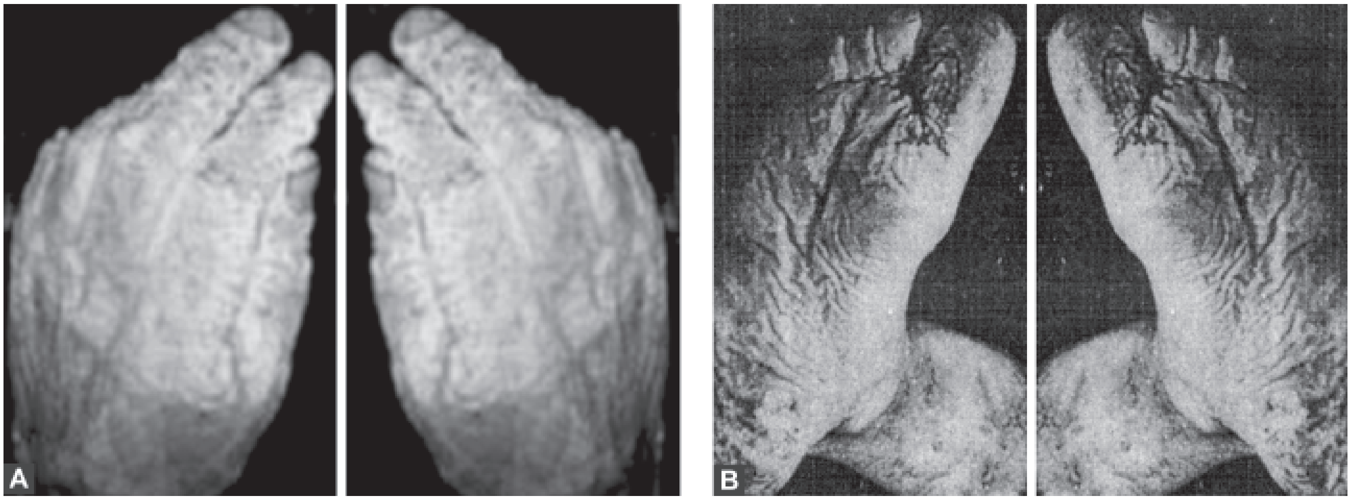
- ***Washerwoman's hands*** —A specific change that is seen in the skin of the palm and sole, which comprises of wrinkled, swollen, whitish *sodden appearance*, constitutes *washerwoman's hands* (Figs 15.20A and B). It is also considered as *water maceration* of skin of the hand, basically due to imbibation of water into its outer layers. The change however could help in detemining time since death as following sequence has been reported in its onset and complete formation.[4-6]
- It is first seen in the tips of the fingers usually by 3-4 hours of drowning.
- Entire hand will be involved, may be by 24 hours.
- Peeling of cuticle in about 12 hours
- Bleaching, corugation and soddening — in about 24 hours.
- Peeling of cuticle from palm and sole in about 48 hours; easity peeled off in about 3-4 days.
- ***Findings in the feet***—Skin of the sole of the feet may also present with similar changes as seen in the hands, constituting *washerwoman's feet* (Fig. 15.20).

Note: Though the *washerwomen's hand* and *feet* changes are seen invariably in dead bodies recovered from water, this may ***not*** be considered as a sign of antemortem drowning.

Rigor mortis—When initially recovered from water, the body may be in full rigor mortis, even though only a short time has passed from the time of the drowning. This is due to violent struggling at the time of drowning with decrease in ATP and rapid development of rigor mortis (Fig. 15.21).

Decomposition – Bodies cool much more rapidly in water than in air. Thus process of decomposition of bodies in water takes longer duration.

Leaching out of blood from antemortem wounds– Immersion of a body in water for several hours may cause leaching out of the blood from antemortem wounds. Thus a dead body may be found with a number of what may appear to be bloodless postmortem wounds which are actually



Figs 15.20A and B: Washerwoman's hand and feet (*Courtesy:* Spitz WU, Fisher RS. *Medicolegal investigation of Death*, Charles C Thomas Springfield, 1980)

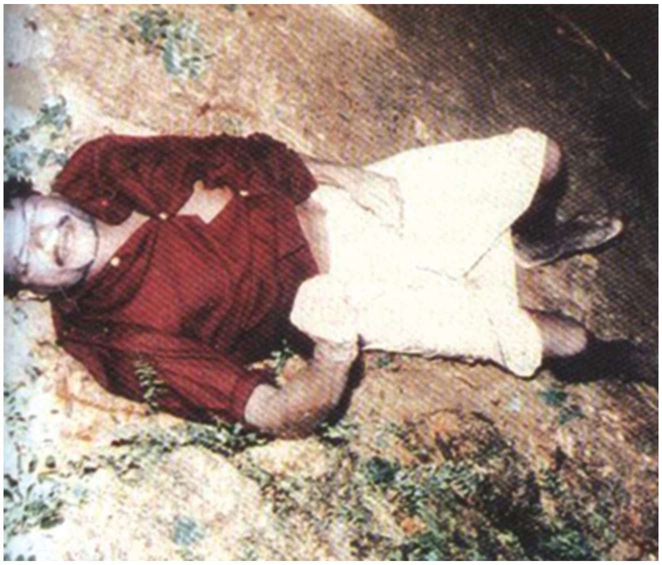


Fig. 15.21: *Signs of antemortem drowning:* Scene of crime—case of death due to drowning. Dead body removed from water (with froth around nostrils, cadaveric spasm of hand, washerwoman's hand and foot changes seen)

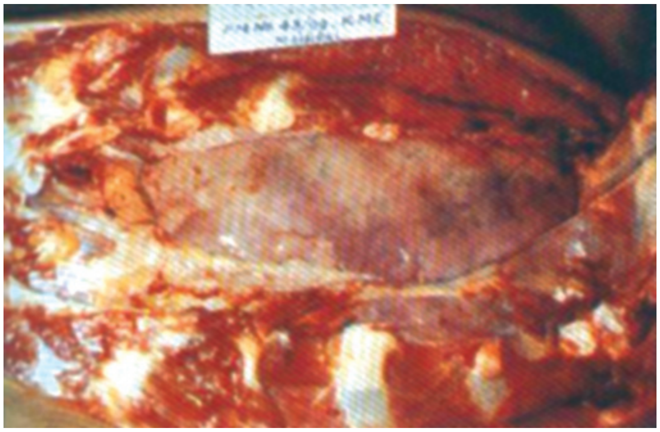


Fig. 15.22: *Signs of antemortem drowning:* Voluminous oedematous lung bulging out like balloon on cutting open of sternum

antemortem, and may be the cause of death. This artifact can cause problems when a body is pulled out of water with propeller cuts on it. These may look like postmortem injuries as there will be no bleeding from these wounds due to leaching out of blood, while in fact the injuries are antemortem.

Internal

All findings are in favour of asphyxia. Changes in the important viscera like *lungs, middle ear* and *blood* and in *other viscera* in favour of antemortem drowning are as follows:

Lung Findings

Findings vary in *typical* and *atypical* drowning cases.

I. Typical drowning cases *(Wet drowning cases)*— Change due to typical drowning is called *emphysema aquosum* and is described below:

- ***Gross findings*** – Lungs will be pale and grayish, voluminous, oedematous, and bulges out like balloon on cutting open the sternum (Figs 15.22 and 15.23).

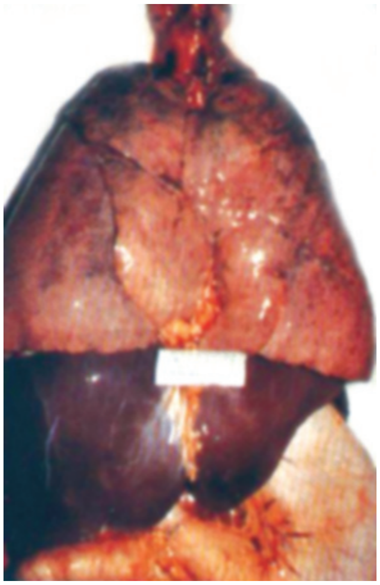


Fig. 15.23: *Signs of antemortem drowning:* Voluminous, oedematous lungs as seen on en masse removal of viscera in same case as in Figure 15.22

They will be heavy, boggy and doughy with a surface, which show *rib impressions* and *pits on pressure*. When a victim submerges consciously, violent respiratory efforts to breathe results in rupture of the alveolar walls, particularly underlining the pleura near the lower margins. The ruptured large alveolar spaces contain watery, thin, haemolysed blood and some amount of air and presents in the form of bullous lesions called *emphysematous bullae* (Fig. 15.24) and *Paultauf's hemorrhages*. Apart from this *Tardieu' spots* due to asphyxia may also be present.

- ***Cut section*** – Streaming out of fine froth, blood with sand, mud and slit particles in the trachea, bronchi and bronchioles are the usual observations in cut section of the lung, in favour of death due to drowning.

II. Atypical drowning cases

- In case of say a victim who is unconscious at the time of drowing the findings are known as *oedema aquosum*, which will never be the same as in typical drowning cases. Lungs may show little water in the respiratory passages flown in passively, but there will not be any pulmonary oedema/other findings as described under *emphysema aquosum*. It may be noticed here that when a dead body is thrown into water, water can trickle into the lungs, the condition called 'hydrostatic lung', but the picture can never be of emphysema aquosum.

Middle Ear Findings

In a case of antemortem drowning there will be water in the middle ear.

Mechanism – Normally no water can get into the middle ear. While in drowning due to the violent attempts made by the victim to breathe in air, water also gets aspirated with air into the naso/oropharynx, which forces little water into the middle ear also.

Medicolegal importance – Demonstration of air in the middle ear at autopsy is suggestive of antemortem drowning.

Haemorrhage in Petrous Temporal and Mastoid Bone

This again, is nonspecific and if seen, can be found in individuals dying of heart disease, drugs overdose, or such other causes. Thus, the drug overdose victim dumped in water or the heart attack victim is collapsing into water, can have the 'washerwoman' appearance of the palms and soles, 'goose flesh', pulmonary oedema, and hemorrhage into the petrous temporal and mastoid bones.

Changes in Heart

The right ventricle may show dilatation. Changes in the heart basically refer to *biochemical changes* in the heart blood. The most popular Gettler's Chloride Test, was developed based on these changes. Accordingly blood was analysed from right and left side of the heart. If the chloride level was less in right side, compared to left side, the person was assumed to have drowned in salt water. If the level was elevated on right side of the heart, in comparison to left side, then one was thought to be died of fresh water drowning. Tests have been done for other elements in blood as well as comparing specific gravity of blood in right versus left atria. All of these tests are presently considered obsolete.[1]

Findings in Other Viscera

Stomach: May contain the water swallowed along with the various contents in it, such as mud, algae (diatoms), planktons, etc. detected microscopically. Brain, liver, spleen, bone marrow may also show the presence of algae (diatoms), planktons, etc. A specific test *Diatom test* is specially designed for the purpose.

Diatom Test

This is a more exotic, though controversial test which involves the identification of diatoms in tissues of a drowning victims.

Principle: Diatoms are microscopic unicellular algae (Fig. 15.25) which live in water. They vary in size (5 to 400 μm) and shape and have got a hard-cell wall made of silica, which resists acid digestion. They are seen in both sea and fresh water. They enter systemic circulation via lungs and reach various organs and remain there. Thus, their presence in these viscera is suggestive of antemortem drowning. However, others say that it is not possible to come to this conclusion due to wide spread distribution of these organisms throughout the environment.[1]

The question also revolves around whether diatoms are normally present in human organs, if present what are the types present. Researchers have analysed lung, liver, kidney, and bone

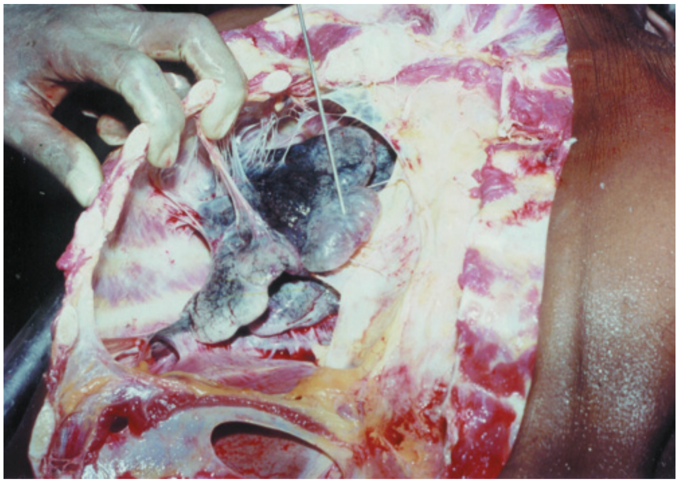


Fig. 15.24: Gross findings of lungs of a victim antemortem drowning. Note the probe pointing at the emphysematous bullae (*Courtesy:* Capt. Dr. Santha Kumar, Professor and HOD, Dept of Forensic Medicine, Govt. Kilpauk Medical College, Chennai, TN).

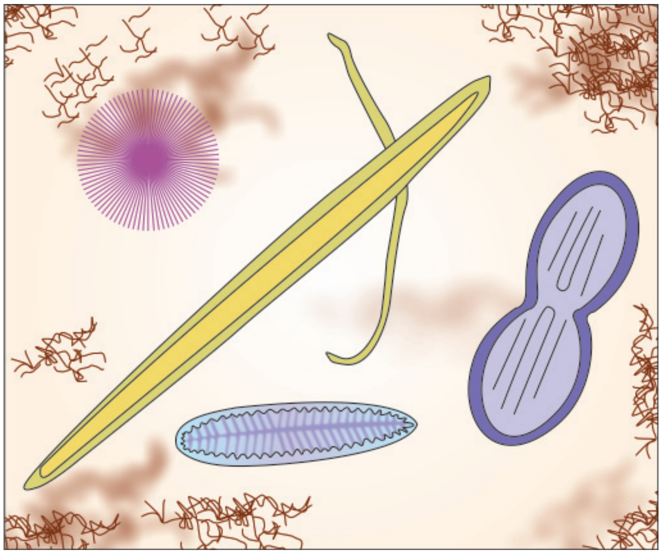


Fig. 15.25: Diatoms

marrow for diatoms and have come to conclusions based on the presence or absence of these organisms. Some workers have found diatoms in the organs of non-drowning cases, while others have not.

If diatoms are present in a body, there are three possible ways they could enter there. First is by inhalation of airborne diatoms, second is by ingestion of material containing diatoms, and third is by aspiration of water containing diatoms, with subsequent circulation of these throughout the body. Complicating all this is the fact that diatoms are so ubiquitous that part of the problem with some of the analyses may have been contamination of the glassware and reagents by diatoms.[1]

Procedure

It includes several steps and are enumerated in the schematic representation as shown in Figure 15.26.

It must be remembered here that at present times, if diatom test is to be used for diagnosing drowning death, it should fulfill following five criteria:

1. Diatom analysis is done in closed organ system, such as femoral bone marrow or an encapsulated kidney from a non-decomposed body.
2. Contact of the sample with water is limited to triple distilled water.
3. The instruments are specially cleaned to prevent contamination with diatoms.
4. The deposit is examined with standard microscope for presence of the diatoms.
5. The water in which the individual has allegedly drowned is sampled to see what type of diatoms are present and a comparison is made between that in the water and those found in the body.

Inference

- If diatoms are present, drowning is confirmed as antemortem.
- However, while a positive comparison is helpful, a negative result does not rule out drowning.
- Even a completely negative analysis for diatoms does not rule out drowning.

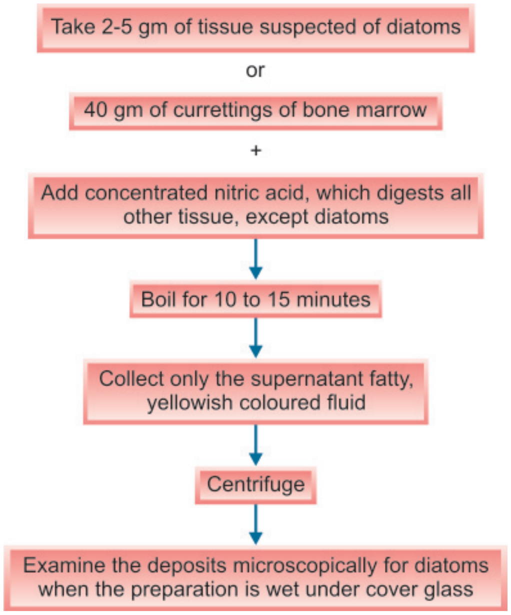

Take 2-5 gm of tissue suspected of diatoms
or
40 gm of currettings of bone marrow
+
Add concentrated nitric acid, which digests all other tissue, except diatoms
Boil for 10 to 15 minutes
Collect only the supernatant fatty, yellowish coloured fluid
Centrifuge
Examine the deposits microscopically for diatoms when the preparation is wet under cover glass

Fig. 15.26: Procedure of diatom test

Medicolegal Aspects of Drowning

1. At autopsy there are no pathognomonic findings to make the diagnosis of drowning.
2. The diagnosis is based on the circumstances of death, plus a variety of nonspecific anatomical findings.[1]
3. Chemical tests put forth to make the diagnosis are nonspecific and essentially unreliable.
4. If an individual is found dead in water and all other causes of death have been excluded, he is presumed to have drowned.
5. It must be remembered that people who have had fatal heart attacks and fallen into water, and that disposal of a victim of a fatal drug overdose in water is not unknown.
6. Attachment of heavy weights to the body to keep it under water is consistent with both homicidal and suicidal drowning as well as disposal of a body of person who died of some other cause.
7. *Position and floatation of a dead body in drowning* – when a person drowns, the body sinks, assuming a position of head down, buttocks up, and extremities dangling downward. Unless there are strong currents, the body will not move very far from its initial position. In relatively shallow water, the extremities or face may bump or drag against bottom of water column. Postmortem injuries to the face, back of the hands, knees, and toes may be present due to dragging along the bottom. The crown of the head and the buttocks may be at water level. In deeper water, the body stays below the surface until decomposition begins and gas formed, the body then gradually rises to the surface. Occasionally, the issue of time interval the body takes to appear on the surface water, may become an issue and important. In other words, floatation of body in water may also help in crime investigation, especially in deciding time since death. Despite of several factors such as specific gravity of human body, body fat, age, sex, prior physical status etc, discussed elsewhere above, in India, floatation of the body occurs in summer by about 24 hours and in winter by 2-3 days of drowning death.[4-6,9]
8. Depending on how long the body was in the water, there may be evidence of animal activity, for example, fish, turtles, crabs, or shrimps. In very cold water, body may stay submerged for months before decomposition produces enough gas for it to rise to the surface.
9. *Drowning in bath tubs* – relatively uncommon. Usually involves young children unattended by parents. Some are undoubtedly homicidal as well. If, while taking bath, one's feet are grasped and one is pulled underwater by them, there may be sudden flow of water into nasopharynx. This in combination with panic and being in smooth walled, wet, slippery container may result in inability to struggle effectively with rapid loss of consciousness and death. No injuries may be seen at autopsy. Overdose of drugs, seizure disorder, heart attack are some of the causes that can drown adults accidentally. There are well documented reports where victims slipped deliberately (homicidal) or accidentally in the bathtub, struck their head, and drowned.[1] R. v/s Smith case famous as *'Brides of the Bath case'* is a typical example of such incidence, wherein not less than three newly married girl brides were drowned in the bath tub by the bridegroom, by suddenly lifting the legs up and pushing the head under water, and immersing without any traume anywhere, giving no scope for investigating officer to suspect and agree that

each case was accidental death. Suspicions arose on chance by reading in the news paper of series of similar accidents, involving same man (bridegroom). In each case Mr Smith got his three victims (who were exceptionally rich) and drew up wills in favour of him ofter marrying.[4]

10. *Scuba Divers* – deaths can occur with use of scuba diving equipment, may be caused by natural disease, as a consequence of being under water at increased pressure. It could also result due to defective equipment. If a diver too rapidly does an ascent to surface it may cause air embolism, pneumothorax, or interstitial emphysema. Divers may die also because of getting trapped in caves or under water debris. Contamination of air within the tank may also kill the diver. Hence it is suggested that in every scuba diving related death, the equipment used must be examined thoroughly by an expert with analysis of residual air in the tank.[1]

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Part IV: Clinical Forensic Medicine

Chapter 16

Trauma, Injury and Wound

Trauma has always been a part of human existence and, particularly in industrial countries, has increased in significance. Inspite of safety legislations and attempts to raise public awareness of the problem, accident rates remain high. Although modern medicine is able to deal with many of the dangerous complications, trauma still represents a more or less serious risk to health, reducing work efficiency and costing money.

Trauma means a wound or injury, especially produced by external force. A significant percentage of all deaths occur following a traumatic episode, therefore obviously necessitating the need of a thorough knowledge on trauma. According to United State's Centre for Disease Control and Prevention (CDC), 74 per cent of deaths in USA, is due to trauma and it occurs in the age group of 10 to 24 years.[1] A comparative data reported by National Crime Record Bureau (NRCB) in India is 62 per cent of all deaths and it occurred in the age group of 15 to 44 years.[2] In practice the terms, *trauma, injury, wound* and *hurt* are used as synonyms. However, as per the law in India the term *'hurt'* means *'whoever causes bodily pain, disease or infirmity to any person is said to have caused hurt' (IPC Section 319).*[3]

Definitions

Trauma or injury is defined in two different ways, *legally* and *medicolegally* or *clinically.*

Legally

Trauma or injury is defined as "any harm whatsoever in nature illegally caused to the body, mind, reputation, or property" *(IPC Section 44)*.[3]

Medicolegally (Clinically)

Trauma or injury is defined as breach or dissolution of the natural continuity of any of the tissues of a living body by actual physical violence.[4-6]

CLASSIFICATION

Trauma is accordingly classified in two ways, i.e. legal and medicolegal.

Legal Classification

Depending on the *legal aspects*, trauma includes *two types* and they are:

- Simple
- Grievous.

Medicolegal Classification

Depending on the *causative factor,* medico-legally trauma includes *five types* and they are:

- Mechanical
- Thermal
- Chemical
- Electrical/lightning
- Radiation.

Of all these, *mechanical trauma* is discussed first for the reason that it is most commonly encountered in routine medical practice. Others are dealt separately. Chemical injuries comprise integral part of toxicology section.

Classification Depending on Order of Importance

Depending on order of importance, trauma, referring in particular to severe trauma, is also classified as following types:[7]

- Accidents at home
- Accidents at work
- Road traffic accidents (RTA)
- Sports injury
- Homicide and suicide.

Within these groups one finds injuries caused by blunt objects, by sharp objects and, by firearms. Thermal injuries and the effects of electricity are also commonly seen. However, for the purpose of *better understanding* the *mechanical trauma* is discussed in detail.

MECHANICAL TRAUMA

All injuries sustained due to physical violence to the body constitute *mechanical trauma* or *mechanical injury*. Possible *mechanisms* of mechanical trauma and classification of mechanical trauma are discussed here.[4-9]

Mechanism

Usually there are *two mechanisms* encountered and they are: *impact of a moving object and impact of a virtually non-moving object on actively moving victim.*

Impact of a Moving Object

Here the trauma is due to the impact of a moving object on a relatively non-mobile victim, *e.g. a cricket ball hit by a batsman strikes a spectator watching the game.*

Impact of a Virtually Non-moving Object on Actively Moving Victim

Here the mechanical trauma is due to the impact of moving victim who directly hits against a non-moving object, *e.g. a scooter driver collides with an electric pole.*

Classification

Mechanical trauma is further subclassified into *blunt force trauma, sharp force trauma and firearm injuries.*

Blunt Force Trauma

It is injury produced by weapons or objects with blunt edges or surfaces. Basically they are:

- Abrasion
- Contusion
- Laceration.

Sharp Force Trauma

It is injuries produced by weapons or objects with sharp cutting edge or edges. Basically they are:

- Incised wound
- Stab wound
- Chop wound.

Firearm Injuries

These are injuries produced by firearms. They are further classified into:

- Injuries by rifled firearms *(gunshot injuries)*
- Injuries produced by smooth bored firearms *(shotgun injuries)*
- These constitute a major subdivision under trauma, and technically entitled as *forensic ballistics*, which is dealt separately in this book for the sake of conveniences and easy understanding.

ABRASION

Definition

An abrasion is defined as a superficial injury, inducing displacement of only epidermis in the skin by friction.

Causes

Various causes leading to development of an abrasion are:

- Fall on a rough surface
- Blow with blunt weapon
- Dragging by a vehicle
- Hurt by fingernails, thorns, teeth bite, etc.

Clinical Findings

Lesion produces minimum bleeding, heals rapidly within seven days and leaves no permanent scarring on healing.

Classification of Abrasions

Various types of abrasions encountered routinely (Figs 16.1A to E) *include scratches, grazes, brush burns, rope burns, and impact abrasions.*[6,9]

Scratches

These are linear injuries due to a sharp object, e.g. pin, thorn, fingernail, etc (Fig. 16.1A).

Grazes

Sliding, scraping, or grinding abrasion These are injuries due to frictional force of rubbing by a blunt object moving with great force, e.g. shoe kicks, dragging on the rough road by a vehicle, etc. (Fig. 16.1B).

Brush Burns (Brush Abrasion, Tangential Abrasion)

Brush burns are grazes involving wider area due to the frictional force of rubbing against a rough surface and resembles a burn injury, e.g. as in dragging on the ground in a road traffic accident. Lesion when examined by a magnifying lens shows a serrated margin at the point of commencement and heaped epithelium at the other end, with grooves and furrows in between, which helps in deciding the direction of wound[6,9] (Fig. 16.1C).

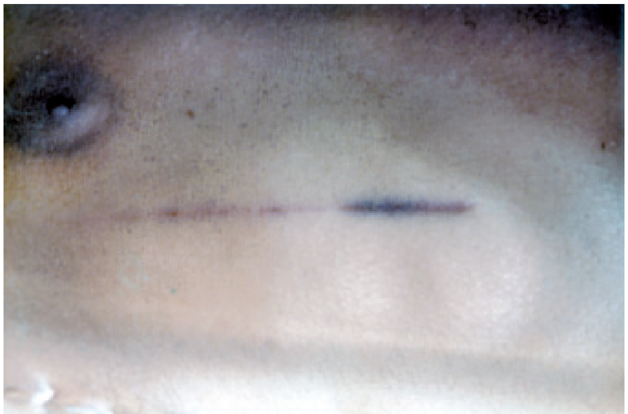


Fig. 16.1A: *Types of Abrasions:* Scratch abrasion (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

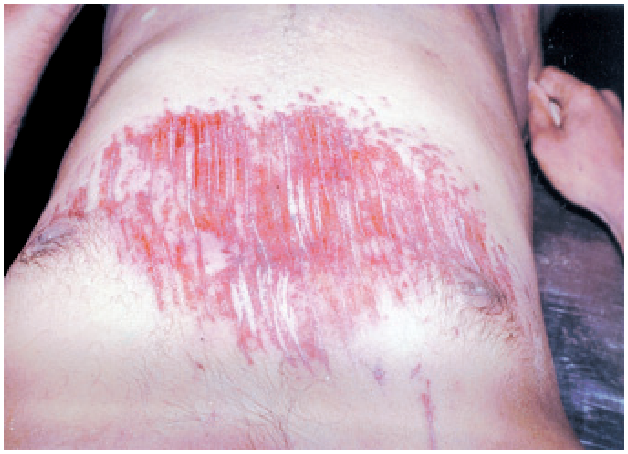


Fig. 16.1B: *Types of Abrasions:* Graze abrasion (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

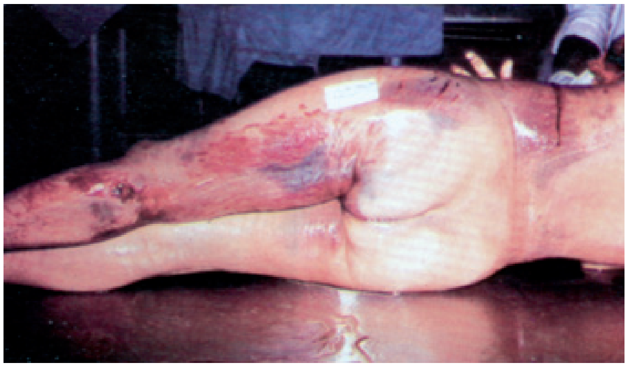


Fig. 16.1C: *Types of abrasions:* Brush burns (with contusions bruises)

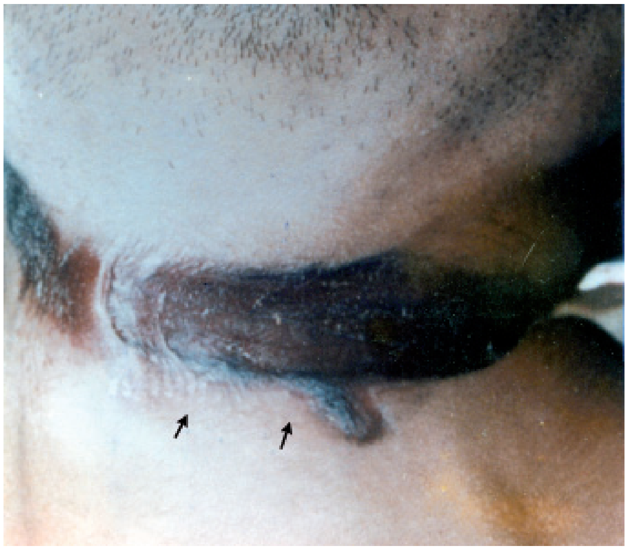


Fig. 16.1D: *Types of abrasions:* Rope burns (Ligature mark of hanging—Arrows) *(Courtesy:* Dr SC Mestri, Professor and HOD, Forensic Medicine, JSS Medical College, Mysore, Karnataka)

Rope Burns

Rope burns are due to heat produced by frictional force of a rope against the skin. This results in blisters due to expression of tissue fluids into the upper layers of the skin (Fig. 16.1D).[9]

Pressure Abrasion (Friction Abrasion, Crushing Abrasion)

This will be caused by direct impact or linear pressure of a rough object over the skin accompanied with slight movement directed inwards resulting in crushing of superficial layers of the cuticle with some bruising underneath. This type of abrasion will be found in ligature marks in hanging and strangulation (Fig. 16.1E), in case of striking by a whip or lashes, this is also noticed in tender skinned young children along the friction areas under pressure of garments, etc. These friction abrasions on getting dried up look brown and parchmentised.

Impact Abrasions (Imprint Abrasion, Contact Abrasion, Patterned Abrasion)

Impact abrasions are due to direct impact or pressure of some object, which on crushing the cuticle casts a reproduction of its shape and surface marking upon the skin, e.g. gravel marks, (Fig. 16.1F) radiator grill mark, tyre treads marks (Fig. 16.1G), nail and thumb markings in throttling, teeth marks in biting, whip marks on beating with a whip, muzzle marks in gunshot injuries, etc. Imprint abrasion becomes more defined, when injured cuticle dries up and becomes brownish and parchmentised, in contrast with the surrounding uninjured skin surface.

Age of Abrasion (Time since Injury)

Though an exact time or age of the wound cannot be assessed, an approximate time range since injury can be determined by noting the following changes in the wound.[6,7,10]

- Bright red lesion—freshly produced
- Blood and lymph, which dries up and forms scab—12 to 24 hours
- Scab turns brownish—2 to 3 days
- Scab dries, shrinks and falls off—7 days.

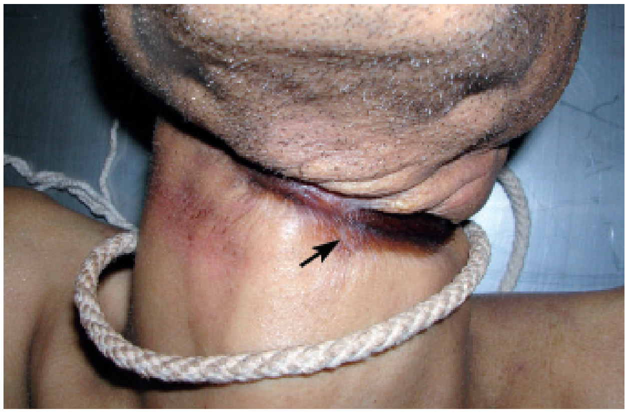


Fig. 16.1E: *Types of abrasions:* Pressure abrasions (hanging ligature mark with intact ligature material)

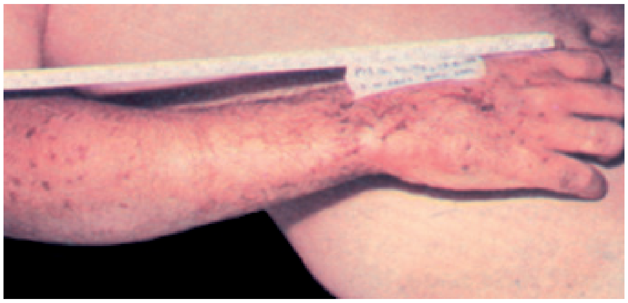


Fig. 16.1F: *Types of abrasions:* Imprint abrasions due to gravels

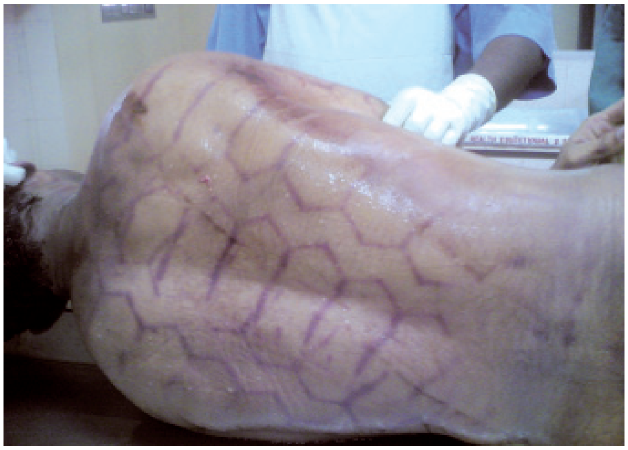


Fig. 16.1G: *Types of abrasion:* Imprint/impact abrasion-tyre tread marks (*Courtesy:* Dr KR Nagesh, Assoc Professor, HOD Forensic Medicine, Father Muller's Medical College, Mangalore, Karnataka)

Differential Diagnosis

An abrasion may be mistaken for:

- Postmortem abrasions
- Postmortem injuries *(erosions)* produced by insects/ crustaceans
- Excoriation of the skin by excreta (fecal matter)
- Pressure sores.

Postmortem Abrasion

Though it grossly looks like antemortem abrasions, it is different when examined closely (Table 16.1). However in doubtful situations, a *histopathological* examination will confirm.

Table 16.1: Differences between antemortem and postmortem abrasion[6,9,11]

Features	*AM abrasion*	*PM abrasion*
Site	Anywhere on body	Only at bony prominence
Colour	Bright red	Yellowish, parchment like
Exudate	More and scab raised	Less and no scab
Microscopy	Vital reaction +ve	Vital reaction –ve

Postmortem Injuries (Erosions, Pseudoabrasions)

Insects and crustaceans produce these. Seen especially at mucocutaneous junctions like around eyes, nostrils, mouth, anus, armpits, genitalia, etc.[2-6] (Figs 16.2A to E)

Excoriation of the Skin by Excreta (Fecal Matter)

These are due to contamination of the skin by dried up fecal matter, and can be washed with water.[4-6]

Pressure Sores

These are also known as *bedsores.* These are due to pressure necrosis of the skin in a person who is bedridden for long-time and not under proper care (Fig. 16.2E).

Medicolegal Importance of Abrasions

Different opinions that can be derived from the abrasions are as follows:[6,11]

- Can provide clues on *site of impact and direction of force*
- Can be the only *external sign* of a severe internal injury
- Can help to identify *causative weapon*, e.g. imprint abrasions
- Can help to determine *time since injury*
- Can help to assess *the motive*, e.g. ligature mark of hanging, strangulation, etc
- Abrasions may also be developed on a cadaver (postmortem abrasion).

CONTUSION (Bruise, Haematoma)

Definition

A contusion is a haemorrhage into the skin, or into the tissues under the skin or both, as a result of rupture of small blood vessels, especially capillaries by a blunt impact.

Causes

These injuries can be produced by blunt force with fist, stone, stick, bar, whip, cane, shoes, etc.

Clinical Findings

Examination of a Bruise

Under this a mention must be made about the colour, site, size, shape, etc. of a bruise.

Lesion

A bruise usually shows no external bleeding, heals completely in 15 days leaving no permanent scars. However, contusions of the viscera such as brain, heart, liver, lungs, etc. could be fatal.

Classification of Bruises

Basically bruises are of three types[6,9,11] and they are *superficial, deep and patterned bruises.*

Superficial Bruises

These are usually seen raised above the surface of the skin *(swelling)* due to infiltration of blood into the subcutaneous tissues (see Fig. 16.1C).

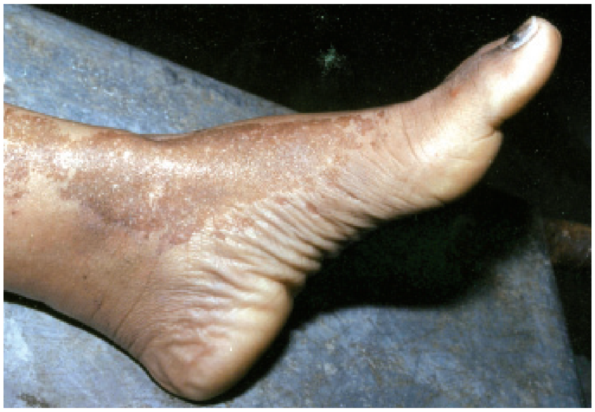


Fig.16.2A: *Pseudo abrasions:* PM injuries by ants (*Courtesy:* Dr Dias Sepeco SCA, Professor and HOD, and Dr Rodriguez EJ, Professor, Dept. of Forensic Medicine, Goa Medical College, Bambolim, Goa)

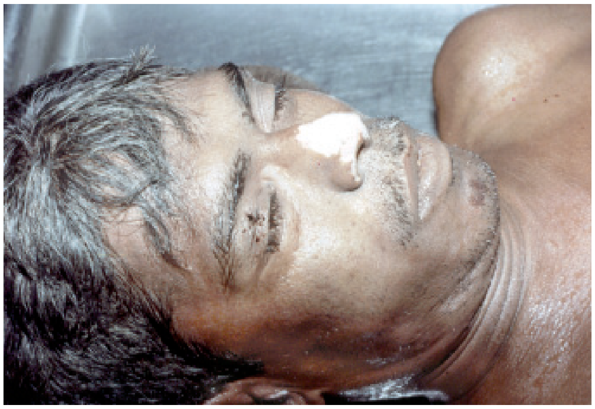


Fig. 16.2B: *Pseudo abrasions:* PM injuries by ants (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

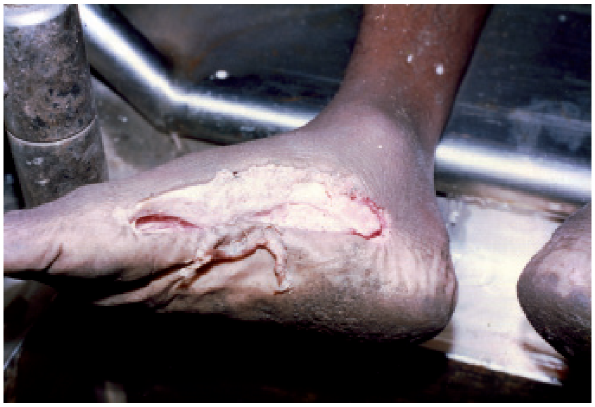


Fig. 16.2C: *Rodents gnawing:* PM injuries (*Courtesy:* Dr Santha Kumar, Professor and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

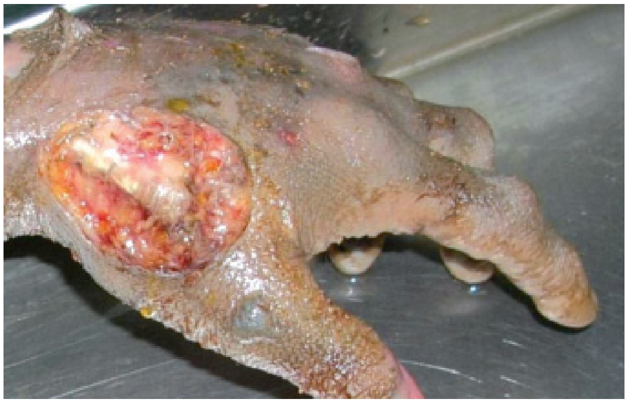


Fig. 16.2D: *Gnawing by aquatic animals:* PM injuries

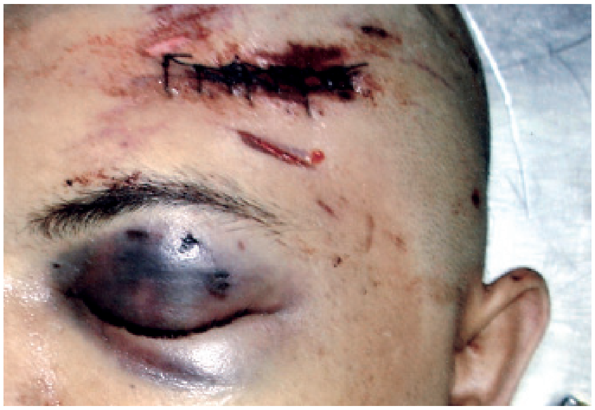


Fig. 16.3A: Black eye

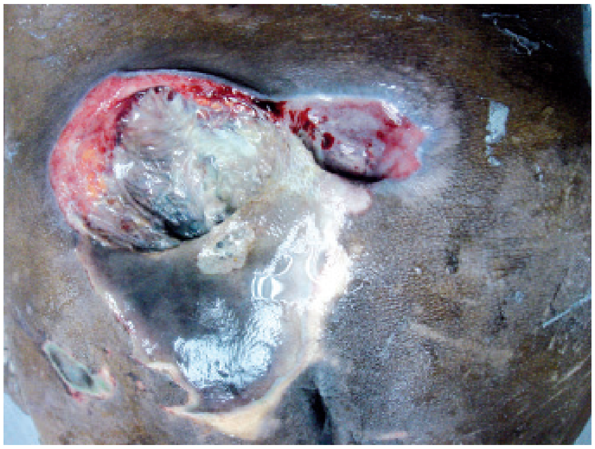


Fig. 16.2E: *Pressure sore/bed sore* in the sacral region (*Courtesy:* Dr Ritesh G Menezes, Assoc. Professor of Forensic Medicine, KMC, Mangalore, Karnataka)

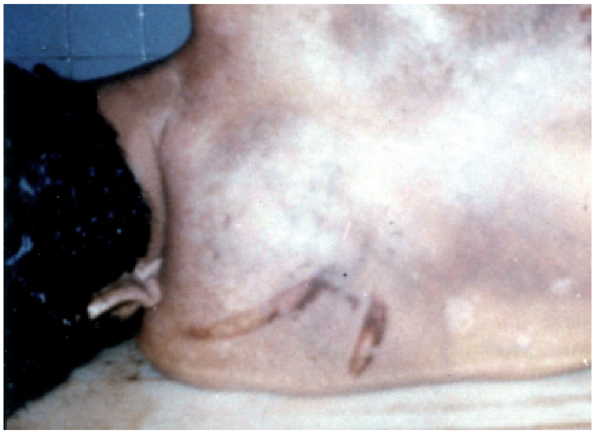


Fig. 16.3B: *Types of bruises:* 'Tram-line/Railway-line' bruising on the back and left shoulder. (*Courtesy:* VV Wase, Professor and HOD, Forensic Medicine, Grants Medical College, Mumbai, MS)

Deep/Delayed/Migratory/Ectopic Bruises

These may also produce a mild swelling and may take more time to appear externally than superficial bruises. They may also be found in a different location than actual site of injury, e.g. black eye (Fig. 16.3A).

Patterned Bruises

These are bruises that take round or oval shape. Sometimes it could also take up the shape of the causative weapon used. Such bruises are called patterned bruises, e.g. 'Tram-line' or 'Railway-line' bruise (Fig. 16.3B): this comprised of two parallel linear bruises with an unbruised area inbetween resulting from a rod/shok.

Mechanism: When a rod/whip etc in used to hit, skin with blood vessels underneath yeilds to the pressure at the edges and ruptures bleeding at the edges of the impacting object resulting in parallel bruises.

Factors Affecting Bruising

Various factors, which affect formation of a bruise, are:[6,11,12]

- Type of tissue/site involved
- Age
- Sex
- Colour of skin
- Natural diseases
- Gravity shifting of blood, etc.

Type of Tissue/site Involved

More vascular part such as vulva, scrotum, etc. can develop big bruises with little force. Stronger part such as palm, sole, etc. develop smaller bruises with great force.

Age Factor

Children and elderly people can be more easily bruised than young adults.

Sex Factor

Females can be more easily bruised than males.

Colour of Skin

Fair skin individuals develop bruises clearly which are easily recognised.

Natural Diseases

In persons suffering from certain diseases such as atherosclerosis, purpura, leukemia, scurvy, etc. bruising is easy.

Gravity Shifting of Blood

This factor is many a times responsible for the appearance of bruises at a site other than site of injury, e.g. *black eye*—blow/fall on forehead can cause bruising around the eye and *kick on the calf* can result in *bruising around the ankle. Thus, site of bruise does not always indicate the site of injury.*

Age of the Bruise (Time since Injury)

Time since injury can be assessed usually for superficial visible bruises[4,5,6,8,13] than deep bruises. Though exact time or age of the wound cannot be assessed, a rough estimate can be done by colour changes in the bruises, as presented in Table 16.2.

For Deeper Bruises

It is difficult to assess the age, as they take much longer duration to appear.

Differential Diagnosis

A true bruise may have to be differentiated from *artificial bruise, postmortem bruise* and *postmortem lividity.*

Artificial Bruises (Fabricated wounds)

These are skin lesions produced by the application of certain irritant substances such as plumbago rosea-roots/twigs of the plant and marking nut juice over skin, which resembles a bruise apparently (Fig. 16.3C). However, differences between them noticed by close examination are given in Table 16.3.

Postmortem Bruising[5,6,12,14]

Bruises cannot be produced after 2 minutes of death. However, small bruises can be developed by great violence even up to 3 hours of death. The characteristics by which they can be differentiated are given in Table 16.4.

Can Be Misinterpreted as Postmortem Lividity

Table 16.5 presents the differences between them.

Table 16.2: Bruise and time since injury

Colour change	*Time since injury*
Bright red (Swoolen tenden)	Freshly produced
Bluish	Few hours to 3 days
Bluish black/brown	4th day
Greenish	4th to 5th days
Yellowish	7th to 12th days
Normal	2 weeks

Table 16.3: Differences between artificial and true bruise

Characteristics	*Artificial bruise*	*True bruise*
Cause	Irritant chemical	Trauma (blunt)
Site	Accessible parts	Anywhere
Colour	Dark brown	Typical changes
Shape	Irregular	Regular
Margin	Regular + vesicles	Nil
Inflammation	+ve	–ve
Itching	+ve	–ve
Vesicles under nails	+ve	–ve
Ecchymosis	–ve	+ve
Content	Acrid serum	Blood
Chemical test	+ve	–ve

Table 16.4: Differences between antemortem and postmortem bruises

Characteristics	*AM Bruise*	*PM Bruise*
Swelling	+ve	–ve
Damage to epithelium	+ve	–ve
Extravasation and inflammation of blood in tissue	+ve	–ve

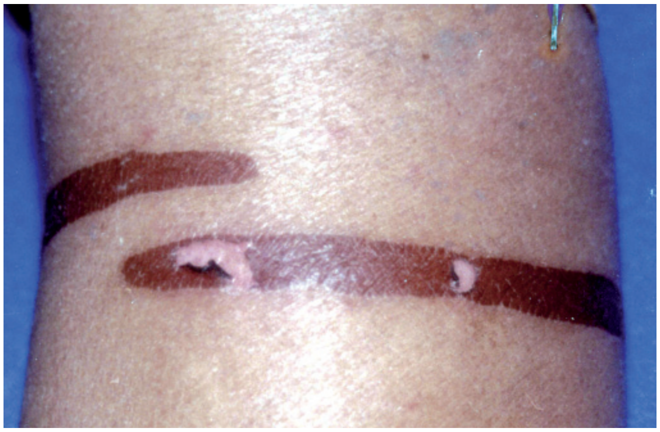


Fig. 16.3C: Artificial/fabricated bruise, by applying crushed twigs of Plumbago rosea plant. (*Courtesy:* Dr SC Mestri, Professor and HOD, Forensic Medicine, JSS Medical College, Mysore, Karnataka)

Medicolegal Importance

Bruise is of Lesser Importance than an Abrasion

Reasons for this are:

- Size of a bruise may not correspond to the size of the weapon used
- Bruise may appear late after injury
- Bruise may appear in far away regions of the body from the actual site of injury.

However Contusion can Help Law and Criminal Investigation

It can help in assessing certain facts such as:[6,11]

- Causative weapon
- Time since injury
- Degree of violence
- Characteristics and manner of injury, e.g. bruising of inner aspects of thighs or genitalia is suggestive of rape or such other sexual offences, bruising of shoulder blade are suggestive of assault or struggle.

LACERATION (Tears, Gashes Ruptures)[4-6,9,15,16]

Definition

A laceration is a disruption of the continuity of tissue, produced by stretching or crushing type of blunt forces.

Causes

Tearing up of tissues by blunt force, e.g. splitting of the skin by the overstretching on fractured bones underneath, crushing of skin between two hard objects, etc.

Clinical Findings

Lesion

Lacerations will have the following features:[15]

- *Margins* – irregular with pieces of tissues attached in between, called *tissue tags* or *bridges*
- *Bruising* – seen around the margin
- *Deeper tissues* – unevenly divided with tissue tags
- *Hair bulbs* – crushed
- *Depth of the wound* – varies with force, and contains abundant foreign matter such as dust particles, paint material of the vehicle involved, etc
- *Laceration of viscera* – heavy bleeding may be seen, which may ultimately turn fatal

Table 16.5: Differences between bruise and postmortem lividity

Characteristics	*Bruise*	*PM Lividity*
Swelling	+ve	–ve
Vital reaction	+ve	–ve
Incision of the lesion	Blood is found extravasated into the subcutaneous tissue	Blood is found intact within the blood vessels

- *Skin laceration* – usually one of the margins overhangs the other, which could help in assessing the direction of force
- *On healing* – it produces permanent scar.

Classification of Laceration

Basically lacerations are of three types:

1. Split laceration,
2. Stretch laceration and
3. Avulsions.

Split Laceration (Incised-like Laceration)

Occurs when the skin is crushed between two hard objects, e.g. incised-like laceration of the scalp and forehead. During a fall, the scalp, getting crushed between skull from inside and hard ground outside, splits, resulting in a wound, which usually mimics an incised wound, unless, examined closely (Figs 16.4 and 16.5).

Stretch Laceration

Occurs due to the over-stretching of the skin, beyond its elasticity, e.g. in fracture of femur/other long bones the fracture fragment of the bone may stretch and pierce the skin and project out; in *run over accidents* when the tyre passes over the anterior abdominal wall, stretch laceration (superficial, multiple, placed closely) are developed in the inguinal region.

Avulsion (Avulsed Laceration)

Avulsion is a lesion wherein the skin (only) separates by tearing from the underlying tissues or peals off and overhangs the wound (Figs 16.6A to C), e.g. common in road traffic accidents.

Medicolegal Importance

1. Lacerations can provide clues regarding the *motive of injury, i.e.* whether it is *accidental or homicidal or suicidal*, as narrated below:
   - Accidental—commonly seen anywhere on exposed parts of the body
   - Homicidal—especially noticed on non-accessible parts of the body especially in assault cases
   - Suicidal—are very rarely seen as they are painful to produce.
2. Foreign matter in the wound could give *clues about the weapon used*, etc. e.g. paint material of causative vehicle may be transferred onto the lacerated wound.
3. The skin flap, which overhangs the cut margin *(avulsion cases)* can indicate the *direction of force* applied.

INCISED WOUND[4,6,8,9,17,18]

Definition

An incised wound is a clean cut through tissues by an object with sharp cutting edge (Figs 16.4, 16.7, 16.8A and B).

Clinical Findings

Lesion

Lesion produced could be simple or dangerous. Incised wound is simple when it is superficial and bleeds minimum and heals completely in 15 days. The scar will be permanent. It is dangerous or fatal when deep and involves the viscera or major blood vessels.

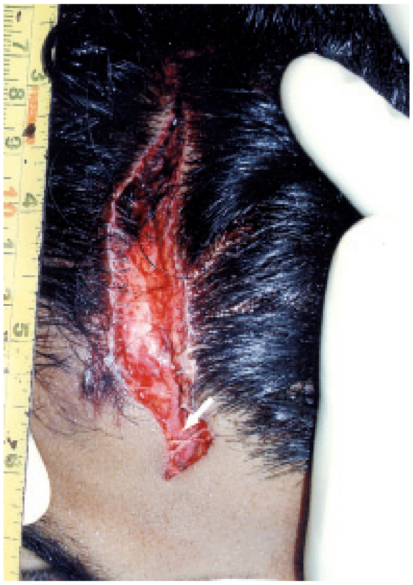


Fig. 16.4: Incised like laceration on the forehead with contused margins and tissue tags/bridges (arrow), etc.

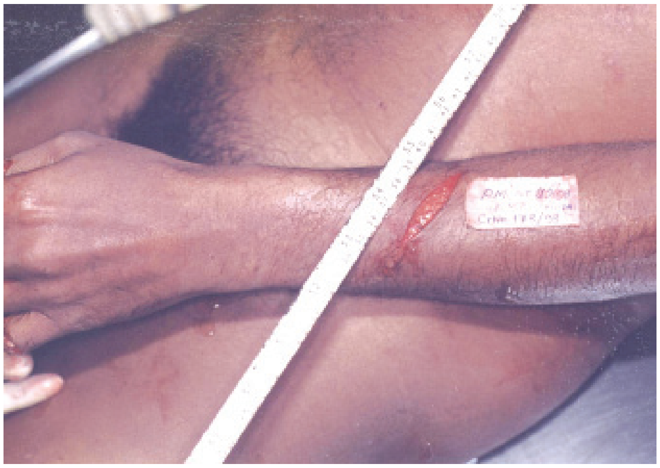


Fig. 16.5: Incised wound (defense wound) of the forearm (Note its resemblance to incised like laceration)

Examination of Incised Wound

Equipment : Magnifying lens
Describe : Site, size, shape, etc. as observed

Table 16.6 provides the characteristic features of incised wound.

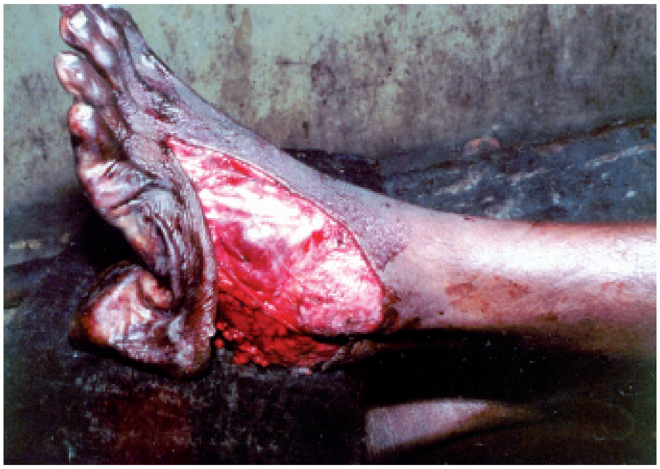


Fig. 16.6A: Avulsed lacerations of left heel and foot (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

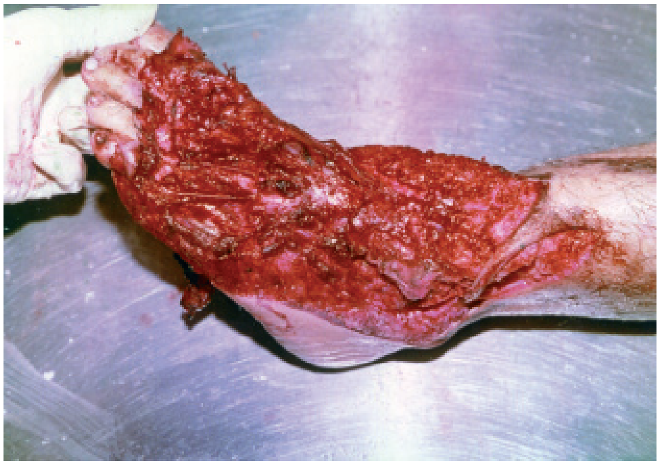


Fig. 16.6B: Avulsed lacerations of Left ankle and foot (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

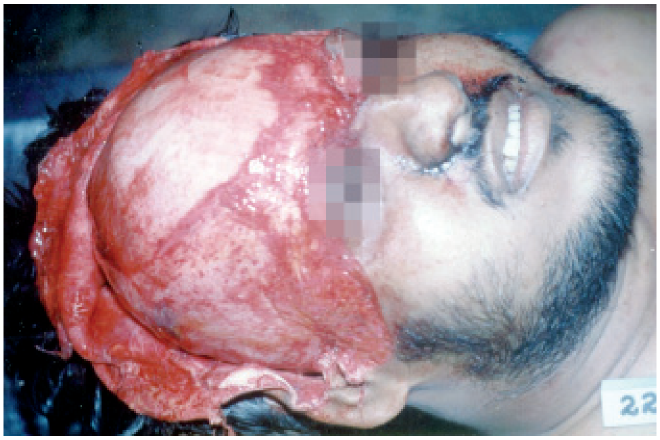
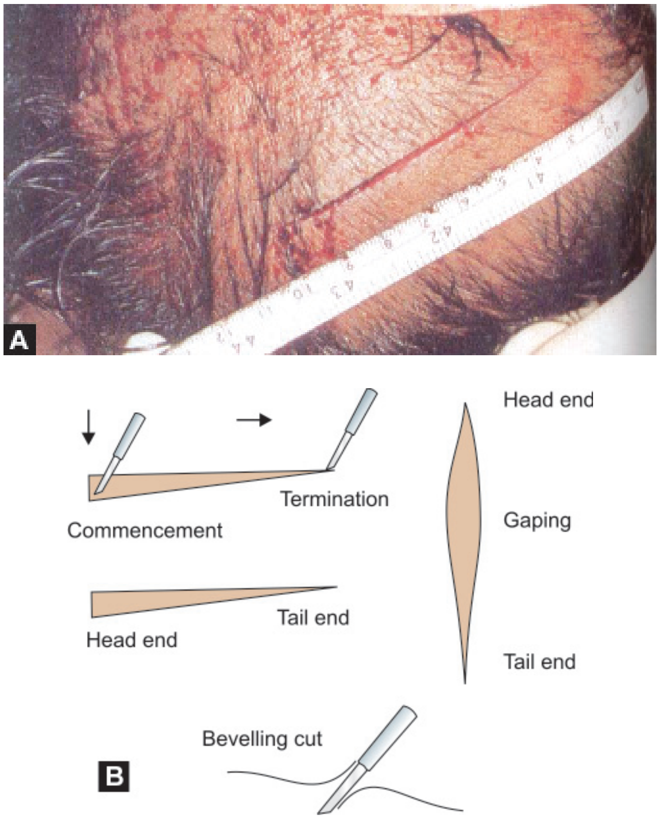


Fig. 16.6C: Degloving/avulsed lacerations of scalp (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

Table 16.6: Characteristic features of incised wound

Shape	Spindle-shaped due to gaping by skin elasticity, zigzag where skin is lax, e.g. Axilla
Edges	Clean, well defined and everted
Bleeding	Minimum if smaller vessels are cut, more if bigger vessels are cut
Length	It is greater than width/depth
Width	It is greater than edge of weapon



Figs 16.7A and B: (A) Incised wound on the scalp (Note the tailing) (B) Line drawing illustrating the tailing phenomena and beveling

Direction of the Wound

This is established by the phenomena of *tailing of the wound.* According to this, all incised wounds are deeper at point of commencement and shallower at termination. The deeper end is called *head of the wound* and the shallower end *tail of the wound* and the phenomena is known as *tailing of the wound* (Figs 16.7A and B).

Beveling cuts

Here one edge of the wound overhangs the other, and is due to penetration of the blade at an angle, which can decide the relative position of the victim, and assailant (See Figs 16.7A and B).

Age of Incised Wounds (Time since Injury)

It is assessed roughly by the stage of wound healing *grossly* as well as *microscopically* at the time of examining as shown in Table 16.7.

Medicolegal Importance

- The wound may have to be distinguished from an *incised-like laceration (tissue tags/bridges are not seen in incised wound)*
- The wound could give clues regarding the *motive* by noting the following.

Table 16.7: Gross and Microscopic changes in estimating time since injury

Observations	*Time since injury*
Gross Observation	
Red and with clotted blood	Fresh wound
Blood and lymph scab formed	12 hours
Microscopic Observation	
Epidermal cells fill the gap	24 hours
Capillary network formed	36 hours
Growth of connective tissue fibroblasts	2-3 days
Parallel growth of vessels	3-5 days
Scar tissue formed	7 days

Hesitation cuts/tentative cuts are parallel superficial cuts (Figs 16.8A and B) suggestive of suicidal motive, seen in the vital parts of the body such as neck, chest on left side *(precordial area*), wrists, etc. and are inflicted by the victim, prior to the final fatal deep cut or stab, e.g. *suicidal cut-throat wound*. Usually hesitation cuts are not seen in homicidal cut throat wound (Refer Regional Injury).

Defense cuts are incised wounds suggestive of homicidal motive or assault with sharp weapon, seen on a victim's forearms and hands, while making either an attempt to grab the weapon by its blade resulting in *active defense wounds* or just taking the weapon on his parts in order to protect himself from the attack resulting in *passive defense wound* (Figs 16.8C to F). Thus defense wounds are of two types: *Active* and *Passive*.[6,8,9,11,12]

- The wound could also give *clues on weapon causing the wound*, *time since injury*, *site of impact* and *direction of force*.

STAB WOUNDS (Punctured Wound)[2-12,18,19]

Definition

Stab wounds are wounds produced by sharp pointed objects penetrating the skin and underlying structures (Figs 16.8G to J and Figs 16.9A to D).

Causative weapons: knife, dagger, nail, screwdriver, needle, spear, arrow, sword, etc.

Clinical Findings

Lesion produced is usually dangerous or fatal due to bleeding which may be external or internal, mild or severe and associated with injury to vital organs.

Examination of the Wound

Site Anywhere, especially chest/abdomen.

Size Depends on size of the weapon used.

Shape Usually corresponds to the type of weapon used (Fig. 16.8G).

- If the weapon is a *single edged knife* – it will be wedge/triangular shape, blunt edge of the knife *corresponds* to base of the triangle (Fig. 16.10B_2). Single edged knife can also produce special effect '*Fish tailing*' (Fig. 16.10B_{2a}) phenomena, at times at the entry wound in a stab wound. Figure 16.10B_2, will highlight this concept. This is produced by the blunt edge of the weapon, which usually forms base for the triangular wound. However, in fishtailing phenomena, at the base of the triangular wound, a small triangular piece of skin remains attached to the blunt edge, giving a fish-tail like appearance.[8,21]

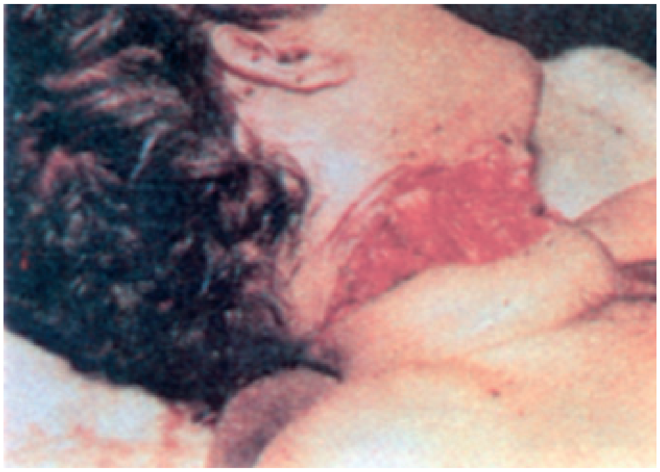


Fig. 16.8A: Suicidal cut-throat wound with hesitation cuts

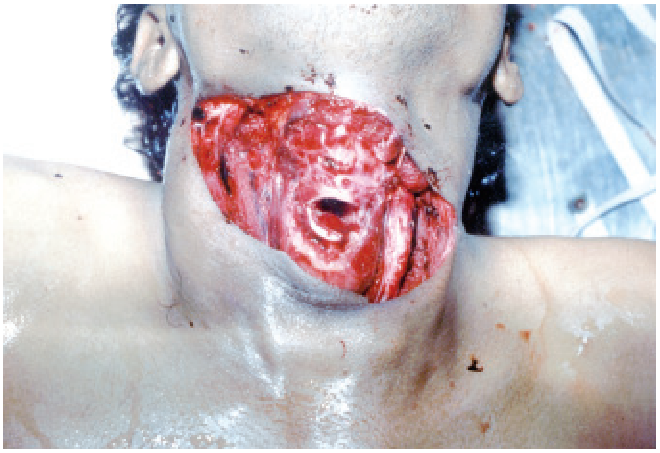


Fig. 16.8B: *Homicidal cut throat wound. Note:* No hesitation cuts (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Kilpauk Medical College, Chennai, Tamil Nadu)

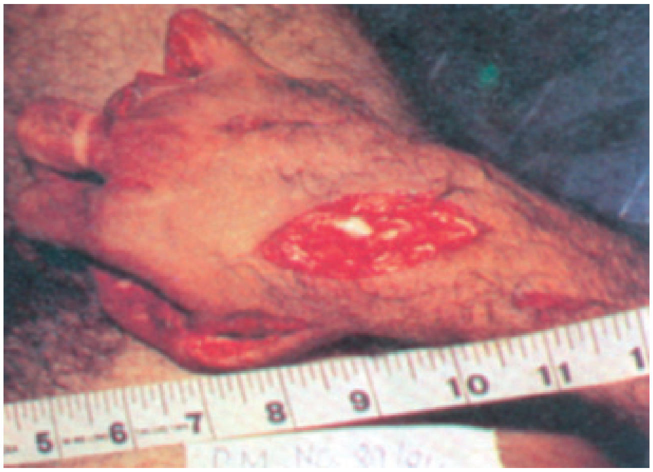


Fig. 16.8C: Passive defense wound

- If the weapon is a *double-edged knife/dagger,* the wound would be spindle shaped (Figs 16.10B_3).
- If the weapon is a *nail, screw-driver, etc.* the wound would be circular shaped (Figs 16.10B_4).

Gaping: Depends on location of the wound with reference to the *Langer's Cleavage lines* (Figs 16.9A and B), which are lines of the tension determined by the direction of *elastic and collagen fibers* in the skin.[6,9] A wound cutting across these lines produces maximum gaping, while a minimum gaping is seen if the wound is along the direction of fibres.

Length: Usually equal to the breadth of the weapon. It will be more it the weapon is pulled upwards or downwards, during insertion or withdrawal.

Breadth: More than thickness of the blade—gaping (skin elasticity and muscle pull). However reduction of the gaping manually can help in actual measurement (Figs 16.9C and D)

Depth: Is either equal to or a little less than the length of the weapon used for stabbing it could be more in yielding areas like abdomen.

Margins: Clean and sharp but may show few abrasions around, if the hilt/handle of the knife touches the skin, i.*e. on full penetration*. Unusual stab wounds are common depending on shape of the hilt, angle in which the knife is thrust, etc. (Fig. 16.8H).

Direction: It is indicated by the under cutting of the skin at the wound of entry or by the track taken by a blunt probe.[6,9,11,12,16] Probing a wound blindly from outside is not an accurate method as it could produce artifacts. It is reliable when done under supervision along with meticulous dissection. However, *layer-by-layer and stepwise dissection is the best method to establish the direction of stabbing during autopsy* (Fig. 16.8I). This can help to assess the relative positions of the victim and assailant (Corrections relative to anatomical levels of viscera while standing, lying supine, etc. may be implemented carefully in deriving accurate direction).

Volitional Activities

Volitional activities are the actual voluntary acts or physical activity observed in a victim of fatal stab injury.[12,18-20] At times the victim may walk all alone, without any help from the site of crime to a far away hospital or police station, for treatment or lodging the complaint respectively.

Classification

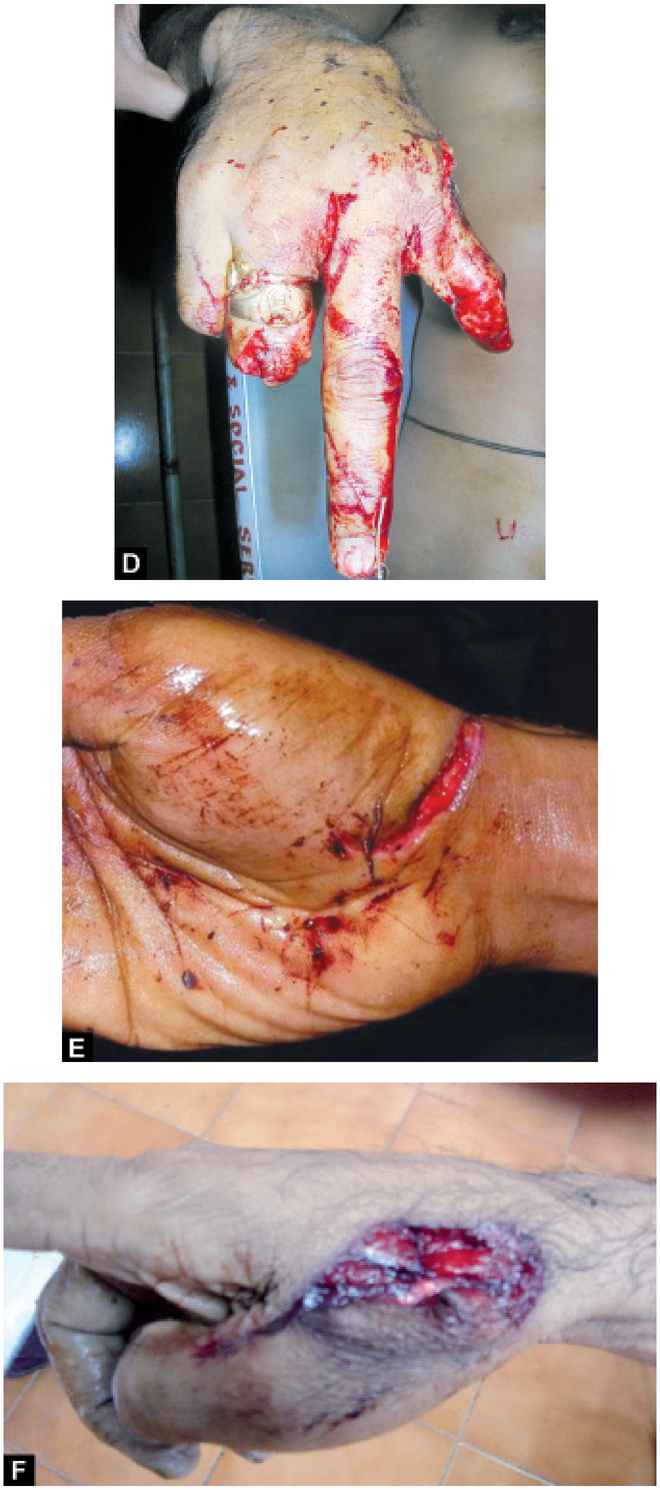
Clinically the stab wounds are of three types – *Punctured wound*, *penetrating wound and perforating wound*[21] (Figs 16.10A and B).

Punctured wound – Here the weapon just enters into the part of the body without entering into any of the body cavity. Technically it is a *typical stab wound* as its depth is greater than width. Example: Knife entering into the gluteus muscle in the buttocks (Figs 16.10A$_1$ and 16.8J).

Penetrating wound – Here the weapon just enters into the body cavity producing only one wound, i.e. wound of entry. Example: Knife entering into the abdominal cavity, without coming out of the abdomen. Thus it penetrates a body cavity. Major body cavities are chest, abdomen and skull (penetrating wounds of the skull are extremely rare though). Other body cavities, upon entering which the wound would qualify as a penetrating wound are eye balls, major joints such as knee joint, hip joint, shoulder joint etc (Figs 16.10A$_2$, 16.9B and C).

Perforating wound (through and through punctured wound) – Here the weapon after entering into one side of the body will come out through the other side, producing two wounds (Fig. 16.10A$_3$):

- Wound of entry – through which the weapon enters the body. It is larger and with inverted edges.
- Wound of exit – through which the tip of the weapon emerges out of the body. It is usually smaller with everted edges.



Figs 16.8 D to F: *Sharp force trauma:* Defense wounds: D. Active and E, F Passive. (*Courtesy:* Fig. 16.8D—Dr Mahabalesh Shetty, Assoc Professor and HOD, Forensic Medicine, KSHEMA, Mangalore, and Fig. 16.8E—Dr KR Nagesh, Assoc. Prof and HOD Forensic Medicine, FMMC, Mangalore, Karnataka)

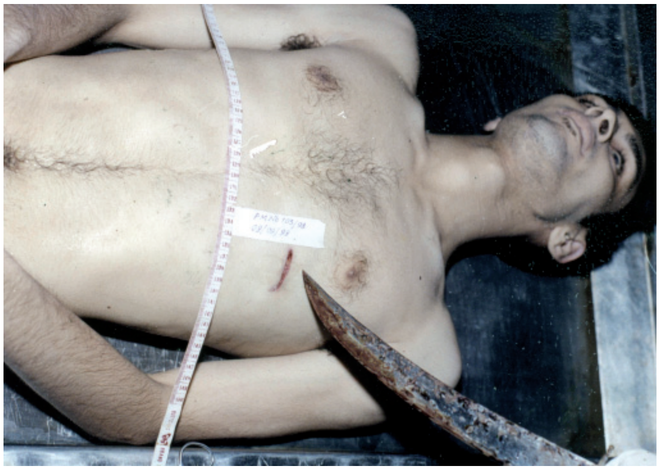


Fig. 16.8G: Stab wound and alleged weapon of assault for comparison

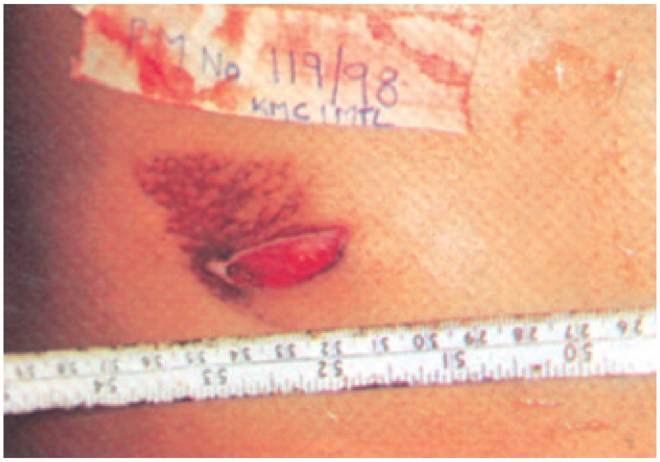


Fig. 16.8H: Unusual stab wound with contused abrasion just above the wound when thrust with blade of the knife held at an acute angulation

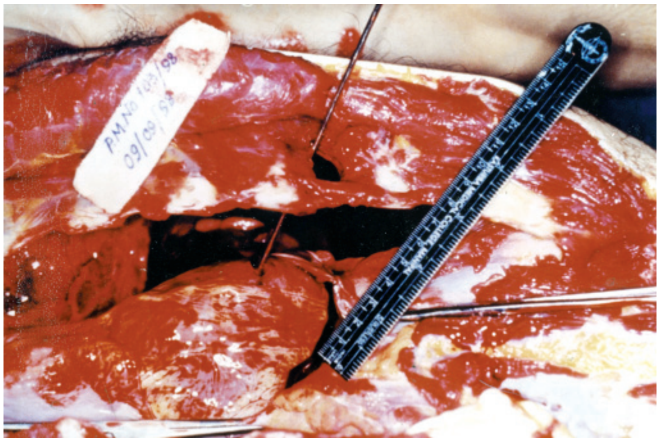


Fig. 16.8I: Direction of stab wound by layer-by-layer dissection (note the probe)

Complications

Could be any one or all the following with an outcome of consequent fatality:[6-12,17-20]

- Exsanguinations *(severe haemorrhage)* and hypovolemic shock. However, mere external haemorrhage may not

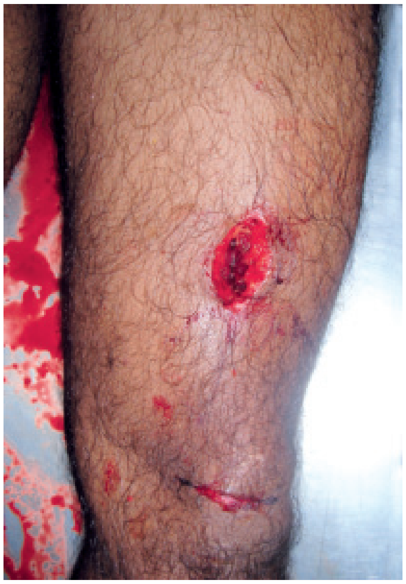


Fig. 16.8J: Punctured wound of the thigh by a knife

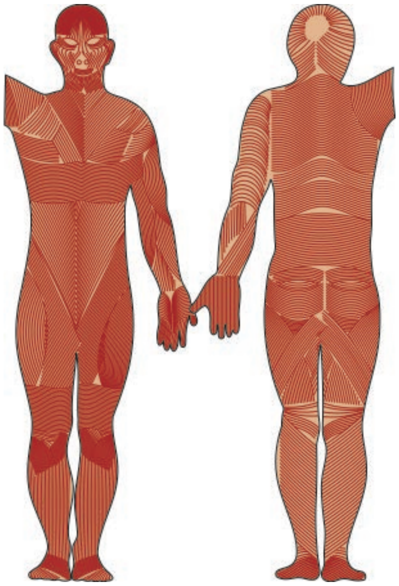


Fig. 16.9A: Langer's cleavage lines

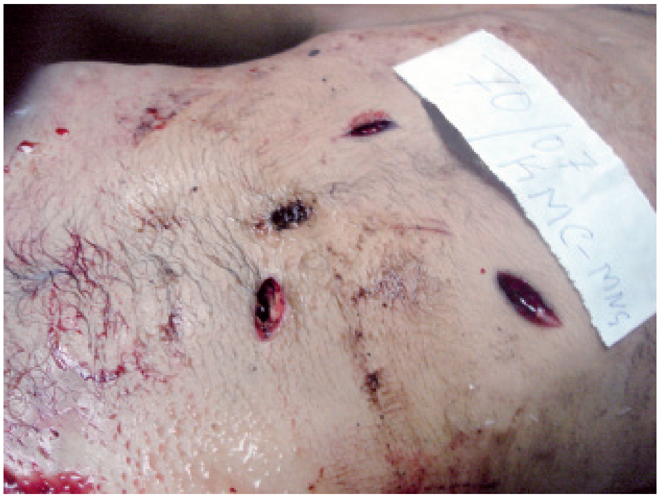


Fig. 16.9B: Multiple stab wounds of the chest showing different degree of gaping

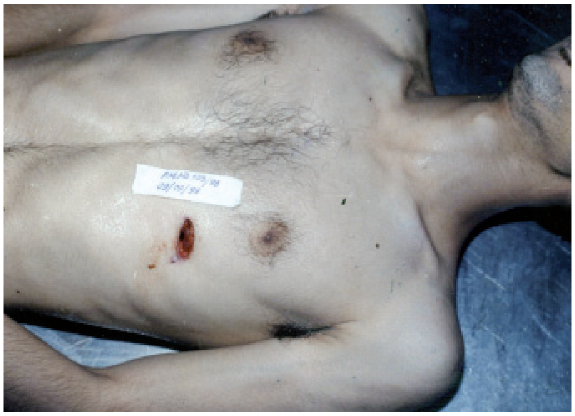


Fig. 16.9C: Stab wound of the chest with wide gaping

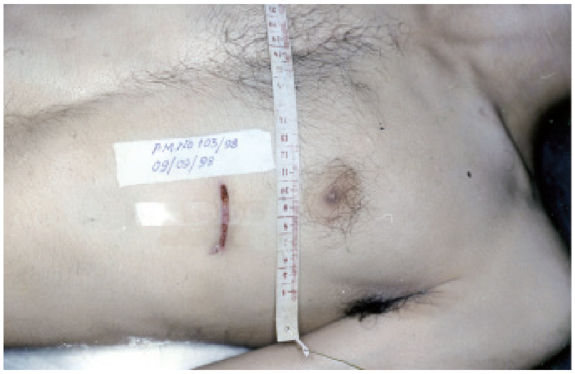
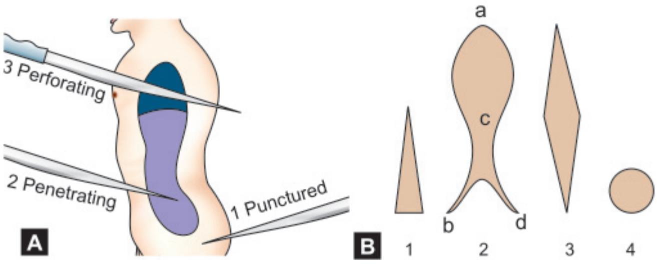


Fig. 16.9D: Stab wound of the chest (same as in 16.9C) manually reduced and reconstructed with adhesive tape-indicates approximate width of the wound



Figs 16.10A and B: A. *Types of stab wounds:* 1. Punctured (in to the Gluteus muscles) 2. Penetrating (in to the abdominal cavity) and 3. Perforating (through and through from front side of the body to back side of the body). B. *Various shapes of stab wounds:* 1. Triangular shape: Single edge knife: tip of the triangle is sharp edge of the knife, while base of triangle marks the blunt edge of the weapon. 2. Fish tailing shape wound: a. Sharp edge of weapon; b, c, and d: Corresponds to the blunt edge of weapon with triangular piece of skin remains attached to blunt edge of weapon, creating a fish tail resemblance. 3. *Elliptical shape:* double edge knife/dagger 4. *Rounded shape:* Screw driver

necessarily be the criterion for assessing the danger to life. There may be very little external haemorrhage, and yet profuse haemorrhage might have taken place internally owing to some vital organ having been penetrated, and the signs of which may be delayed.[6]

- Air embolism (systemic/pulmonary)
- Injury to vital organs (heart, lungs, liver, kidney, etc.)
- Secondary infections
- Pneumothorax.

Medicolegal Importance

1. The wound could give clues about:
    - The motive, i.e. if seen on *accessible parts* of the body along with a few *hesitation cuts* around wound of entry, it is suggestive of *suicidal stabbing*.
    - Weapon *causing* the wound
    - Direction and *force* of penetration
    - Time since injury.
2. *Vendetta murder*—is a phenomena of revengeful homicide wherein the assailant, on his or her victim stabs *several times*, especially *mutilating the face* by dragging the knife in a criss-cross manner.[6,9]
3. *Concealed punctured wounds*—these are punctured wounds made by *pins and needles,* usually difficult to locate by naked eye examination and missed by the inexperienced autopsy surgeon. They are commonly inflicted on such parts of the body such as *fontanels, inner canthus of the eyes, up in the nostrils, down into the throat, into the nape of the neck, axilla, vagina, rectum, etc.*[2,6,9,12]

CHOP WOUNDS (Chop Lacerations)

Chop wounds,[2-12,16] are injury produced by a blow with the sharp cutting edge of a fairly heavy weapon like an axe, hatchet, saber, etc. (Figs 16.11 to 16.14).

Clinical Findings

Lesion produced is always dangerous and fatal due to

- Bleeding
- Usually involving deeper viscera.

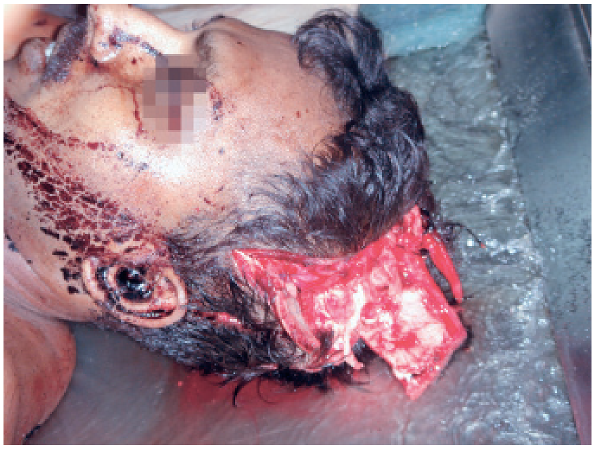


Fig. 16.11: Chop wounds of the head

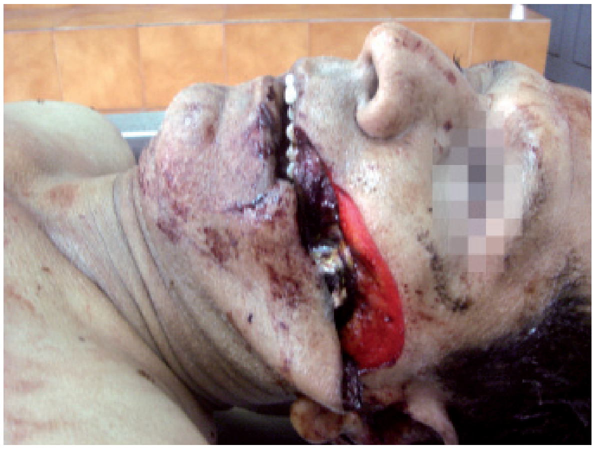


Fig. 16.12: Chop wounds of the face

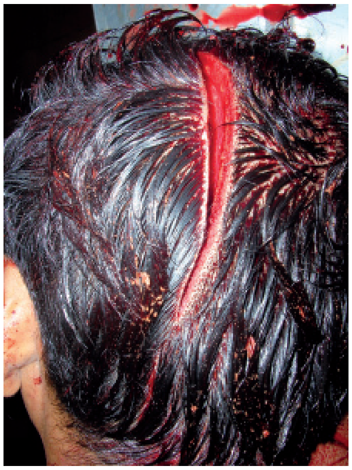


Fig. 16.13: *Chop wound of scalp:* Note the clean cut, no contused margins or tissue tags/bridges

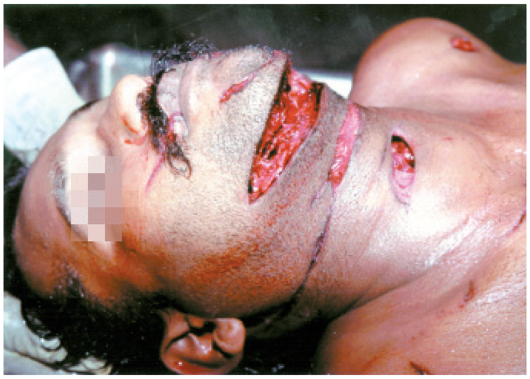


Fig. 16.14: Chop wound of the chin and cut throat wound of neck. *(Courtesy:* B Dr Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

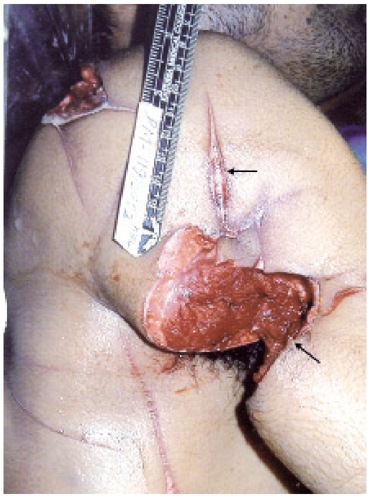


Fig. 16.15: Chop wound of the right upper arm

Examination of the Wound

Equipment used—Magnifying lens

Site, size, and shape—Varies

Margins—Sharp

Edges—Show abrasion and bruising

Dimension of the wound—Corresponds to the cross-section of the penetrating blade.

Medicolegal Importance

- Chop wounds are usually seen on exposed parts such as head (Figs 16.11 and 16.12), neck, face (Figs 16.13 and 16.14), shoulders and extremities (Fig. 16.15)
- Usually they are accompanied by severe injury to underlying bones and organs
- Majority are homicidal
- A few are accidental due to machinery such as—propeller injuries
- Very rarely they could be suicidal
- Wound examination could reveal clues regarding causative weapon.

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Chapter

17

Regional Injuries

Regional injuries comprise of injuries to various anatomical regions of the body having medicolegal significance and include injuries to the—head, neck, spine and spinal cord, chest, abdomen, limb bones and joints. The correct interpretation of these injuries is of vital importance in reconstruction of events. Hence, each of these injuries is discussed separately.

HEAD INJURY

According to Tedeshi, head injury is a morbid state, resulting from gross or subtle structural changes in the scalp, skull, and/or the contents of the skull, produced by mechanical forces.[1,2] These injuries include craniocerebral injury, trauma to the skull and brain, etc.

Of all regional injuries, those of the head and neck are most common in routine forensic practice.[2-6] A reason for this being – head is the target of choice in the great majority of assaults involving blunt trauma.[7-10] It is also true that when a victim is pushed or knocked to the ground, he often strikes the head; the brain and its coverings are vulnerable to degrees of blunt trauma that would rarely be lethal if applied to other areas.[2]

Correct interpretation of head injuries is of great importance in providing the proper treatment in the living victim. It is also important for the purposes of accurate reconstruction of the events of injury in the dead.

Classification

Head injuries are basically classified into two types depending on the involvement of dura mater.[2,7,11]

1. Closed head injury – wherein dura mater is intact.
2. Open head injury – wherein dura mater is torn.

However, based on involvement of gross anatomical structures head injuries are also classified into five heads namely:

1. Scalp injuries
2. Facial injuries
3. Skull injuries
4. Injury to meninges
5. Brain injuries.

Causes

They often result due to road traffic accidents (RTA), assaults, falls, etc. Blunt force trauma to head with a non-penetrating object or from fall, or the head striking a flat surface or a firm object can produce closed head injury. The mechanical force, which produces a brain injury, usually produces injury of the scalp, and fractures of the skull. It may also be mentioned here that a fatal brain injury may be caused without any damage to the scalp or skull.[2-11] However, every case of head injury should be radiographed and the victim must be hospitalised for minimum of 24 hours for observation.

SCALP INJURIES

Forensic Anatomy of Scalp

Scalp is the outermost covering of the head and is continuous with the skin of the face and has five layers (Fig. 17.1A) from outside to inwards: skin, connective tissue, galea aponeurotica, loose areolar tissue, and pericranium (periosteum).[5]

Injury to each layer is dealt independently. Injuries of scalp include the following examples, which have a special medicolegal significance.[2-9]

- *Abrasion* – Brush abrasions are less common than in other sites because of the protective effect of the hairs, which also tends to prevent or blur the patterned effect of less severe impacts. Thus unless the hairs are shaved at autopsy the lesser degree abrasions may be missed inevitably.
- *Contusion* – Especially black eye and spectacle hematoma (also known as Raccoon eye[13]) are important as they could give clues about the deep-seated injuries.
  - *Black eye:* This is a condition due to the bleeding in the soft tissue around the eye owing to blunt trauma of the forehead rupturing the blood vessels and the blood tracks along the facial attachments around the lower margin of the orbits (Figs 17.1B and C; also refer Fig. 16.3); in fact it is a ectopic contusion or periorbital haematoma.
  - *Spectacle haematoma:* This is a condition in which the blood is collected in the soft tissue around the eyes, due to the fracture of the base of the skull – anterior cranial fossa fractures (Fig. 17.1D).
- *Laceration* – of the scalp, is usually a split laceration and sometimes called a lacerocontused wound. Remember to look for tissue bridging (tissue tags). If this is present, it has

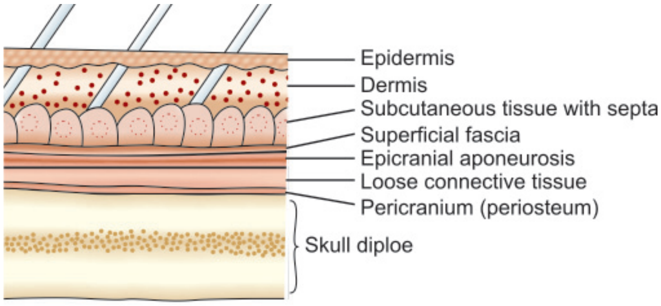


Fig. 17.1A: Anatomy of scalp

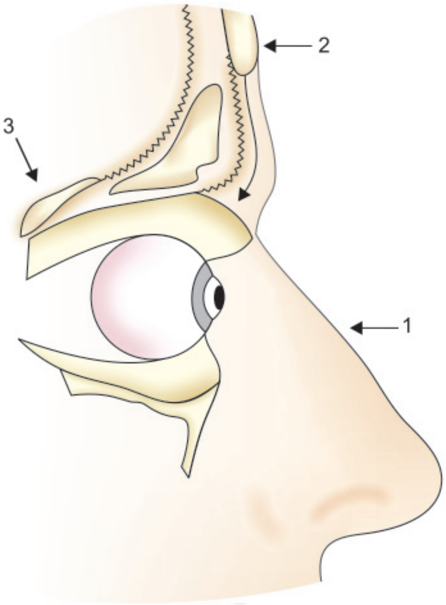


Fig. 17.1B: Line drawing illustrating the production of a 'black eye': (1) A direct blow into the orbit, (2) An injury to front of scalp, (3) A fracture of base of the skull (*Courtesy:* B Knight, Forensic Pathology[5])

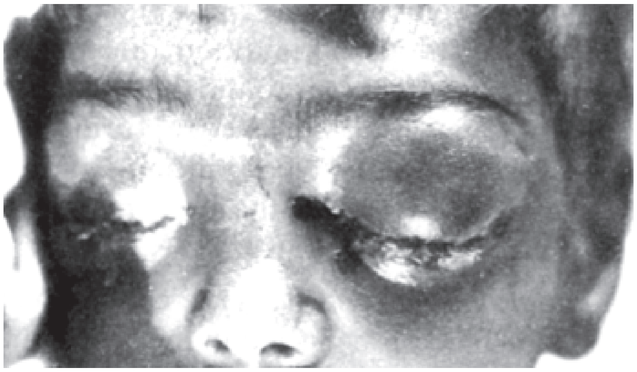


Fig. 17.1C: Black eye

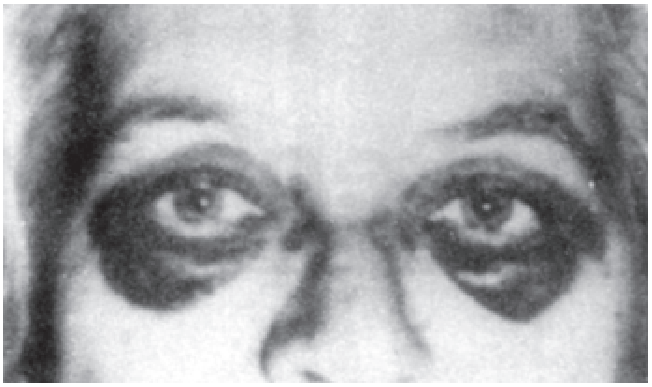


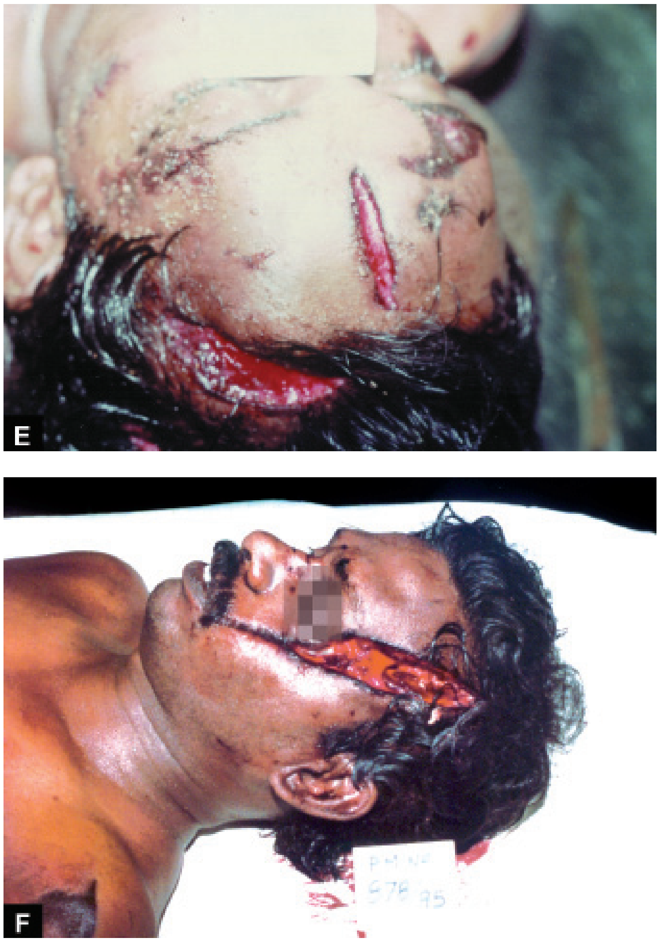
Fig. 17.1D: Spectacle haematoma

to be laceration and therefore most likely to have been caused by a blunt instrument[5,7,13] (Figs 17.1E and F).

- *Firearm injuries* – (refer specific chapter), which may resemble a laceration.

Precautions in Examining the Scalp Wound

Care must be taken to shave or trim the hair around the wound. The doctor must look for any foreign materials/particles in the wound, which may provide clues about the cause and nature of the wound, e.g. paint material of a hockey bat / stick; a splinter from a wooden club or baton used for assaulting the head; a glass piece from the broken bottle, etc. These should be collected and retained.



Figs 17.1E and F: Incised looking laceration. *Note:* Tissue tags in the gaping wound with abrasions and bruises around (*Courtesy:* Dr B Santha Kumar, Professor and HOD Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

Medicolegal Importance

Many a time a scalp injury may be the only clue about a deep-seated fatal trauma, and it can also be of help in establishing the time since injury. Scalp lacerations bleed extensively particularly in persons who are intoxicated and in the appropriate situation may even result in death from exsanguinations.[11] They rarely – if ever – become infected because of the very good blood supply of the scalp. The possibility of tetanus however should always be considered. The scars they produce on healing will be designated and contain no hair. In the rare instances when they become infected, they may lead to transfer of the infection through emissary veins into the superior sagittal (capital) sinus and more rarely into the cavernous sinuses. The latter infections are much more common in infected facial wounds and in wounds involving the accessory nasal sinuses.[12,13]

FACIAL INJURIES

Facial injuries comprise of injury to the eyes, nose, ears, lips and teeth.[14,15] Referring to facial trauma following facts are conventional:

- Facial injuries are usually prone for heavy bleeding as face is a highly vascular part of the body.
- Wounds of the face, which are more than skin-deep can cause disfiguration
- Penetrating injuries of the face are mostly fatal, particularly of the area where sense organs are present.
- Blindness is a common sequel of blows to face as retina is usually affected resulting in detachment of retina. This may also occur without any external visible injuries.

Injuries to the Eyes

Blunt force on the eyes can produce any one or more of the following:

- Lacerated wounds of the eye/s.
- Permanent injury to cornea, iris or lens.
- Vitreous haemorrhage
- Detachment of retina
- Rupture of retina
- Traumatic cataract
- Penetrating injuries to eyes or gouging out of the eyes by finger may prove fatal as this may set up meningitis.
- Black eye (refer injury to scalp).

Injuries to the Nose

Nasal injuries signify dishonor. Commonest injury is biting or cutting off of the nose, out of enmity, vengeance, sexual jealousy or suspicion of infidelity of a woman.[5-8] Left nostril may be injured in Indian female if the nose ring or stud is snatched.[3,7] Sharp pointed objects cause penetrating wound. Rarely a concealed punctured wound could be through the nostril.[5,7,8] A blow on the head may cause nose bleeding (epistaxis) due to partial detachment of mucous membrane without any injury to the nose.[5]

Injuries to the Ears

Ears may be cut off or beaten off. A blow can cause rupture of the tympanum resulting in permanent deafness. Severe blow can cause injury to labyrinth.

Injuries to the Lips

A blunt force trauma such as a blow with a fist or any blunt weapon can result into contusions or lacerations. Sometimes lips are also cut in sexual jealousy.

Injuries to the Teeth

With violence, there may be fracture or dislocation of teeth, with bleeding from sockets and contusion or laceration of the gums. A radiograph of the jaw may exhibit the fracture of the alveolar margin at the site of dental injury.

Injuries to the Facial Bones

Most of the injuries to facial bones are corollary to the application of blunt force trauma on the face. Blunt force trauma includes – blow with blunt weapons such as stick, fist etc or by fall from height over the face which can bring about fracture of the facial bones[14,14a] such as:

- Nasal bones (frequently)
- Ethmoidal bones (with more force)
- Maxilla
- Malar bones
- Mandible. A heavy blow on the jaw can drive the condyles against the base of the skull, producing a fissured fracture. Unusually the condyles may be driven through the base of the skull. Striking the face with a heavy stone can result in – pulping of the face.

Medicolegal Importance of Facial Trauma

- Commonly facial injuries include-cutting nose, cutting of lips, cutting of ears, etc.[3,4,6-9]
- Any injury resulting in permanent privation of eye sight of either eye, or hearing of either ear or/and fracture dislocation of tooth are considered as grievous hurt (Section 320 IPC).[16]

SKULL INJURIES

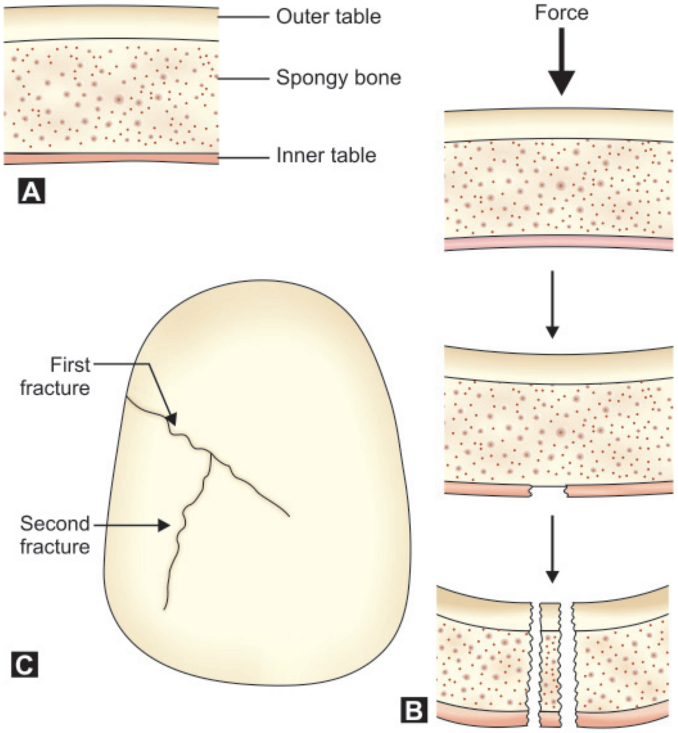
Forensic Anatomy of Skull[5,7]

Skull is a diploic bone having an outer table and inner table. The outer table is thicker and stronger, while the inner table is thinner and weaker. The spongy (often marrow-containing) bone in between is known as the diploe and gives greater elasticity to the bone (Fig. 17.2A). In children and babies the sutures have not united and thus there is even much larger lendency to give way between bones. These sutures may also spring open in head injuries in adolescents.

Skull injuries may involve the cranial vault, the base of the skull and the facial skeleton.[2-15] Though the presence of a skull fracture indicates severe traumatisation, the fracture itself rarely threatens victim's life. There are definite mechanisms of injury, effects of forces and several types of skull fractures to be distinguished (Figs 17.2B and C; Figs 17.3A and B), at this juncture.

Mechanism of Injury to Skull[2-13]

A blow with an object with a broad flat surface area or by a fall on the head causes single or multiple linear fractures or deformity of the skull (flattening/indenting at the point of impact



Figs 17.2A to C: (A) Structure of the skull bone (B) Mechanism of fracture of skull bones, and (C) Diagram to illustrate Puppe's rule[5]

and outward bending/bulging in the periphery). The fracture lines originate where the bone is bent outwards (Fig. 17.3A) and therefore is exposed to traction forces exceeding the limits of bone's elasticity. From these extruded parts of the skull fractured, fracture lines extend not only towards area of impact, but also in the opposite direction. For this reason, either of the ends is often in congruity with the impact injury of the scalp. Several fracture lines may radiate outward from a central point of impact where the skull is often depressed and/or shattered to pieces forming a spider's web or mosaic fracture consisting of circular and radiating linear fractures (Fig. 17.3B) (refer below).

Longitudinal fractures of the base of the skull frequently occur due to fall on occiput; in such instances the linear fractures typically run through posterior fossa either ending near foramen magnum or extending to floor of the middle and anterior cranial fossa. On the other hand, longitudinal fractures of the base can also be produced by impaction of frontal regions. Blunt force on the occiput, mostly as a consequence of a fall on the back of head, frequently causes independent fractures of anterior cranial fossa such as cracks of the thin orbital roofs (secondary fractures at the site of the countrecoup).

Thus injury to the skull mainly comprise of fractures which are dependent on various factors, such as:

- Violence acting / force of the active blows or passive impact
- Site of injury on the skull and
- Age of the victim.

Each of these factors are analysed below. However, it may be recalled here that the skull fractures always follow the line of the force applied to the skull, which has produced the fracture and thus can be very informative in terms of reconstruction.[5,7]

Violence Acting/Force of the Active Blows or Passive Impact

Violence/energy/force acting (F), is derived by weight (mass) and velocity (V) in accordance to the equation:[6-10]

$$\text{Kinetic Energy} = \frac{\text{Mass (m)} \times \text{Square velocity } (v^2)}{2}$$

Thus, a small bullet although of a low weight, when fired from a firearm at speed, it is more effective in terms of damage production due to the high velocity with which it moves, than an iron crowbar which is though heavy by weight, cannot be made to move with the same high velocity as a bullet. Violence could be direct or indirect.[2-8]

Direct violence – Here the forces acting directly on the bone produce a fracture, i.e.

- Compression of head of the fetus (unusual in these days) during forceps delivery of a baby or clinical use of a cranioclast.
- Head getting crushed under the wheel of a moving vehicle in a road traffic accident (RTA).
- A moving object such as a stone or bullet strikes the head.
- Falls from a height onto the head.
- Kicking and stamping injuries.
- Repeated mutual punches/blows of boxers may result in a condition called punch drunk,[3-10] wherein the victim manifests with clinical finding which may be mistaken for drunkenness, the cause being intracranial injuries and haemorrhage mainly within the basal ganglia area of the brain with Parkinsonian-like effects.

Indirect violence – Here the force is acting on the skull indirectly, through some other structures, which receive the primary impact, i.e.

- Fall on the buttocks or feet from a height, transmits the force through the vertebral column onto occipital bone and produces a Ring fracture around foramen magnum.
- Blast explosions of the face with the direction of the force coming from below.
- Heavy blows on the chin wherein the force is transmitted to the skull, leading to fracture of base of the skull.

Site of Injury on the Skull

Fracture of skull could be seen on outer or inner table alone, or in a combination involving both tables.

Explanations: As the skull is a diploic bone with limited elasticity; the actual mechanism of skull undergoing fracture may be explained as follows (Figs 17.2B and 17.3A).

- Initially due to the bone elasticity, only the outer table undergoes bending.
- Further increase in force results in fracture of the inner table first, which is weaker, followed by outer table ultimately.

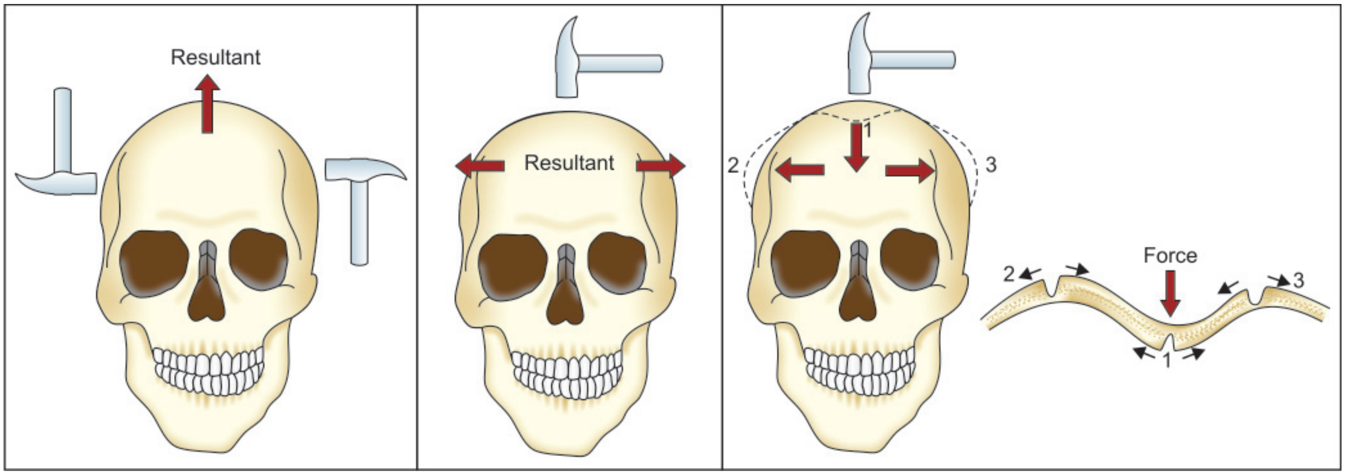


Fig. 17.3A: Mechanism of skull fracture and general effects of forces acting on the skull (Hammer represents the force applied and arrows, resultant fracture sites)

Age of the Victim

In infants and children: The skull is more elastic in infants and children. Hence it is less prone to fractures. However the brain tissue being softer in them, it is more highly vulnerable to injury.

In adults Skull: Bones are less elastic and compliant in adults. Hence, they are more prone to fractures. Fractures tend to absorb quite an amount of the energy of the blow and thus spare the energy going into the brain which is thus saved injury.

Sequence of Skull Injuries

This may be determined by *Puppe's rule*.[5] According to this, a later fracture does not cross a pre-existing fracture line but terminates on reaching the fracture line of the earlier fracture (Fig.17.2C).

Classification of Skull Fracture

Skull fractures are of two types and they are:

I. Fracture of the vault of the skull
II. Fracture of base of the skull

I. Fractures of the Vault of the Skull

Under the fractures of the vault of the skull following are included (Figs 17.3B, 17.4A and Figs 17.4B to G):

- Fissured fracture (Figs 17.3B, (i), 17.3C X-ray and 17.4A)
- Depressed fracture (Figs 17.3B (iii), 17.3C X-ray and 17.4B)
- Comminuted fracture (crushed fracture) (Fig. 17.4E)
- Stellate fracture (Radiating fracture)
- Mosaic fracture (Fig. 17.3B(ii))
- Elevated fracture
- Diastactic fracture (suture line fracture/diastactic fracture/ diastases/diastasis) (Fig. 17.3 C X-ray, 17.4C)
- Gutter fracture (Fig. 17.4D)
- Perforating fracture (Fig. 17.4F)
- Pond fracture (Indented fracture) (Fig. 17.3B (v)]
- Cut fracture
- Combined fracture.

Fissured Fracture (Figs 17.3C and 17.4A)

This is a linear fracture involving only outer or inner table or both tables simultaneously. Also called as crack fracture. When it occurs round the foramen magnum it constitutes ring fracture (Refer below). The inner table being more fragile, a linear fracture is more common in the inner table, which many a times can be missed in the radiographs and mostly is detected at autopsy. Note that the CT scans are not very good at detecting skull fractures.[5,7] They could again be of three types, namely:

- Coup-fissured fracture – occurs when head is stationary.
- Contra coup-fissured fracture – occurs when the moving head strikes a stationary object.
- Bursting-fissured fracture – occurs on the vertex and may extend up to the base of the skull or vice versa, when the head is compressed between mechanical force on one side and a hard surface on other side.

Depressed Fracture (Fractures ala signature/ Signature fracture/Bone impression fracture)

This is a fracture wherein the fractured bone fragment is driven inwards for some distance below the rest of the adjacent skull's

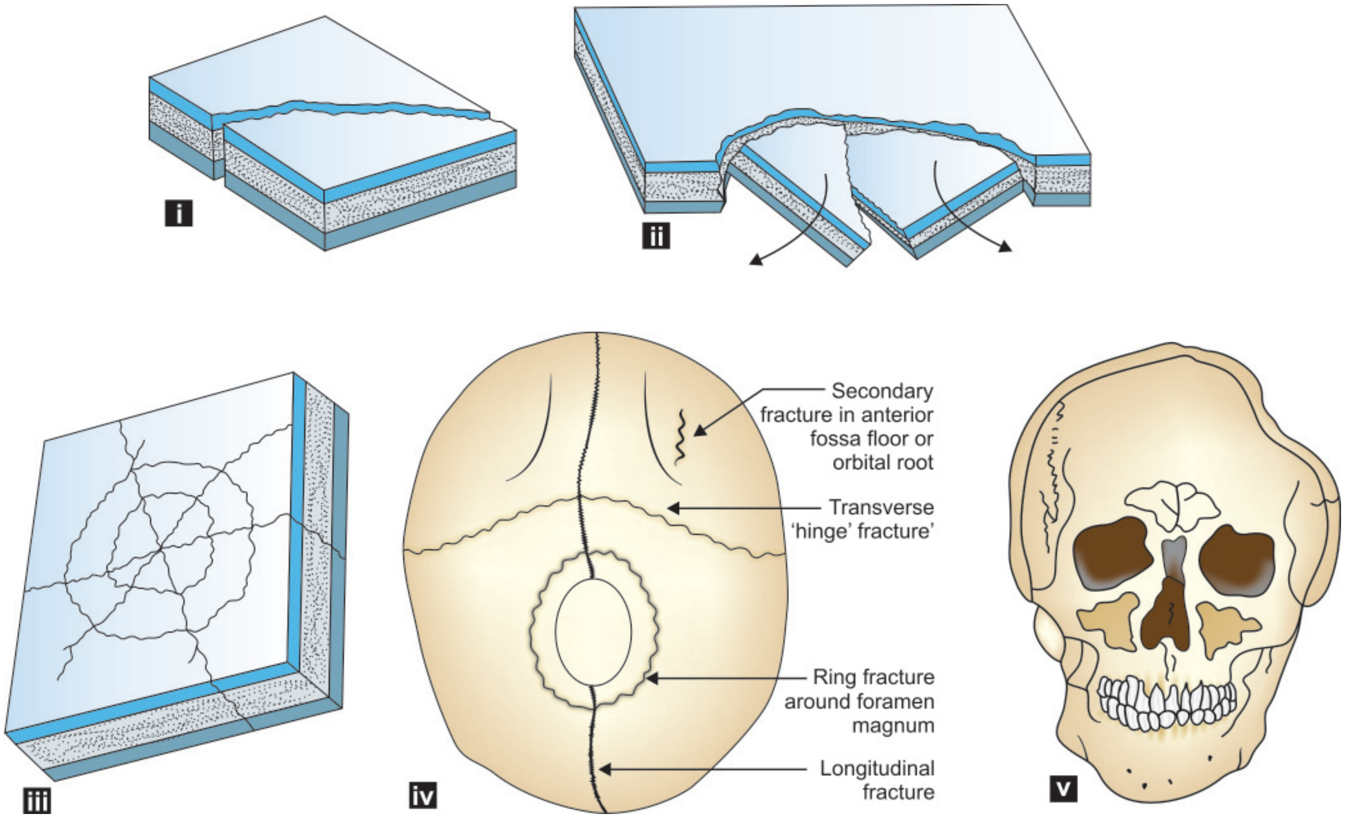

i
ii
iii
iv
Secondary fracture in anterior fossa floor or orbital root
Transverse 'hinge' fracture'
Ring fracture around foramen magnum
Longitudinal fracture
v

Fig. 17.3B: *Line drawings of types of skull fractures:* (i) Fissured fracture (ii) Mosaic fracture (iii) Depressed fracture, (iv) Fractures of base of skull (v) Pond fracture (*Courtesy:* Bernard Knight Forensic Pathology)[5]

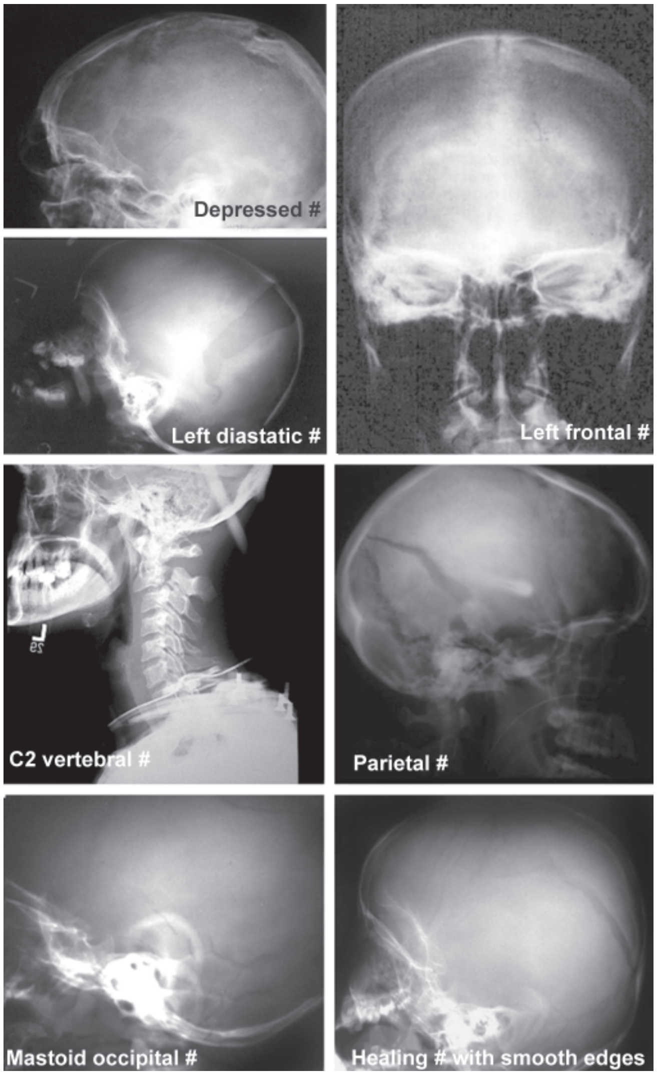


Fig. 17.3C: Radiographs/X-rays showing traumatic fracture skull—various sites

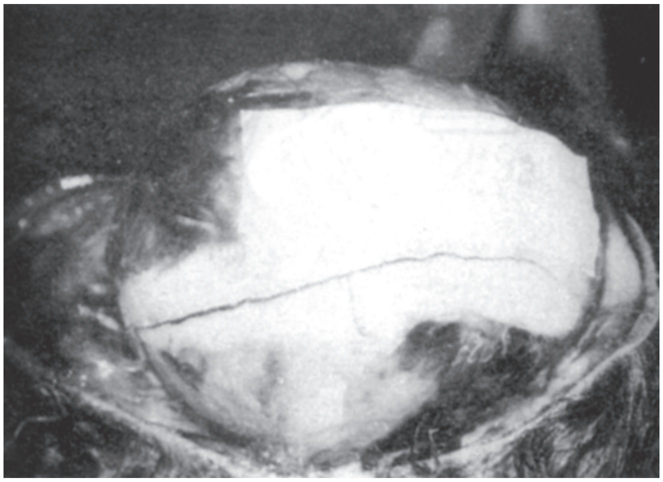


Fig. 17.4A: Autopsy view of fracture skull—fissured fracture of the vault

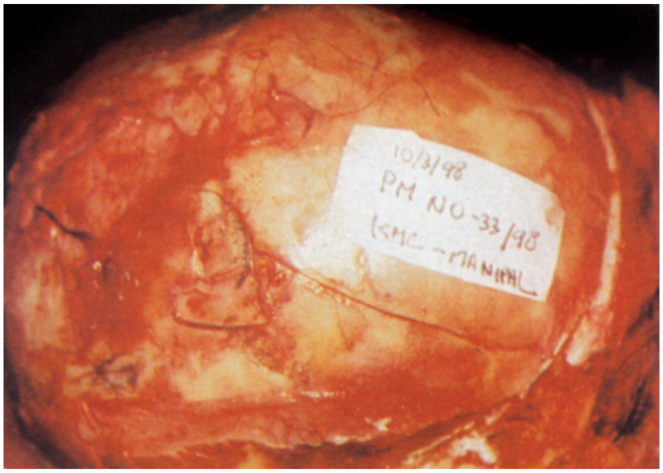


Fig. 17.4B: *Fracture skull:* Depressed fracture, with one end elevated and extended into a fissure fracture

contour (Figs 17.3C X-rays, 17.4B, Autopsy View), and it may correspond in size, shape, etc. to that of the causative weapon (heavy weapon with small striking surface, e.g. hammer, stick, stone, wrench, etc.). Hence, the fracture is also known as *signature fracture*, as it gives some clues regarding the causative weapon.

Comminuted Fracture (Crushed Fracture)

The word comminuted literally means reduced to small pieces or particles. This is a fracture, wherein the bone is broken into several pieces of different sizes. Whenever the fractured fragments are not displaced it gives a stellate appearance. Usually it occurs as a complication of depressed fracture. Causes are usually vehicular accidents, fall from height, or skull receiving blows from a weapon with large striking surface, such as a heavy iron-bar. (Fig. 17.4E). It may be useful to attempt to reconstruct the skull as this may give some indication of the causative agent. Comminuted fracture may be with or without depression of the affected area. The former is called depressed comminuted fracture, while the later is referred to as non-depressed comminuted fracture or merely as comminuted fracture.

Stellate Fracture (Radiating Fracture)

This is a comminuted fracture with fissured fractured fragments radiating from it and held intact. The causes are the same as with comminuted fractures.

Mosaic Fracture (Spider's Web Fracture)

This is a non-depressed comminuted fracture where fractured site appears in the form of several fissures forming a spider's web or cob web or mosaic pattern. Degree of depression is minimal or absent here [Fig. 17.3B (ii)].

Elevated Fracture

This is a fracture, wherein one end of the fractured fragment is elevated above the surface of skull, while the other end may dip down into the cranial cavity and injure the dura mater of the brain directly. A blow from a moderately heavy sharp-edged weapon, e.g. an axe causes this.

Diastactic Fracture (Diastasis, Suture Line Fracture)

This is a fracture occurring along skull sutures, and occurs in children and young persons (Fig. 17.4C).

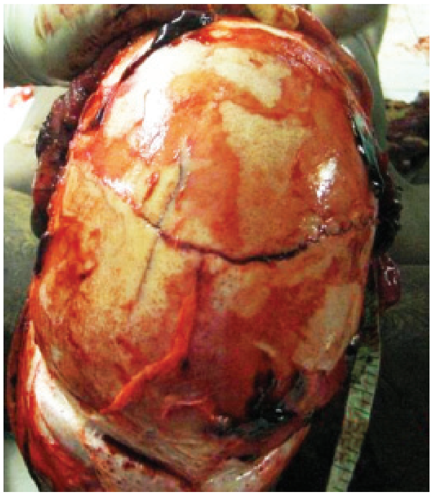


Fig. 17.4C: *Fracture skull:* Diastases along coronal suture (Courtesy: Dr Bhaskar Reddy K, Professor and HOD Forensic Medicine, SVT Medical College, Thirupathi, Andhra Pradesh)

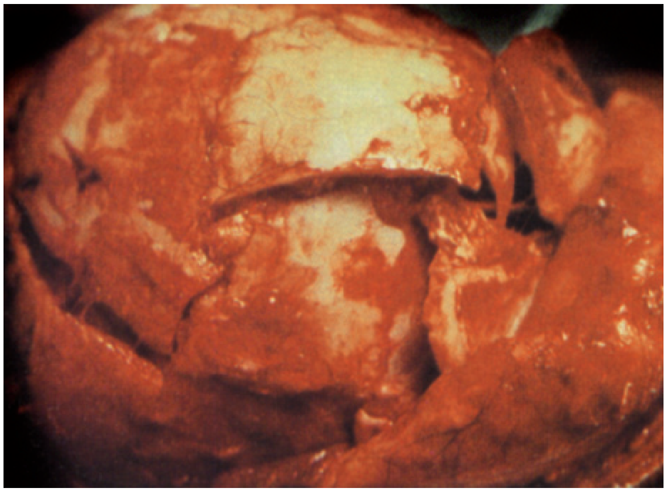


Fig. 17.4E: *Fracture skull:* Comminuted fracture (Crushed fracture)

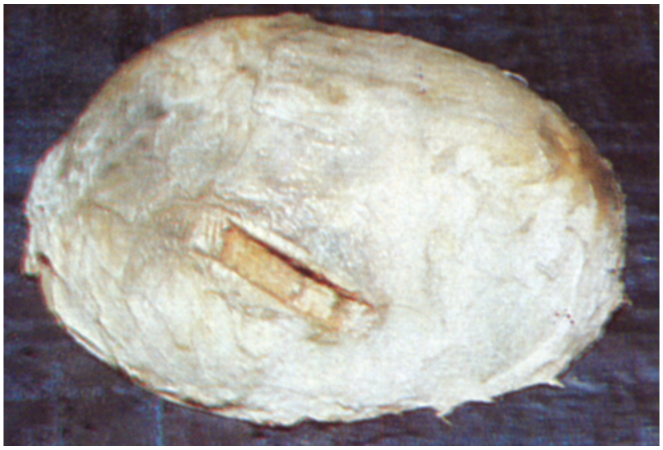


Fig. 17.4D: *Fracture skull:* Gutter fracture

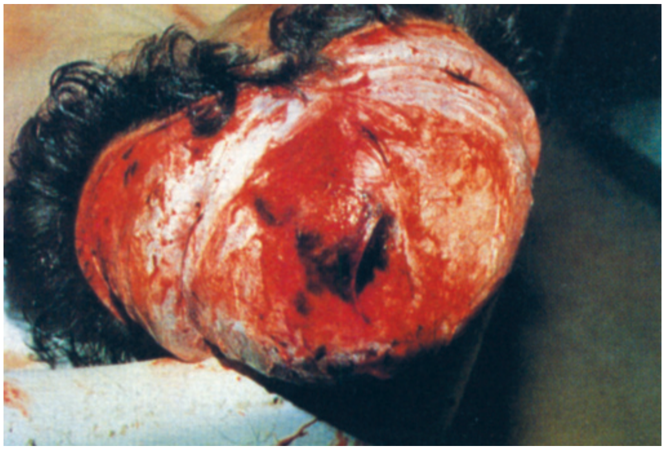


Fig. 17.4F: *Fracture skull:* Perforating fracture skull (*Courtesy:* Prof Shreemathy Rajgopal, Formerly HOD, Forensic Medicine, St. John's Medical College, Bengaluru)

Gutter Fracture

It is a fracture wherein the thickness of the skull bone is affected leading to an irregular depressed fracture of the inner table (Fig. 17.4D). At times this may be only a longitudinal depression of the outer table alone without any loss of bone underneath. A glancing bullet wound causes these fractures.

Pond Fracture (Indented Fracture)

This is a simple dent in the skull, which results from an obstetric forceps blade, or a blow from blunt object or forcible impact against some protruding object. This is basically due to the elasticity of the skull, and is especially likely to occur in the infants. The inner table of the skull here is usually intact. However, fissured fractures may be seen around the periphery of the dent. The duramater is also seen intact here and brain tissue is rarely injured. In an infant it resembles dent on a ping-pong ball.[4,7,10]

Perforating Fracture (Punctured Fracture/Hole Fracture)

This is a fracture wherein thickness of skull is affected (Fig. 17.4F) leading to an irregular depressed fracture of the inner table. At times there may be only a longitudinal depression of the outer table alone, may be observed without any loss of bone integrity underneath. A glancing bullet is one more common cause of these fractures.

Cut Fracture

A heavy cutting weapon can cause straight chops of the skull, involving either the outer table alone, or sometimes affecting both tables. In these fractures, except for the line of contact with the edge of the weapon, there may not be any lack of continuity of the bony substance anywhere.[5,10,13]

Combined Fracture

This is a fracture, wherein there is a combination of more than one, mentioned above.

II. Fracture of Base of the Skull

Fracture of base of the skull includes following subtypes [Fig. 17.3B (iv)].

- Fracture of the anterior cranial fossa
- Fracture of the middle cranial fossa
- Fracture of the posterior cranial fossa
- Fracture around foramen magnum
- Hinge fracture.
- Longitudinal fracture of base of the skull.

Fracture of the Anterior Cranial Fossa

It is due to direct impact or as a result of contracoup injuries, resulting in a black eye or escape of CSF and blood from the nose and damage to the roof of the orbit.

Fracture of the Middle Cranial Fossa

It is due to direct impact behind the ears or crush injuries of the head resulting in escape of CSF and blood from the ear when the petrous part of the temporal bone is fractured.

Fracture of the Posterior Cranial Fossa

It is due to the impact on the back of the head, resulting in escape of CSF and blood into the tissues of the back of the neck.

Fracture around Foramen Magnum (Ring Fracture)

It is fissured fracture occurring around the foramen magnum, situated 3 to 5 cm outside foramen magnum at the back, the middle ear on either side and the roof of the nose anteriorly [Figs 17.3B (iv) and 17.4G]. It is rare and occurs due to falls from a height with the person landing on the feet or buttocks or due to sudden violent turn of the head on the spine or a heavy blow directed on the chin or the occiput from below.

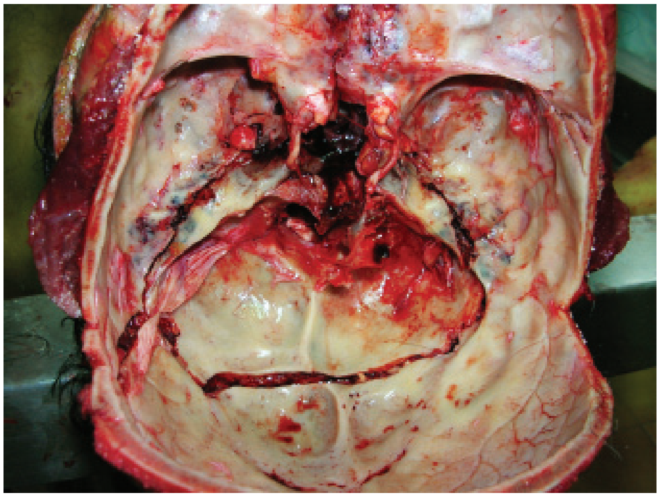


Fig. 17.4G: *Fracture base of the skull:* Ring fracture

Hinge Fracture (Transverse Fracture/Motor Cyclist's Fracture)

It is a fracture of the base of the skull where the fracture line runs from side to side across the floor of the middle cranial fossa, passing through the pituitary fossa in the midline following the course of least structural resistance (Fig. 17.4H).

The victim is usually a young pedestrian hit by a car or a motor vehicle and then sustaing secondary injuries by striking the left side of the head on the ground.[5,7] This injury is also common in road accidents among the two-wheeler riders who may be hit and thrown to the road and then sustain secondary injuries by the head striking on the ground and is hence sometimes referred to as 'motor cyclist's fracture'.[5]

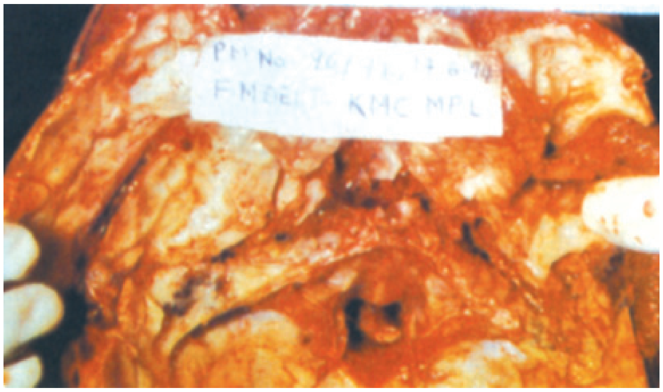


Fig. 17.4H: *Fracture of base of the skull:* Transverse hinge fracture

Longitudinal Fracture

Refer Figure 17.3B(iv).

Clinical Features of Fracture Skull

Fracture of Anterior Cranial Fossa

Bleeding – may be seen into the orbit, with swelling and purple discolouration around the eye (black eye/spectacle hematoma).

CSF leaking – may be seen from the nose.

Cranial nerve trauma – Injuries to any of the first six cranial nerves.

Fracture of Middle Cranial Fossa

Bleeding – may be seen through the ears.

CSF leaking – may be seen through the ears.

Cranial nerve trauma – Injuries to VII and VIII cranial nerves.

Fracture of Posterior Cranial Fossa

Bleeding or CSF leakage may/ may not be seen into the tissues of the back of the neck.

Cranial nerve trauma – Optic nerve may be affected.

Complications of Fracture Skull

These include concussion, compression, contusion and lacerations of brain. Each of these will be discussed separately.

Time since Injury of Skull Fracture

Also known as age of the skull injury is assessed by the changes during the healing stages of the fracture. Unlike in other bones, in the skull being a diploic bone, healing is not by callus formation. It differs depending on the type of fracture.

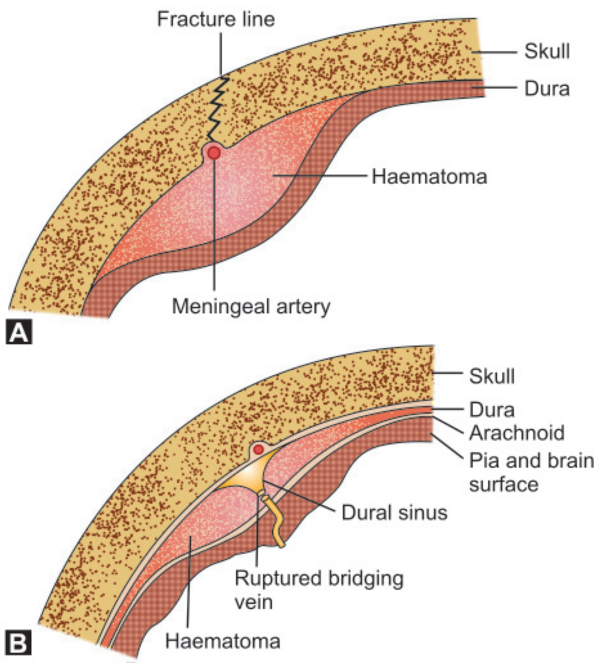
Healing in Fissured Fracture: The edges are adjacent in this type of fracture and these edges begin to stick to each other within one week, calcification of inner table and rounding of the sharp fractured ends takes place by two weeks. Bands of osseous tissue run across the fractured gap; this occurs in 3 to 4 weeks.

Healing of Comminuted Fracture: The edges in this type of fracture are not in opposition. Hence, no new bone formation occurs. The gap is filled up by fibrous tissue within 1 to 3 months' time depending on the size of the gap. However, fracture lines remain permanently visible on the radiograph.

INJURY TO MENINGES

Forensic Anatomy of Meninges

The meningeal membranes covering the brain inside the cranial cavity consist of the dura mater, arachnoid mater and



Figs 17.5A and B: *Anatomical consideration of brain showing site of intracranial haemorrhages:* (A) Extradural haemorrhage, (B) Subdural haemorrhage

piamater (Figs 17.5A and B). Dura mater is the strong and gray/bluish connective tissue membrane and is firmly attached to the skull, penetrated by bridging veins (emissary veins) along the vertex. Polypoid invaginations of dura penetrate the inner walls of venous sinuses, especially the sagittal sinus to form "arachnoid granulations." The arachnoid mater is a thin, vascular meshwork-like membrane beneath dura. Sheaths of arachnoid follow vessels into the brain as they penetrate into the neural surface. These vessels and thin strands of connective tissue anchor the brain within subarachnoid space.

This space is filled with cerebrospinal fluid (CSF) and the width of the space varies from less than one millimeter in young to a centimeter or more in the old, in whom cerebral atrophy has developed. This means that bridging vessels are longer and more vulnerable to shearing and rotatory stress. The pia mater is not a true membrane, but a surface network of glial fibers that are inseparable from underlying brain.

Meningeal Injury

At the moment of impact, the skull moves relative to dura beneath it, and the dura is stripped from the bone. Any of the three layers of meninges can be torn by the edges of fractured fragments of skull or by the penetrating objects. However, pia mater and arachnoid mater can also be ruptured due to the accumulation of blood underneath.

BRAIN INJURIES/TRAUMATIC BRAIN INJURY (TBI)[2,5,6,17-28]

At the time of head injury, only three types of brain damage in the form of diffuse neuronal injury, contusion and laceration can occur alone or in combination; oedema of brain, intracerebral haemorrhage, etc. are all secondary phenomenon, even though they occur soon after the injury.[6]

Brain injuries of traumatic origin are discussed here. They are often referred to as traumatic brain injury (TBI), which happens to be the major cause of death among the population less than 45 years age.[19] Severe TBI may be caused without any actual mechanical force or blow or fall on head. It is well established that the intracranial haemorrhage is a consequence of mere shaking in an infant (Shaking Baby Syndrome).[20-21] It is true that the brain can be injured with or without a skull fracture. So also fracture of skull can result without injury to the brain.[7, 23] Etiological factors and mechanisms responsible for TBI are discussed below.

Etiology

Three major causes of brain injuries are identified and are due to – relative movement of the brain with skull, penetrating wounds of the skull, and fracture and distortion of the skull.[22,23]

Relative movements of the brain with skull: The falx cerebri, and the tentorium cerebelli, divide the cranial cavity into three compartments, which are relatively rigid structures and act in the same way as skull in resisting the movements of the brain and can result in brain injury.

Penetrating wounds of the skull: Knife, bullet, etc. or fragments of skull in a depressed fracture can produce penetrating injury to brain.

Fracture and distortion of the skull: When a localised segment of the skull undergoes deformation or indentation, shear strains may develop in the underlying, brain tissue resulting in a contusion in the surface layers of the brain tissue. Fracture fragments or bone pieces of fracture origin may also penetrate the dura and lacerate the brain.

Mechanisms

Traumatic brain injury (TBI) biomechanics explores the mechanical phenomena that cause initial craniocerebral lesions and thus represents the starting point for the overall understanding of TBI pathophysiology.[22,23]

The linear acceleration theory: This was first published about a century ago. Relative movements and secondary impacts occur between the skull and the brain during a head impact. The pressure increases in the superficial cerebral structures below the impact zone, proportionally to the head's linear acceleration.[24] This theory explains the occurrence of most of the superficial cerebral lesions.

The Holbourn's hypothesis: In 1943 Holbourn proposed the theory of rotational movement or shear strain.[27] Accordingly the traumatic brain injury (TBI) especially the deep cerebral trauma are caused by tensile strains occurring between the superficial and deep cerebral structures during circular head movement. In a large series of experiments on primates, Thibault and Gennarelli particularly supported the role of rotational movements in the occurrence of traumatic brain damage, especially the deep cerebral contusions and diffuse axonal injury (DAI).[25]

Accordingly TBI is the consequence of spatiotemporal pressure variations occurring inside the brain during head traumas. Spatial distribution of the pressure gradient (PG) is responsible for tissue strains (compression, tensile, shear), cerebral lesion localisation and consequent neurological signs.[20,22-24]

Thus, some of the common mechanisms involved in traumatic brain injury are enumerated and explained below:

- Accelerating injury
- Decelerating injury

- Shear strain/rotational injury
- Coup and contrecoup injury.

Accelerating Injury

When a moving object hits the head, which is static, the skull picks up the momentum first and hits the brain, which is still at rest, yet to pick up the momentum a fraction of a second later. This is called an accelerating injury, e.g. a blow on the head with a hockey stick leading to brain injury.

Decelerating Injury

When a non-moving object suddenly arrests the head in motion, the skull loses its momentum much prior to brain, which hits the inner aspects of the skull before becoming static. This is called a decelerating injury, e.g. the head of a person riding a scooter, motorcycle sustains head on collision with a roadway pole or tree leading to brain injury.

Shear Strain/Rotational Injury

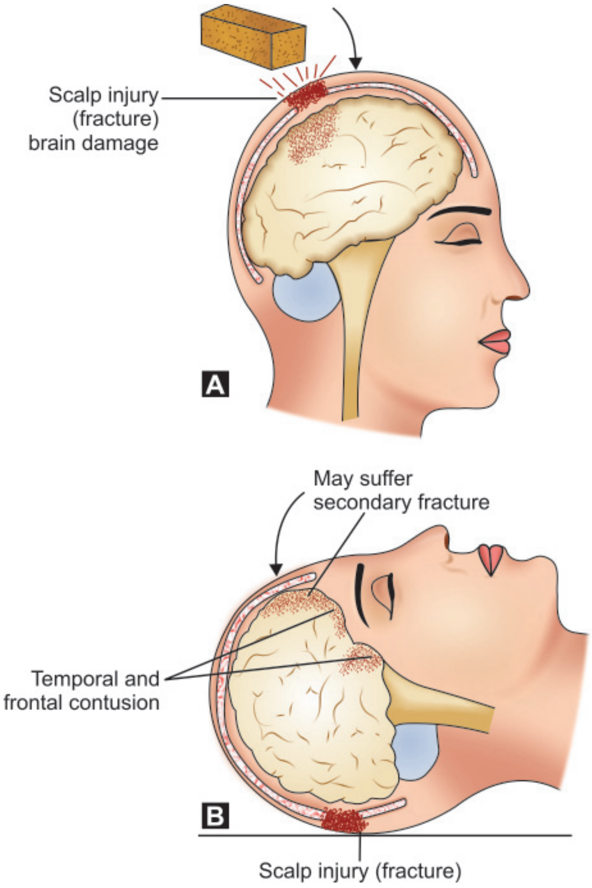
This is due to side to side or rotational movement of the head, which makes the brain to get jolted on the sharp edges of the tentorial attachments in the interior of the skull and on the bony buttresses produced by the bones of the base of the skull and get injured, accordingly.

Coup Injury

Here the injury to skull and brain both occurs at the site of impact (Fig. 17.6A). This is more common with blunt force blows to the head.

Contrecoup Injury

Here the injury to skull and brain occurs on the opposite side of the site of impact (Fig. 17.6B). This tends to be commoner in falls rather than blows.



Figs 17.6A and B: (A) Coup injury of brain in a fixed head (accelerating head), (B) Contrecoup injury to moving head (decelerated head)[5]

Role of Meninges and CSF in Preventing Traumatic Brain Injury (TBI)

Meningeal layers, which cover the brain almost all around due to its normal attachments, prevent side-to-side movement of the brain. Also the CSF almost bathes or acts as a floatation tank for the brain and provides a water cushioning effect for the brain and thus reduces the intensity of the force acting on it from all directions. However, there are no anatomical structures to limit or prevent the anteroposterior movements of the brain, which makes both ends (poles) of the brain quite vulnerable to injury. Thus, traumatic brain injuries (TBI) may take the form of cerebral concussion; diffuse neuronal injury or diffuse axonal injury (DAI), cerebral contusion, and cerebral laceration.

In real-life head trauma all these phenomena coexist. In the mean time, DAI and brain concussion also occur in purely linear acceleration head-trauma experiments.[23-25] Even, under the current approach, the linear acceleration theory cannot explain how the deep cerebral structures can be injured while the superficial cerebral structures are uninjured. However emerging of a newer concept of the stereotactical theory has made an attempt to explain these injuries.

Stereotactical Theory[24]

This is a newer approach considering the geometrical shape of the skull-brain interface, the close interactions between the two structures during their relative movements, and the resultant pressure waves propagation. The shape of the skull-brain interface is approximately spherical. Skull-brain relative movements, caused by acceleration phenomena (linear or rotational) and by skull vibrations, generate secondary pressure waves with an approximately spherical wavefront. Because brain tissue is isotropic on concentric planes, the wave propagation velocity towards the deep cerebral structures is spatially homogenous.

$$C = (E/r)\ 0.5,$$

wherein C=wave propagation velocity; E=resilience; r=density.

The spherical shape of the wave front is thus preserved. Its spoke and its surface progressively decrease. Despite attenuation phenomenon and, according to the law of energy conservation, the amplitude of the pressure waves, and thus the pressure gradient, progressively increase toward the deep cerebral structures. It will be maximal in the geometrical centre of the implied skull vault segment (Fig. 17.7), particularly if no prior significant energy consumption process occurs in the superficial cerebral structures.

Thus stereotactical phenomena can explain common posttraumatic neurological signs and cerebral lesions. It complements other biomechanical theories, which could allow us to integrate TBI biomechanics in a common concept in order to better understand TBI pathophysiology and related pathological entities like boxers' chronic encephalopathy or even Alzheimer's disease.[10] Further experimental and especially human observational research in TBI biomechanics is needed.

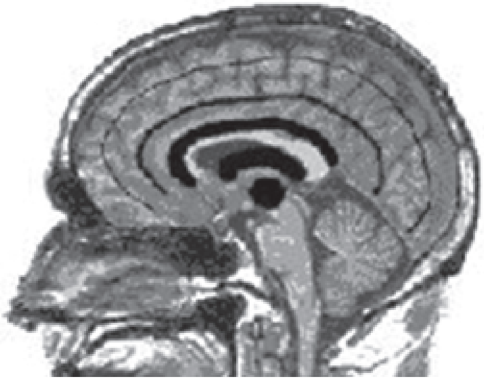


Fig. 17.7: Illustrating the stereotactical concept on a sagittal MRI view[25]

Cerebral Concussion (Stunning Brain Shock, Commotio Cerebri)

Cerebral concussion is a condition wherein there is a reversible or irreversible derangement of the neuronal activity without any demonstrable organic lesion in the brain.[2-11,17-19,22,27-29]

Cause: Trauma to the brain especially due to the decelerating injuries.

Types: Cerebral concussion are of two types namely, reversible – with mild trauma, and irreversible – with severe trauma.

Reversible Cerebral Concussion

Here clinically the patient will present with loss of consciousness and flaccidity of muscles, followed by recovery of the consciousness, leading to a second loss of consciousness later. The period of recovered consciousness between two bouts of unconsciousness is also called lucid interval. Mentally a person is found to be perfectly normal (compos mentis and fully orientated to time, place and person) during this interval.

However, the victim may also show signs of cerebral irritation, which is often due to cerebral oedema, manifests itself as constricted pupils, and altered blood pressure and respiration.

Medicolegal importance: A head injury patient should be kept under observation for 24 hours as the person may develop extradural haemorrhage insidiously.

- *Postconcussion syndrome* – It is characterised by headache, dizziness, agitation, emotional problems and nervousness, etc., after recovery.
- *Epilepsy* – Severe head injury cases may develop post-traumatic grand mal or focal epilepsy. This is medicolegally important; it is important to show by EEGs that the victim is not shamming this complication for personal gains.
- *Retrograde amnesia* – Victim is unable to recollect the exact manner in which the injury occurred and even the events occurring before and after the accident. This may extend over a period of a fortnight to a month. This is medicolegally important, as it may sometimes be due to malingering. The longer the genuine duration of this amnesic period, the more severe would be the head injury.
- *Punch-drunk syndrome*[2,5,7,8,24,26] – This is a syndrome of confusion in a setting of impaired physical function resembling Parkinson's disease.[24] Usually caused by repeated blows to the head. It also resembles the condition found in chronic alcoholics. For boxers, one of the most devastating conditions goes by many names: punch-drunk syndrome, dementia pugilistic, and chronic progressive traumatic encephalopathy. All these terms mean the same thing.

  Medical literature says this syndrome afflicts up to 25 per cent of professional fighters. It is most commonly found in heavyweights because they tend to suffer more forceful blows to the head. There are three stages of punch-drunk syndrome. The first stage is manifested by affective disturbances and mild in-coordination. In the second stage, psychiatric symptoms increase, with paranoia, and mild dysarthria and resting tremors may appear. The third stage is characterised by a decrease in general cognitive functions, memory deficits, impaired hearing, hyperreflexia, dysarthria, tremors and incoordination. Punch-drunk syndrome does not manifest rapidly; with an age of occurrence ranging from 7-35 years, with the average being 16 years.[26]
- *Drunkenness* – A head injury victim may present with clinical manifestations, which resemble drunkenness (intoxication with ethanol). It creates difficulties particularly with persons taken into police custody where the head injury may be mistaken for a drunken state, and that person dying in custody of intracranial injuries with all the repercussions that this involves.
- *Alcohol and head injuries* – A head injury sustained by a person, who has already consumed alcohol, usually creates great difficulty in assessing the relative importance. Similarly, a victim of head injury on recovery may be in a confused and disoriented condition that the clinical manifestations may simulate alcohol intoxication. Though the diagnosis is difficult, it is not impossible. Important features are described in Table 17.1.

Table 17.1: Differences between drunkenness and concussion*

Features	*Drunkenness*	*Concussion*
Alcoholic odor	Present	Absent
Skin	Suffused, flushed	Pale, cold, clammy
Blood pressure	Raised diastolic	Variable or normal
Pulse	Fast, bounding	Slow, feeble
Pupils – Accommodation	Sluggish	Variable
Eyes – Nystagmus	Present	Absent
Pupils – Reaction to light	Sluggish	Brisk
Conjunctive	Suffused, congested, injected	Variable
Breathing	Sighs, eructates (hiccup)	Shallow, slow, Irregular
Memory	Confused, but improves with time	Retrograde amnesia
Behaviour	Uncooperative, abusive	Quiet, curled up and perhaps photophobic

* If uncertainty prevails the patient should be hospitalised and kept under continuous neurological observation

Irreversible Concussion

Severe trauma can bring about brainstem (vital centre) damage resulting in fatality.

Postmortem findings usually have no specific findings, except petechial haemorrhages in the brain substance.

Diffuse Neuronal Injury

Neurons and nerve fibers sustain injury in a diffuse manner when the force of impact is transmitted through the brain. This may be wholly or partly reversible. Shear strains due to rotational movements are the probable cause for this.

Diffuse Axonal Injury (DAI)[5,17-26,29,31,33]

Diffuse axonal injury (DAI) is a clinical condition, which is one of the most common and devastating types of brain injuries[29] (Iwata et al, 2004), occurring in almost half of all cases of severe head trauma[30] (Park and Hyun, 2004). It is a type of diffuse brain injury, meaning that damage occurs over a more widespread area than in focal brain injury. DAI, often refering to extensive lesions in white matter tracts, is one of the major causes of unconsciousness and persistent vegetative state after head trauma.[30] The majority of DAI cases documented have been due to traffic accidents and some due to falls from height while occasionally it could also be due to assault.[31] In this clinical condition, there is a diffuse injury of the axons with immediate loss of consciousness and coma of more than six hours. The brain consists of billions of nerve cells located in the grey matter which communicate with distant nerve cells through long nerve fibres called axons, composed of the white matter. Deforming effect of shear strains is the main cause, which causes the disruption of axons and vessels as a consequence of impact. Severe sudden twisting or torquing of the brain, as occurs in a sudden acceleration/deceleration – whiplash – accident, can stretch, twist, and damage these delicate axonal fibres (Fig. 17.8). Under the microscope the axonal damage is called Diffuse Axonal Injury (DAI). Although diffuse axonal injury generally results from a severe whiplash injury that renders a patient comatose, recent studies have shown that diffuse axonal injury can also occur – but to a lesser degree – when there has been only brief loss of consciousness (LOC). Since DAI causes microscopic damage, it cannot be visualised on CT or MRI scans.[32]

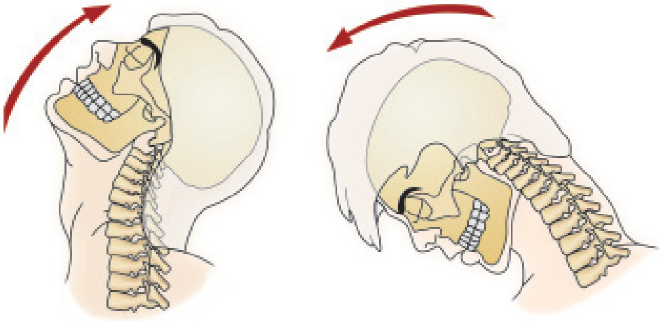


Fig. 17.8: Lateral views showing motion of head and neck during whiplash injury[32]

In mild DAI, there is coma for 6 to 25 hours. In moderate DAI, there is coma for more than 24 hours, but there are no clinical signs of brain stem dysfunction. In severe DAI, there is coma of more than 24 hours with signs of brain stem dysfunction. In mild DAI, some axons may be involved in the white matter of the cerebral hemispheres, corpus callosum, and upper brain stem, with focal haemorrhages in the corpus callosum and dorsolateral rostral brainstem.

Microscopic Examination Findings of DAI

Microscopic examination does not show axonal injuries up to 12 hours after the injury. After 12 hours, the axons first appear dilated, then club-shaped and finally appear as round balls known as "retraction balls",[31,36] which indicate the transected axons (Fig. 17.9). The number of retraction balls begins to decrease 2 to 3 weeks after the injury, and clusters of microglial cells appear, followed by astrocytosis and demyelinisation. It is generally considered that axonal injury is apparent only on electron microscopy in the very early stage after a closed head injury.[33]

DAI and the Biochemical Cascades

DAI is characterised by axonal separation, in which the axon is torn at the site of stretch and the part distal to the tear degrades. While it was once thought that the main cause of axonal separation was tearing due to mechanical forces during the

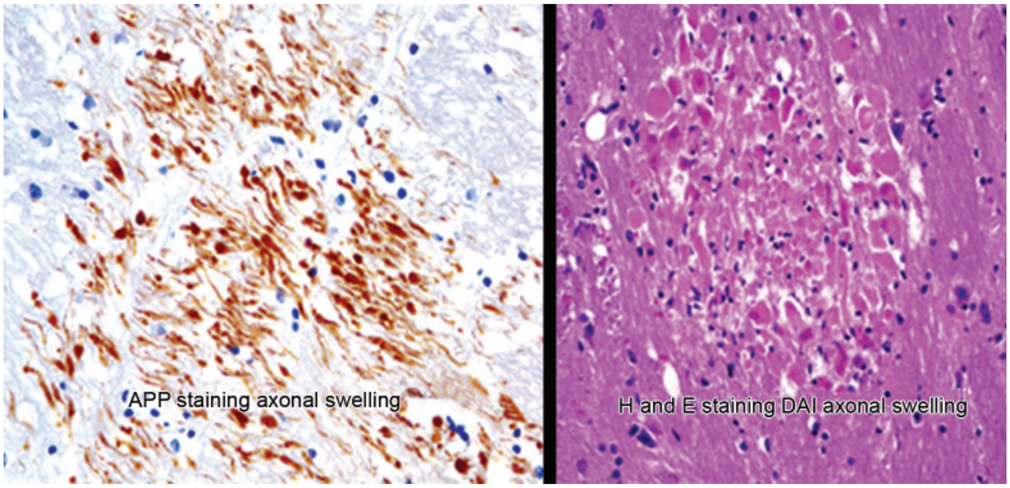

APP staining axonal swelling
H and E staining DAI axonal swelling

Fig. 17.9: Illustrating the diffuse axonal injury (DAI), with retraction balls and axonal spheroids (axons appearing as round balls). Although axonal spheroids are best demonstrated by silver stain or immunostaining for neurofilament protiens, it can often be seen with hematoxylin eosin stain. Silver staining can detect spheroids 15-18 hours after the injury. Immunostaining for amyloid precursor proterin (APP) can detect spheroids much earlier (2-4 hours after injury) than silver staining. Immunostaining for neurofilament proteins can also work

trauma, it is now understood that secondary biochemical cascades, which occur in response to the primary injury and take place hours to days after the initial injury, are largely responsible for the damage to axons.[34,35]

Though the processes involved in secondary brain injury are still poorly understood, it is now accepted that stretching of axons during injury causes physical disruption and proteolytic degradation of the cytoskeleton (Iwata, et al. 2004). It also opens sodium channels in the axolemma, which causes voltage-gated calcium channels to open and Ca^{2+} to flow into the cell.[29] The intracellular presence of Ca^{2+} unleashes several different pathways, including activating phospholipases and proteolytic enzymes, damaging mitochondria and the cytoskeleton, and activating secondary messengers, which can lead to separation of the axon and death of the cell.[34]

Thus the DAI will have a triad of gross focal lesions of haemorrhages and/or lacerations in the corpus callosum and brain stem, and microscopic demonstration of axonal damage – retraction balls.[31,36]

Cerebral Contusions[2-13,19,22,30]

It is bruising of the cerebral tissue (Figs 17.10A and B). There may not be any apparent injury of the scalp or skull outside.

Causes

Common injuries that can result in cerebral contusion are:
- Coup and contrecoup injuries
- Shear strain injuries
- Absorption shock waves
- Penetrating injuries
- Skull fractures.

Site

Usual sites prone to develop a lesion with any of the causes mentioned above are:
- Frontal lobe contusions—commonly.
- Cerebellar and medullar contusion—observed often with coning or herniation of brain into foramen magnum.

Complications

Routinely only two complications are encountered:
- Cortical atrophy
- Infection – resulting in brain abscess.

Cerebral Laceration[2-13]

Cerebral laceration is tearing of the cerebral tissue and is usually found with cerebral contusion of several degrees (Figs 17.10A and B).

Cause

Same as for cerebral contusion.

Site

Frontal lobes and temporal lobes—commonly.

Types

It could be of three types, wherein the lesion is:
- Superficial (gray matter alone is involved)
- Deep (white matter alone is involved)
- In the vital centres or ventricles.

Clinical Features

Loss of consciousness predominantly.

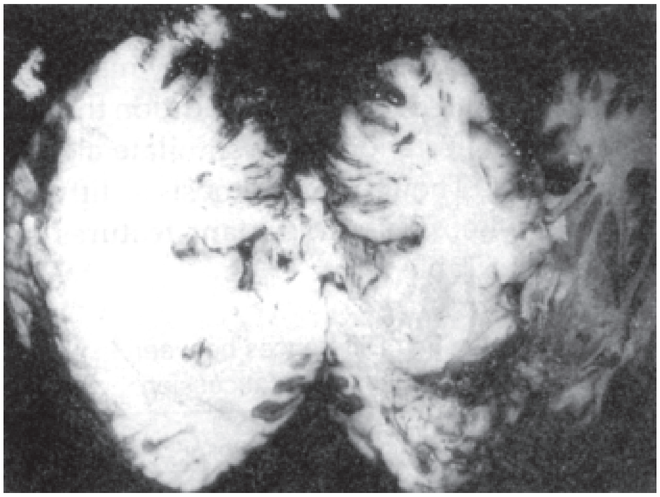
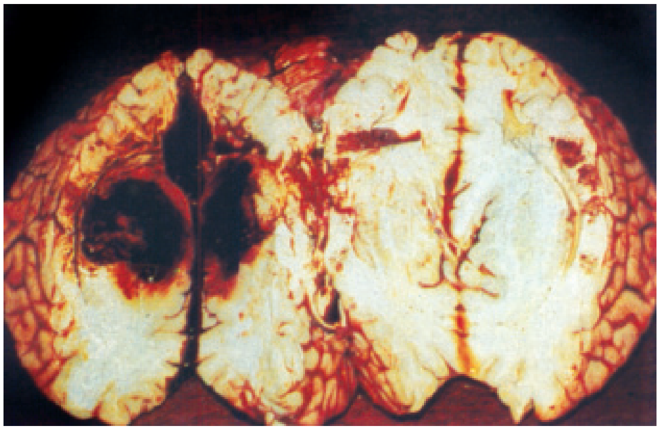


Fig. 17.10A: Laceration and contusion of frontal lobes



Figs 17.10B: Internal laceration of basal ganglion, with cerebral contusion on the right side

Complications

Cerebral lacerations can lead to:
- Bleeding from torn pial blood vessels
- Oedema of the brain tissue
- Increased intracranial pressure
- Death when not properly treated.

Healing

Healing of cerebral lacerations is by gliosis.

Cerebral Irritation

Cerebral irritation includes a peculiar set of symptoms that may follow cerebral concussion, victim lies curled up in the bed, with face hidden beneath the clothes, resents all forms of interference and exposure to light. He is conscious and may become violent and abusive if disturbed.

These symptoms disappear after a varying period with total recovery or may be followed by post-concussion syndrome symptoms.

Cerebral Compression

It is a clinical condition due to increased intra-cranial pressure, which disturbs the function of the brain.

Causes

Formation of pressure over and around the brain stem, due to increase in intracranial pressure as a result of depressed fracture of the skull, foreign body, oedema and haemorrhage are the common causes of cerebral compression.

Pathology and pathogenesis [2-13]

Any increase in the size of the brain, e.g. generalised oedema/ swelling or space-occupying lesions within the cranial cavity; result in compression of the brain. As the brain is incompressible, the compression will diminish the amount of CSF in the subarachnoid space and in the ventricles.

A continued rise in intracranial pressure leads to a progressive interference with the blood supply of the brain. If there is an increase in intracranial pressure above the tentorium, the adjacent uncus or inner margin of the temporal lobe is squeezed down through the hiatus along the midbrain, either on one or both sides, due to which the midbrain is squeezed from side to side and lengthens anteroposteriorly. This stretches the paramedian and nigral vessels, which rupture to produce haemorrhages in the midline and along the substantia nigra, which is fatal.

Sometimes, there is hemorrhagic infarction of the medial cortex of one occipital lobe, due to the twisting of posterior cerebral artery around the edge of the tentorium by herniation.

A rise of pressure below the tentorium forces portions of the cerebellar lobes and tonsils of the cerebrum through the foramen magnum, and the medulla oblongata is compressed, which causes progressive failure of respiration. Figure 17.11 illustrates events of cerebral compression.

Note: Uncal grooving and foraminal indentation of the cerebellar tonsils are common post-mortem findings and must not be misinterpreted as evidence of uncinate herniation and cerebellar coning.

Unconsciousness occurring sometime after the infliction of a head injury suggests cerebral compression. Immediate unconsciousness occurs due to concussion, and then the person slowly recovers consciousness and again loses consciousness gradually due to compression. A so-called "lucid interval" thus intervenes between the two stages of unconsciousness. In some cases, there is no lucid interval. In other cases, there will be no initial unconsciousness, but there is gradual unconsciousness from compression sometime after the trauma. A large extradural haemorrhage can produce rapid compression within a few hours of the injury. A large subdural haemorrhage produces relative slow compression, because it takes several days for sufficient blood to accumulate in the subdural space to produce compression. Delayed death may occur after a head injury due to a chronic subdural haematoma. Hence, patients who have sustained head injuries should be kept in hospital for observation for at least 24 to 36 hours.

Autopsy Evidences of Cerebral Compression[27]

- Flattening of the gyri
- Narrowing of the sulci
- Apparent decrease of CSF
- Deep grooved marking around the uncus of the temporal lobe and cerebellar pressure cone.

Mechanism of Sleep and Unconsciousness

Impulses derived from all parts of the body on the reticular system of the brain stem, maintain arousal/consciousness of an individual. Normally sleep results from reduction of stimuli to reticular system and irresistible sleep results from exhaustion of enzyme system essential to maintain the function of reticular system. Following may result in sleep and unconsciousness:

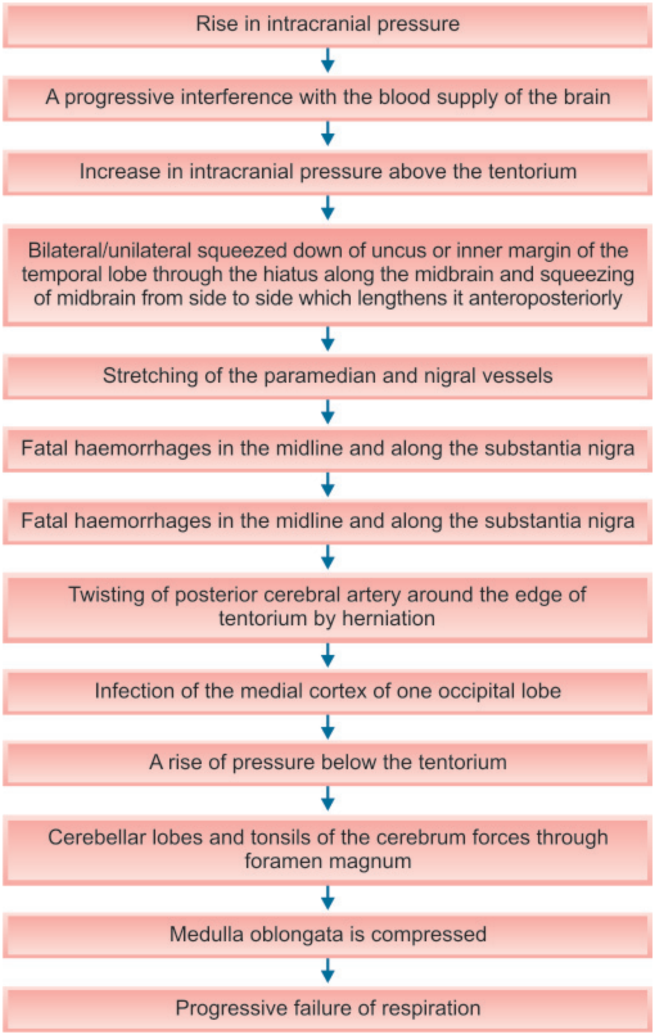

Rise in intracranial pressure
A progressive interference with the blood supply of the brain
Increase in intracranial pressure above the tentorium
Bilateral/unilateral squeezed down of uncus or inner margin of the temporal lobe through the hiatus along the midbrain and squeezing of midbrain from side to side which lengthens it anteroposteriorly
Stretching of the paramedian and nigral vessels
Fatal haemorrhages in the midline and along the substantia nigra
Fatal haemorrhages in the midline and along the substantia nigra
Twisting of posterior cerebral artery around the edge of tentorium by herniation
Infection of the medial cortex of one occipital lobe
A rise of pressure below the tentorium
Cerebellar lobes and tonsils of the cerebrum forces through foramen magnum
Medulla oblongata is compressed
Progressive failure of respiration

Fig. 17.11: Illustrates the various events of cerebral compression

- Damage to the reticular system
- Reduction in afferent activity of reticular system
- Action of toxic agents on enzyme system of reticular system
- Injury to the brain stem occurs by:
  - Stretching of peduncles when the hemispheres shift.
  - Deceleration against basisphenoid and dorsum sellae.
  - Lateral shift of the peduncle against tentorial margin.
  - Stretch or avulsion of cranial nerves from the brainstem.
  - Traction on vascular supply of the brain stem.
- Pontine haemorrhage.

Medicolegal Importance

Diagnosis of cerebral compression is very important clinically, as surgical treatment of the cause can relieve the compression, which is invariably a life-saving measure.

INTRACRANIAL HAEMORRHAGE[2,4-5,7,11,17,19,37,39]

Intracranial haemorrhage includes haemorrhage occurring within the cranial cavity. Fracture of the skull bones, though is the common cause of intracranial haemorrhage, the haemorrhage can occur even without fracture of any of the skull bone, or in absence of any injury to the brain, but due to disease process or effects of trauma on existing diseased area of the brain. A detailed history of past illness, and detailed autopsy may be the answer to various questions that often arise in case of death from head injuries.

Haemorrhage resulting directly from trauma usually occurs over the surface of the brain, but deep-seated haemorrhage can also occur in the cerebrum, cerebellum or brain stem, due to trauma. The intracranial haemorrhage is usually accompanied with contusions or lacerations of the brain as coup or contra-coup injuries. A single area of deep-seated intracerebral haemorrhage is usually due to some disease process other than trauma. In an arteriosclerotic and hypertensive subject, emotional excitement or physical exertion may precipitate intracerebral haemorrhage; if such person falls down with scalp injury, the haemorrhage may appear to be traumatic in origin.

Forensic Anatomy of Brain Membranes

The membranes covering the brain inside the cranial cavity consist of the dura mater, arachnoid mater and piamater (See Figs 17.5A and B). Dura mater is the outermost covering which is tough, penetrated by bridging veins along the vertex. Polypoid invaginations of dura penetrate the inner walls of venous sinuses, especially the sagittal sinus to form "arachnoid granulations."

The arachnoid mater is a thin, vascular meshwork-like membrane beneath the dura. Sheaths of arachnoid follow vessel into the brain as they penetrate into the neural surface. These vessels and thin strands of connective tissue anchor the brain within subarachnoid space. This space is filled with cerebrospinal fluid (CSF) and the width of the space varies from less than one millimeter in young to a centimeter or more in the old, in whom cerebral atrophy has developed. This means that bridging vessels are longer and more vulnerable to shearing and rotatory stress. The piamater is not a true membrane, but a surface network of glial fibers that are inseparable from underlying brain.

Causes

Common causes of intracranial haemorrhages (ICHs) are as follows:

- Diseases like aneurysms, arteritis, blood disorders, neoplasm, etc.
- Trauma to the skull, brain or its membranes.
- Effects of injury upon already existing disease.

Classification

According to Graham and Gennareli intracranial haemorrhage is classified into two groups intra-axial and extra-axial depending on the site of bleeding.[37]

I. Intra-axial Haemorrhage

Intra-axial haemorrhage is bleeding within the brain itself. This category includes (Fig. 17.12A):

- Intraparenchymal haemorrhage or bleeding within the brain tissue.
- Intraventricular haemorrhage or bleeding within the brain's ventricles (particularly of premature infants).

II. Extra-axial Haemorrhage

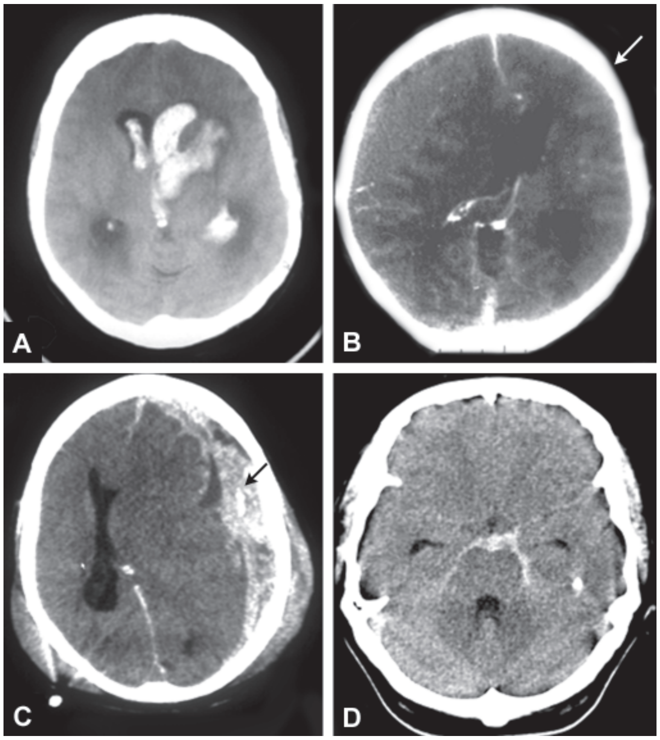
Extra-axial haemorrhage, bleeding that occurs within the skull but outside of the brain tissue, falls into three subtypes (Figs 17.12B to D):

- *Extradural hemorhage/Epidural haemorrhage* – is caused by trauma, and results from laceration of an artery, most commonly the middle meningeal artery. This is a very dangerous type of injury because the bleed is from a high-pressure system and deadly increase in intracranial pressure can result rapidly. Patients have a loss of consciousness (LOC), then a lucid interval, then sudden deterioration (vomiting, restlessness, LOC). A lenticular (convex) deformity in the head CT is diagnostic of this condition (Fig. 17.12B).
- *Subdural haemorrhage* results from tearing of the bridging veins in the subdural space between the dura and arachnoid mater. Head CT shows crescent-shaped deformity (Fig. 17.12C).
- *Subarachnoid haemorrhage*, like intraparenchymal haemorrhage, can result either from trauma or from rupture of aneurysms or arteriovenous malformations. Blood is seen layering into the brain along sulci and fissures, or filling cisterns (most often the suprasellar cistern because of the presence of the vessels of the Circle of Willis and their branchpoints within that space) (Fig. 17.12D). The classic presentation of subarachnoid haemorrhage is the sudden onset of a severe headache. This can be a very dangerous entity, and requires emergent neurosurgical evaluation, and sometimes urgent intervention.

Each one of these intracranial haemorrhages is discussed below in detail:

EXTRADURAL HAEMORRHAGE (EDH)[38]

Extradural haemorrhage (also known as epidural haemorrhage) is a type of intracranial haemorrhage, wherein the bleeding is outside the dura mater (Fig. 17.13A).



Figs 17.12A to D: *CT Scan images of intracranial haemorrhages:* (A) Intra-axial haemorrhages type – *Intracerebral and intraventricular* haemorrhage; (B) Extra-axial type – Extradural/epidural haemorrhage (Lenticular deformity); (C) Extra-axial type – Subdural haemorrhage (Crescent-shaped deformity); (D) Extra-axial type – Subarachnoid haemorrhage

Incidence

Common in adults.

Mechanism

Normally in young adults, meningeal arteries are in close proximity to the skull bone. Hence, fracture of the skull nearer to these arteries can lead to injury to these vessels.

Common Causes Encountered in Routine Medicolegal Practice

Fracture of the temporal bone—especially the squamous portion can result in rupture of middle meningeal artery or vein, leading to heavy bleeding into the temporal fossa.

Fracture of the frontal bone—can result in rupture of anterior meningeal artery or vein, leading to anterior cranial fossa haemorrhage, which in turn can press upon the frontal lobe.

Fracture of the occipital bone—can result in rupture of posterior meningeal artery or vein, leading to posterior cranial fossa haemorrhage, which can develop a large hematoma, which in turn can press upon the cerebellum and the occipital lobes. Usually it is 2 cm thick and if it is more than 2 cm, it can compress the vital centres (medulla and pons) also and lead to immediate death.

Fracture of the vault—can result in rupture of superior sagittal sinus or diploic veins and lead to hematoma formation.

Pathology

The blood collected in the extradural space may produce pressure effects due to displacement of the brain. It can also lead to increase in intracranial tension, both these in turn can result in loss of consciousness and in a few hours or days, the patient may go into coma (symptoms may resemble drunkenness symptoms). Victim may die soon if no proper treatment is given.

Clinical Findings

Following clinical findings are observed.

- Ipsilateral dilated pupil
- Contralateral paresis
- Lucid interval
- Automatic movements

Autopsy Finding

Blood clot is seen in the extradural space (Fig. 17.13A), which can be washed out easily.

Medicolegal Importance

- Victim may present with volitional activities immediately after the injury or prior to death.
- Victim may also present with Lucid interval.
- Victim may be confused for alcoholic intoxication and may be held in custody under drunkenness charge.

SUBDURAL HAEMORRHAGE (SDH)[38,39]

Subdural haemorrhage is a type of intracranial haemorrhage wherein the bleeding is under the dura mater (Figs 17.12C and 17.13B).

Incidence

Common in children and old people. It is rarely found in a pure form. Usually it is associated with subarachnoid haemorrhage.

Mechanism

Some of the possible mechanisms involved are:

- Gliding movement between dura and arachnoid mater leading to overstretching and tearing of communicating veins traversing in the subdural space.
- Injury to the brain such in form to bruises/lacerations.
- In old age, any minor trauma due to sudden jerky movements of the head can lead to bilateral subdural haemorrhages.
- Spontaneous causes.

Pathology

Since the haemorrhage is insidious in nature there may not be any clinical manifestations for a long time, except slight confusion, forgetfulness and emotional disorders, which may be mistaken for schizophrenia in a young patient, whereas in old patient for dementia. Thus, a chronic subdural haematoma (blood cyst) may be formed over the cerebral cortex. As the subdural space has no mesothelial lining, haemorrhage cannot get resolved and such blood cyst is only found at autopsy, which in an unsuspected case can be the cause of sudden unexpected death.

Site

Most commonly observed sites are:

- Frontal/parietal/temporal/basal regions of the brain.
- Unilateral or bilateral.

Classification

SDH are usually one of the following three types:

- Acute type–diagnosed only at autopsy
- Subacute type–here the symptoms manifest after 2 to 14 days
- Chronic type–here the symptoms are further delayed (after a few weeks or more).

Clinical Findings

These include the following:

- Unconsciousness (acute type)
- Ipsilateral pupillary disturbances
- Hemianopia (blindness on any one side)
- Mild headache (subacute type)
- Speech defect (subacute/chronic type)
- Urinary disturbance (subacute/chronic type)
- Lucid interval.

Autopsy Findings

Acute cases—blood clot in the subdural space and it cannot be washed out.

Chronic cases—a blood cyst may be noticed.

Medicolegal Importance

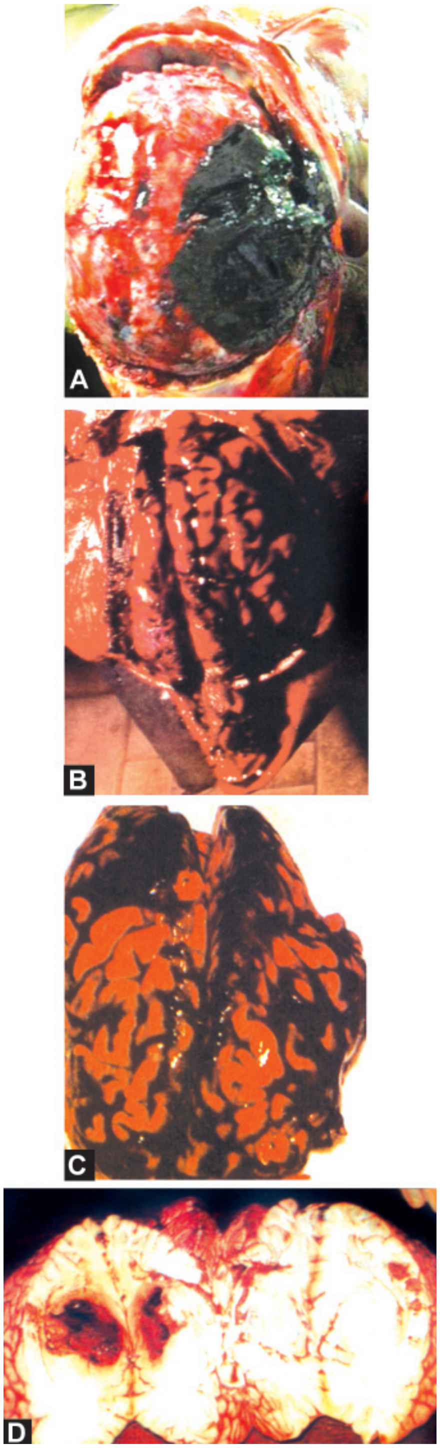
1. Chronic type can result in sudden natural death.
2. Clinical symptoms may be mistaken for:
    - Schizophrenia in young victims
    - Presenile or senile dementia in elderly victims.
3. Lucid intervals.

SUBARACHNOID HAEMORRHAGE (SAH)[40]

Subarachnoid haemorrhage is a type of intracranial haemorrhage wherein the bleeding occurs in subarachnoid space (Fig. 17.12D, Fig. 17.13C).

Specific Causes and Mechanism

Two mechanisms are prevalent in developing SAH and they are SAH of traumatic origin and SAH of spontaneous origin.



Figs 17.13A to D: (A) Extradural haemorrhage/haematoma (*Courtesy:* Dr Bhaskar Reddy K, Professor and HOD, Forensic Medicine, SVT Medical College, Thirupathi, Andhra Pradesh), (B) Subdural haemorrhage, (C) Subarachnoid haemorrhage, (D) Intracerebral haemorrhage in corpus callosum (both side) and basal ganglion on left side

SAH of Traumatic Origin

- All traumatic causes such as contusion or laceration of the brain, explosive blasts, strangulation, traumatic asphyxia, damage to the vertebral arteries due to fracture of the upper cervical vertebrae as a result of blows across the neck from behind.
- Prolonged hyperextension of the neck as with special investigation such as bronchoscopy, which may result in rents in basal vertebral arteries.

SAH of Spontaneous Origin

- Diseases like atherosclerosis and hypertension, leukemia, etc.
- Congenital defects like milliary aneurysms, Berry aneurysms of basilar arteries, which rupture with mild trauma.

Site

SAH can occur either as unilateral or bilateral, coup or contrecoup lesion initially, but soon it spreads all over the brain.

Pathology

No haematoma is formed here, as the blood is removed by lysis or by phagocytosis and the victim can recover completely.

Clinically

Sudden loss of consciousness.

Autopsy Finding

Yellowish brown discolouration of arachnoid mater.

Medicolegal Importance

Testifying trauma as the cause of rupture of aneurysm is very important and it is done by symptoms of more or less continuous headache, identified as thunderclap headache ("most severe ever" headache developing over seconds to minutes), after the trauma, till the actual rupture.

INTRACEREBRAL HAEMORRHAGE (PARENCHYMATOUS HAEMORRHAGE)

It is a type of intracranial haemorrhage wherein the bleeding occurs in the cerebral tissue (Figs 17.13D and E).

Causes

It can occur due to violence alone or due to disease of the cerebral vessels. However, the various causes that can be enumerated are as follows:

- Rupture of berry aneurysms (Figs 17.14A to C)
- Intracerebral stress or impact (vascular tear)
- Coup injury
- Frontal impact resulting in expansion of the skull, which in turn can lead to separation of the brain ventricles
- With cerebral contusion
- Movements of falx cerebri
- Skull fracture.

Size

Varies from petechial haemorrhage (<2 mm) to haematoma.

Site

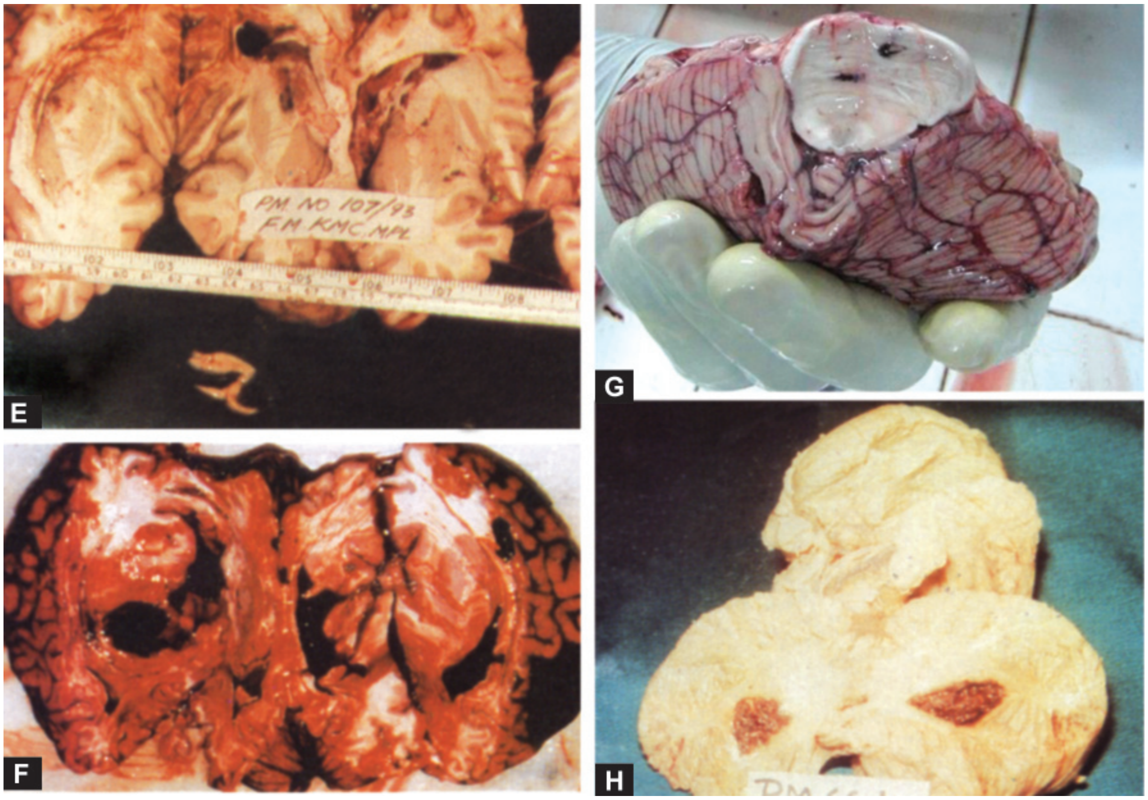
- At the junction between grey and white matter
- In the nuclear collection.

Medicolegal Importance

It is very essential to decide at autopsy whether the haemorrhage is post-traumatic or spontaneous (apoplexy). Table 17.2 depicts the differentiation.

INTRAVENTRICULAR HAEMORRHAGE

It is a type of intracranial haemorrhage wherein bleeding occurs in the brain ventricles. An isolated form is very rare (Fig. 17.13F). It usually occurs with extensive type of intracranial bleeding due to vascular malformation of choroid plexus.



Figs 17.13E to H: *Intracranial haemorrhages:* (E) Intracerebral haemorrhage (right thalamus), (F) Intraventricular haemorrhage with subdural and subarachnoid haemorrhage, (G) Pontine haemorrhage (*Courtesy:* Dr Bhaskar Reddy K, Professor and HOD, Forensic Medicine, SVT Medical College, Thirupathi, Andhra Pradesh), and (H) Intracerebellar haemorrhage (dentate nucleus)

Table 17.2: Differences between haemorrhage due to head injuries and diseases

Causes	*Head injury*	*Hypertension, aneurysm, etc*
Age	Young and healthy	Past middle age
Onset	Insidious	Sudden
Position of head	In motion	Any position
Mechanism	Coup and contrecoup	Rupture due to disease
Site	White matter of frontal or temporo-occipital region	Ganglion regions
Concussion	Present-variable	Absent
Symptoms	Consciousness to coma	Coma from beginning

PONTINE HAEMORRHAGE

Pontine haemorrhage is a type of intracranial haemorrhage wherein bleeding occurs in the pons or brain stem (Fig. 17.13G).

Causes

It usually occurs in person with high BP. Sometimes a natural intracranial haemorrhage in pons may precipitate on an accidental fall or traffic accident, and the resulting head injury makes it difficult to identify the nature of brain haemorrhage.

Types

Pontine haemorrhage could be spontaneous, severe pontine haemorrhage of single type, occupying one-third to half of substance of the pons, or traumatic haemorrhage occurring in multiple separate foci which may unite to form a large area when the victim survives for a sufficient time. Both of these types can rupture into the fourth ventricle. Primary haemorrhages in the brain stem are usually small and are seen in relation to the walls of the third and fourth ventricles and of the aqueduct. Haemorrhages in the rostra brain stem are usually more numerous and severe than those into the medulla resulting in rapidly fatal injuries.

Medicolegal Importance

In most of the victims who die after prolonged unconsciousness, brain stem injuries are often seen.

CEREBRAL OEDEMA

Cerebral oedema occurs due to a localised or diffuse abnormal accumulation of water and sodium, which increases the volume of the brain. Thus cerebral oedema is a serious and potentially fatal complication.[52]

Causes

It is caused due to:

- Increase in the intravascular pressure
- Increased permeability of the cerebral vessels, and
- Decrease in plasma colloid osmotic pressure.

- Focal oedema is almost invariably associated with and secondary to contusions and lacerations of the brain.
- Focal oedema in the brainstem is usually fatal.
- Generalised cerebral oedema occurs with diffuse brain injury.

Diagnosis

Evaluation for cerebral oedema is very essential in a case in order to prevent further progression to brain death in head injury cases. Three methods are reported to be helpful in assessing severity of cerebral oedema,[53,54] and they are:

- Brain oedema severity score (BESS) scale
- Computed tomographic (CT) scan
- Intracranial pressure (ICP).

Brain Oedema Severity Score Scale

According to Wijdicks, E F M et al, CT scan findings can help in calculation of Brain Oedema/Oedema Severity Score (BESS) Scale. Table 17.3, highlights the facts and guides in evaluation of severity of the cerebral oedema and thus the prognostic criteria.

CT Scan Features

Initial CT scanning can reveal radiologic evidence of cerebral oedema. Mainly, loss of cortical sulci or loss of sylvian fissure contours was evident in patients with a BESS between 20 and 14 (Table 17.3). Severity further is assessed by loss of CT evidence of most basal cisterns and fissures, loss of white matter discrimination, and compression of lateral or third cerebral ventricles which point towards fatality. Serial radiologic evidence of rapid progression of cerebral oedema is shown in Figure 17.14D.

Intracranial Pressure

Intra cranial pressures (ICP) between 20-60 mm Hg. are suggestive of cerebral oedema and a reading of >60 mm Hg is diagnostic of fatal cerebral oedema.

Cause of Death in Head Injuries

Most deaths are due to damage to vital cerebral areas, located around the posterior hypothalamus, midbrain and medulla. Usually respiratory failure or paralysis is followed by permanent cardiac arrest. Vital centres may be compressed or concussed directly or they may be injured by secondary changes. Another mechanism is markedly raised intracranial pressure. Other causes of death are infections, hypostatic pneumonia, pulmonary embolism and renal infection.

Table 17.3: Calculation of brain oedema severity score (BESS), based on computed tomographic (CT)* findings

Features	*Scores*
Visibility of cortical sulci	
3 CT scan slices of upper cerebral area (L/R)	6
Visibility of white matter	
Internal capsule (L/R)	2
Centrum semiovale (L/R)	2
Vertex (L/R)	2
Visibility of basal cisterns	
Sylvian fissure (horizontal-vertical, L/R)	4
Frontal interhemispheric fissure	1
Quadrigeminal cistern	1
Paired suprasellar cisterns (L/R)	2
Ambient cistern (L/R)	2
Maximal total †	22

* CT=Computed tomographic; L/R=left and right cerebral hemispheres. † In CT scan with normal findings

SPINE AND SPINAL CORD INJURIES

Causes

Fracture or subluxation of the spine is a must to get spinal cord injury (exception – Whiplash injury).

Classification

Spinal injuries are basically of four types: (i) concussion, (ii) compression, (iii) pithing, and (iv) laceration.

Concussion of Spinal Cord

Concussion of spinal cord includes two types:

1. *Railway spine* – is a condition wherein the victim develops transient paralysis without loss of sexual power. Complete

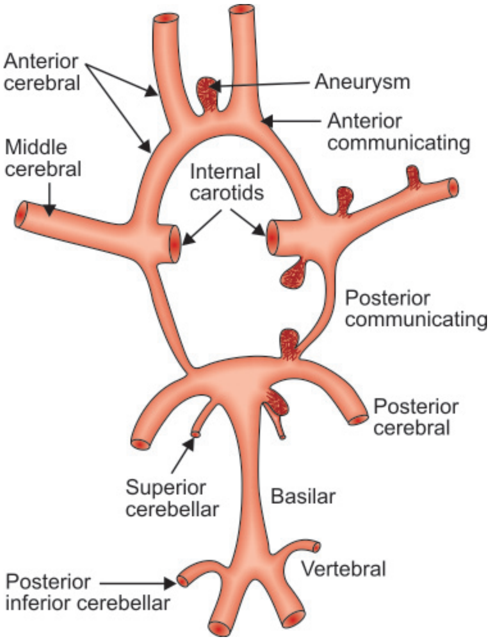


Fig. 17.14A: Circle of Willis and common sites for aneurysms

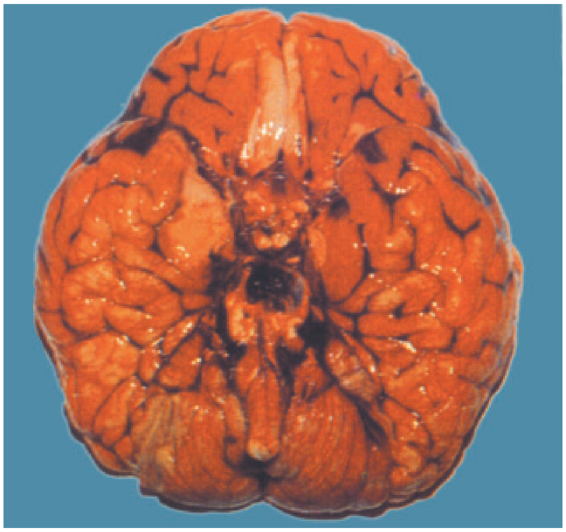


Fig. 17.14B$_1$: An extensive subarachnoid haemorrhage from rupture of berry aneurysms (*Courtesy:* Dr MR Chandran, Formerly Professor and HOD, Forensic Medicine, Medical College, Calicut, Kerala)

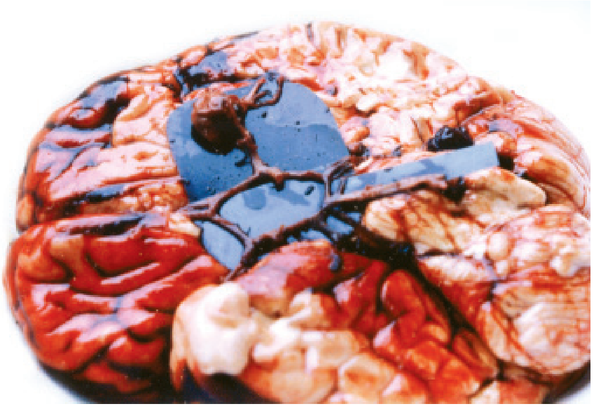


Fig. 17.14B$_2$: Basilar artery with berry aneurysms (*Courtesy:* Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

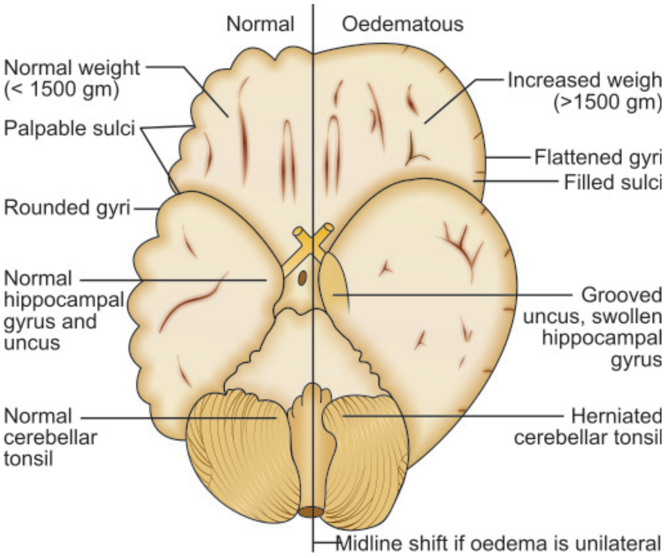

Normal
Oedematous
Normal weight (< 1500 gm)
Palpable sulci
Rounded gyri
Normal hippocampal gyrus and uncus
Normal cerebellar tonsil
Increased weigh (>1500 gm)
Flattened gyri
Filled sulci
Grooved uncus, swollen hippocampal gyrus
Herniated cerebellar tonsil
Midline shift if oedema is unilateral

Fig. 17.14C: Signs of cerebral oedema

recovery is always noticed (causes a severe blow on the back, car collision, railway accidents, etc).

2. *Whiplash injury* – With a severe blow to chin from below with hand or blow on the brow by dashing the windscreen of a car resulting in hyperextension of head, producing dislocation at C4-C6 levels (Figs 17.15A and B, 17.16). Medicolegally it is important as the symptoms are usually diagnosed as hysterical. At autopsy haemorrhage in substance of spinal cord is usual.

Whiplash injury is also caused by the impulsive movement of the head and neck suddenly jerking in one direction and then rebounding. Whiplash may occur if the head and neck move either forwards and then backwards or from one side to the other. The term whiplash was introduced by Crowe in 1928 to describe this whipping motion rather than the condition itself.

This whipping movement is often involuntary and a result of a sudden impact that may be caused by a vehicle accident or whiplash playing contact sports. Rear-end car accidents cause over 97 per cent of whiplash injuries.

Infant Whiplash-Shake Injury Syndrome

A trauma suffered by small children as the result of deliberate abuse, also known as *'Shaken-Baby syndrome'*. Infants may suffer neurological impairment, seizures, haemorrhages and contusions of the high cervical cord all of which contribute to fatality.

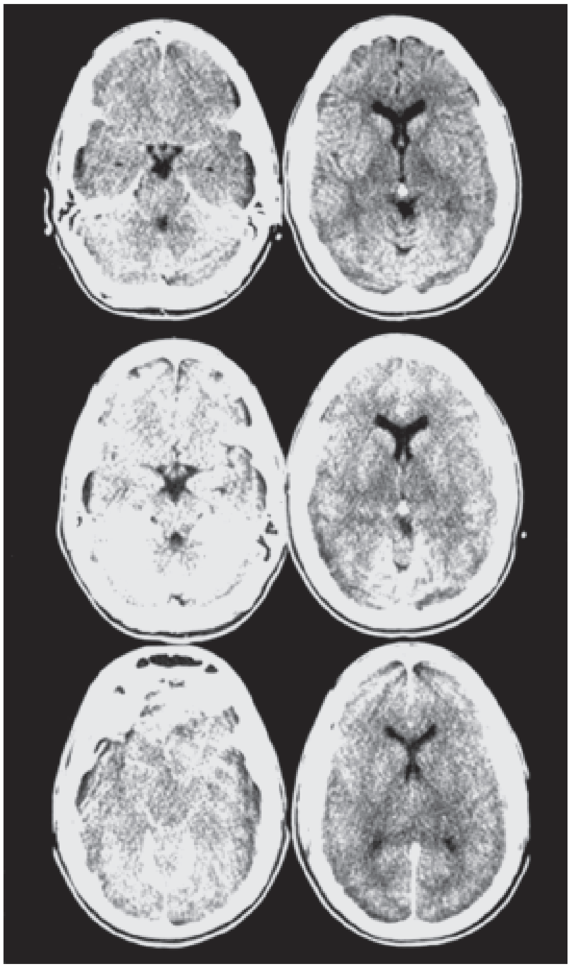
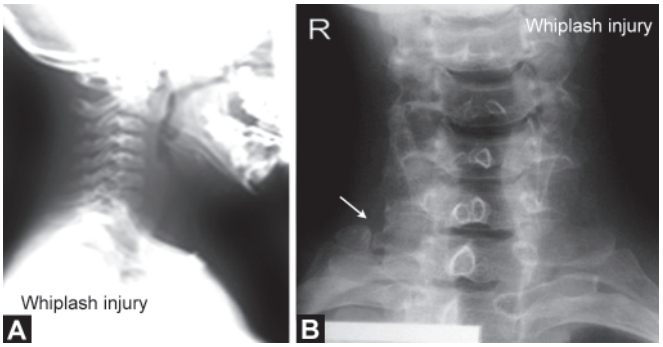


Fig. 17.14D: Serial CT scans of brain with progressive cerebral ooedema. *Top row* – Normal. *Middle row* – Disappearance of cortical sulci and white matter discrimination of internal capsule; third ventricle is barely visible. *Bottom row* – Complete compression of basal cisterns, compression of lateral ventricles, and attenuation of middle cerebral artery (representing clot). (At this time, clinical criteria for brain death were fulfilled)


Whiplash injury
A
R
Whiplash injury
B

Figs 17.15A and B: X-ray showing whiplash injury

Medicolegal Significance of Whiplash Injury

In United States, if the whiplash trauma is due to an accident, one can claim legally for pain and suffering, loss of earnings

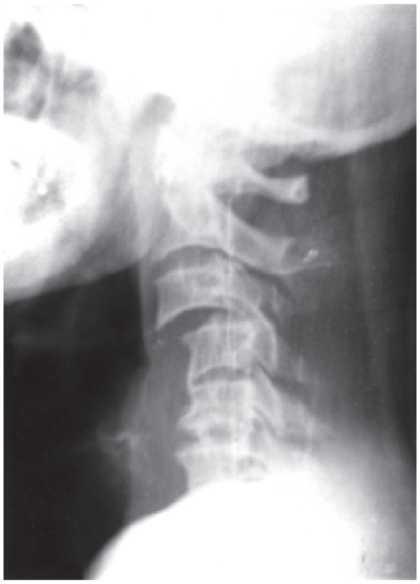


Fig. 17.16: Radiograph showing fracture dislocation of C3-C5

and recover costs for medical treatment and prescriptions, every penny going towards rebuilding ones own life.

Compression of Spinal Cord

Compression of spinal cord is usually due to fracture dislocation, usually at levels such as C4-C6, T3-T6, T10-L3, etc. Victim may die in 24 hours or survive with permanent paralysis.

Pithing

Pithing is killing by pushing a fine needle into nape of neck between base of skull and first cervical vertebra and is a concealed punctured wound.

Laceration of Spinal Cord

Twisting the neck can lead to laceration of spinal cord, which in turn can lead to death without evident external injury.

Examples

- This is common in killing unwanted children (infanticide)
- This can happen in wrestling.
- Firearm injury may also cause spinal laceration even when the missile has not entered the cord, but just has brushed the neck (near missed trajectories).

INJURIES TO NECK AND NECK STRUCTURES

Injuries to neck and neck structures include injuries due to hanging, strangulation, throttling, sharp weapon injuries, etc. They are as follows:

- Scratches and bruises — as occurring with asphyxial death namely, the ligature mark of hanging, strangulation, etc and the nail markings of throttling.
- Injury to hyoid bone — this is usually fracture of greater cornu at the junction of outer one-third with inner two-third. This is common among the victims of age group 40 years and above. In hanging and ligature strangulation, the fractured fragments are always displaced outwards, while in throttling, the fracture fragments are always displaced inwards
- Lacerations of the neck and its structures
- Incised wounds of the neck or throat — Cutthroat wounds are common in the neck with sharp weapons. They could be homicidal or suicidal. The basic differences between these two are depicted in Table 17.4.

Medicolegal Importance

Meticulous examination of neck wounds can give clue regarding motive of wounding, i.e. whether suicidal, homicidal or accidental.

INJURY TO CHEST/THORAX AND THORACIC STRUCTURES[2,4-11,41,42]

Incidence

Commonly seen among adults.[2, 4-11] Rarely seen among the children.[7] Motor vehicle collisions (MVC) represent the most common cause of major thoracic injury among emergency department (ED) patients.[41,42] Several factors associated with a higher risk of thoracic injury are:

- High speed
- Not wearing a seatbelt
- Extensive vehicular damage
- Steering wheel deformity.

Increased mortality and morbidity is associated with multiple rib fractures, increased age, and higher injury severity scores.

Classification

Various types of chest injuries encountered in routine medicolegal practice (Figs 17.17A to C) are:[41,42]

- *Concussion of chest*—can lead to death.
- *Contusion of chest*—resulting in pleurisy or pneumonia or tuberculosis.
- *Fracture ribs*—commonly 4th to 8th ribs are involved and called knobbing fracture ribs
- *Flail chest injury*—wherein multiple bilateral rib fractures are seen.
- *Injury to lungs*—such as bruising, laceration, and stab wounds, etc.
- *Injury to heart due to blunt/and sharp force trauma,* namely:
  a. Buckled sternum

Table 17.4: Differences between suicidal and homicidal cutthroat injury

Particulars	*Suicidal*	*Homicidal*
Side of the neck	Left side in right handed	Any side–Rarely bilateral
Level of the wound	Above thyroid cartilage	Below thyroid cartilage
Hesitation cuts	+ve	–ve
Tailing of wound	+ve	–ve
Defense cuts	–ve	+ve
Cadaveric spasm of hand	+ve	–ve
Injury to carotid artery	–ve	+ve
Location	Isolated	Not so

  b. Bruising/crush injury/rupture/avulsion of heart.
  c. Cardiac tamponade/hemopericardium (i.e. accumulation of 250 ml or more blood in pericardial sac), may present with volitional acts.
- Injury to major blood vessels like aorta/pulmonary artery/inferior vena cava (IVC), etc.
- Injury to trachea and oesophagus.

Medicolegal Importance

Examination of the wound can help to assess motive. Usually they are homicidal or accidental, but rarely suicidal also. Mostly these injuries are of grievous nature, causing death immediately or remotely due to the complications.

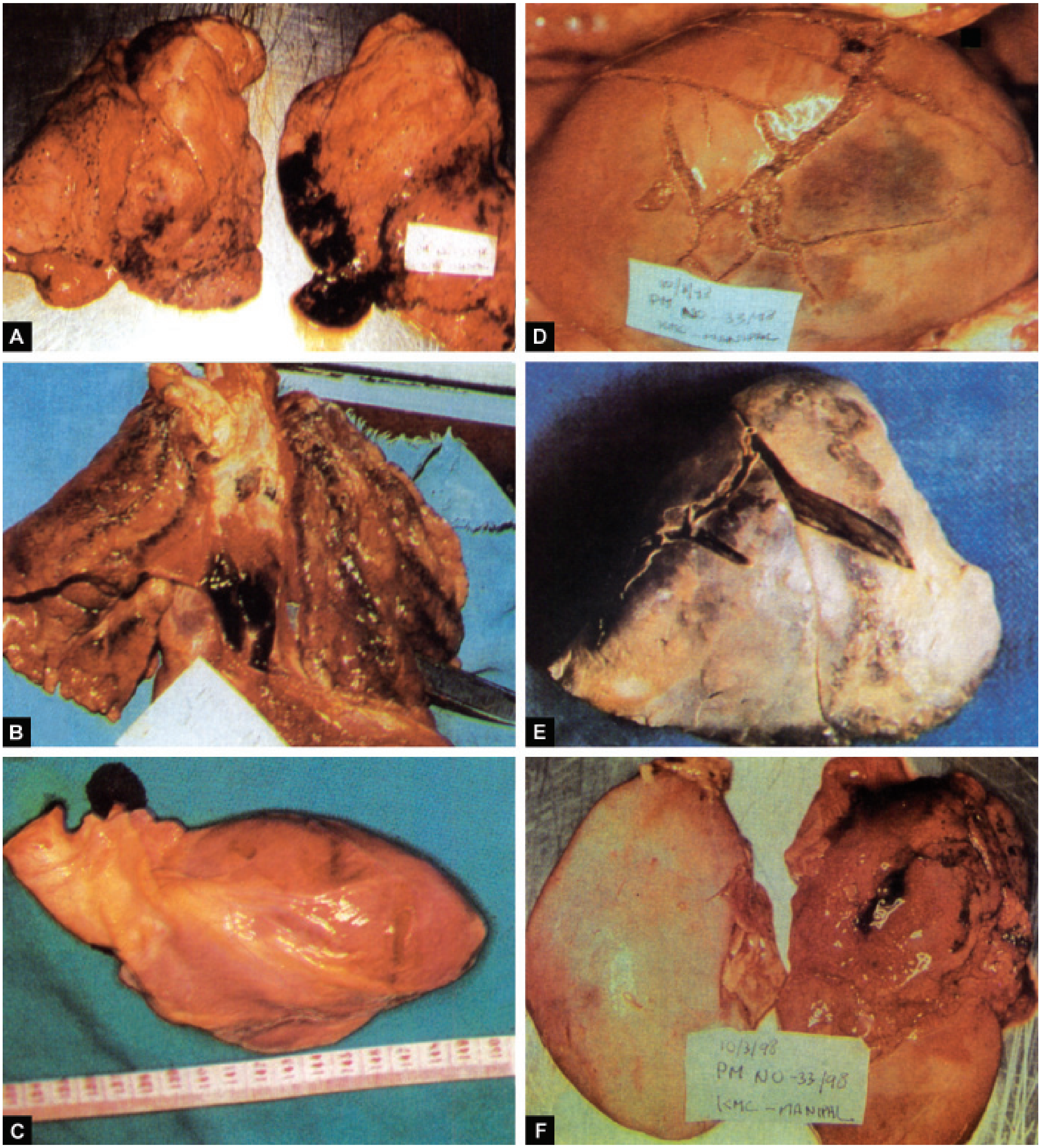
ABDOMINAL INJURIES[2,4-11,43-48]

Commonly among adults but seen rarely in children.[2,4-11]

Classification

Various types of abdominal injuries encountered in routine medicolegal practice (Figs 17.17D to F) are:[43-48]

- Incised wounds, which could be non-penetrating or penetrating, leading to death due to shock, haemorrhage,



Figs 17.17A to F: *Other visceral injuries:* (A) Contusion of the lungs, (B) Stab injury of the lung, (C) Stab injury of heart, (D) Lacerated liver, (E) Lacerated lung, (F) Contused and lacerated kidney

sepsis or injury to vital organs such as liver, spleen, pancreas, kidneys, etc.
- Blunt injuries such as contusions or laceration of viscera such as stomach, intestines, pancreas, liver, spleen, etc.

Causes

Probable causes could be blows/ kicks over the abdomen, fall on a pointed object, stab injuries, road traffic accident injuries, crush injuries, etc.

Complications

Complications could be immediate or remote, which could be any one of the following:

- Haemorrhage
- Peritonitis
- Sepsis
- Vagal inhibition, etc.
- Death due to any of these complications when not treated properly.

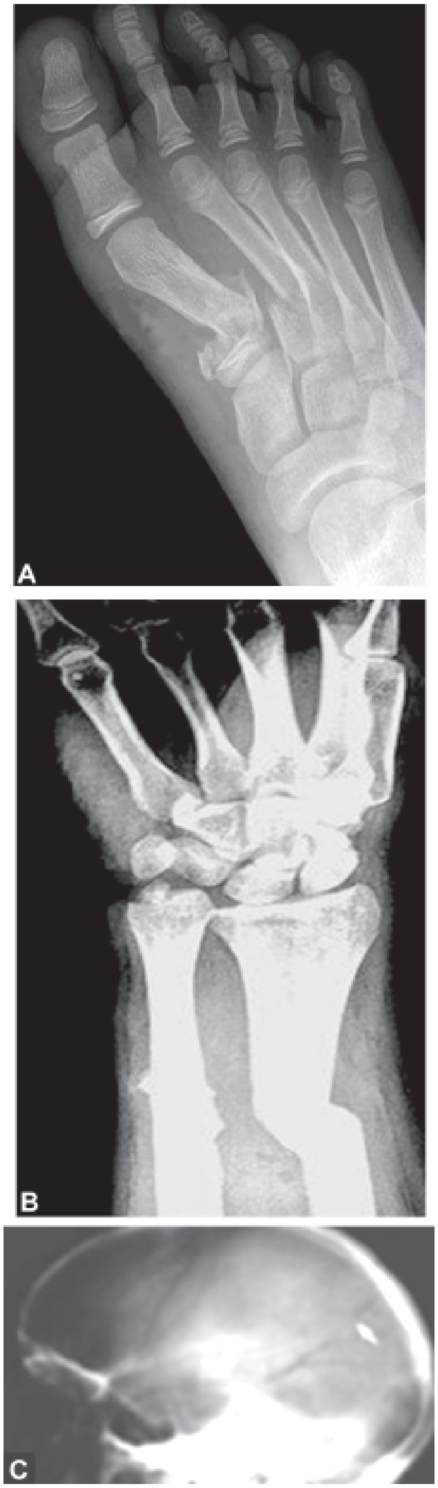
LIMB SKELETAL BONE INJURIES[7,48-51]

Include all types of fracture bones of the extremities.

Types of Bone Fractures

In orthopedic medicine, fractures are classified as:

- *Closed or open (compound)* – Closed fractures are those in which the skin is intact, while open (compound) fractures involve wounds that communicate with the fracture and may expose bone to contamination. Open injuries carry an elevated risk of infection; they require antibiotic treatment and usually urgent surgical treatment (debridement). This involves removal of all dirt, contamination, and dead tissue.
- *Simple or multi-fragmentary (formerly comminuted) (Figs 17.18A to C)* Simple fractures are fractures that occur along one line, splitting the bone into two pieces, while multi-fragmentary fractures involve the bone splitting into multiple pieces. A simple, closed fracture is much easier to treat and has a much better prognosis than an open, contaminated fracture. Other considerations in fracture care are displacement (fracture gap) and angulation. If angulation or displacement is large, reduction (manipulation) of the bone may be required and, in adults, frequently requires surgical care. These injuries may take longer to heal than injuries without displacement or angulation. Lactate dehydrogenase (LDH) levels increase when the bone breaks.
- *Compression fracture* — An example of a compression fracture is when the front portion of a vertebra in the spine collapses due to osteoporosis, a medical condition which causes bones to become brittle and susceptible to fracture (with or without trauma).
- *Other types of fracture* — This includes plethora of fractures as listed below:
  - Complete fracture: A fracture in which bone fragments separate completely.
  - Incomplete fracture: A fracture in which the bone fragments are still partially joined.
  - Linear fracture: A fracture that is parallel to the bone's long axis.
  - Transverse fracture: A fracture that is at a right angle to the bone's long axis.
  - Oblique fracture: A fracture that is diagonal to a bone's long axis.
  - Compression fracture: A fracture that usually occurs in the vertebrae.



Figs 17.18A to C: (A) Fracture of metatarsals; (B) Arm with compound fracture, (C) Skull fracture across line of meningial artery (arrow)

  - Spiral fracture: A fracture where at least one part of the bone has been twisted.
  - Comminuted fracture: A fracture causing many fragments.
  - Compacted fracture: A fracture caused when bone fragments are driven into each other
  - Open fracture: A fracture when the bone reaches the skin
  - Bug fracture: A fracture when the bone is in place, but the fracture has the appearance of a crushed insect.

Special Considerations for Children

In children, whose bones are still developing, there are risks of either a growth plate injury or a greenstick fracture.

- A greenstick fracture occurs because the bone is not as brittle as it would be in an adult, and thus does not completely fracture, but rather exhibits bowing without complete disruption of the bone's cortex.
- Growth plate injuries, as in Salter-Harris fractures, require careful treatment and accurate reduction to make sure that the bone continues to grow normally.
- Plastic deformation of the bone, in which the bone permanently bends but does not break, is also possible in children. These injuries may require an osteotomy (bone cut) to realign the bone if it is fixed and cannot be realigned by closed methods.

Incidence

Common in both adults and children (green-stick fracture).

Causes

- Direct violence (e.g. blows)
- Indirect violence (e.g. fall).

Complications

Haemorrhage, shock, fat embolism, sepsis and death when not treated properly.

Medicolegal Importance

- All fractures are grievous hurts.
- Fractures could be antemortem or postmortem in origin (they may occur shortly before or after death) – Usually an antemortem fracture shows blood clot, which is not seen, in postmortem fractures.

JOINT INJURIES

Joint injuries include dislocation, sprain, punctured/incised/lacerated wounds.

Medicolegal Importance

Fractures of bones or dislocations of joints are grievous hurt.

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Chapter 18

Transportation Injuries

Amongst all traffic accidents, road traffic accidents (RTA) claims largest toll of human life and tend to be the most serious problem world over. Almost 1.2 million people die all over the world each year due to RTA, while about another 50 million are injured.

Any form of transport can lead to injury and to death, but the most common involves the road, rail and air transport systems.[1,4]

TRAUMA IN ROAD TRAFFIC ACCIDENTS

A majority of medicolegal autopsies in India are carried out on the victims of vehicular accidents.[5-8] RTA comprise of mostly injuries to the limbs, face, externally; while more commonly head sustained internal injuries.[9] A careful examination of injuries sustained is necessary for the reconstruction of the accident. From the nature of the injuries inferences can be drawn regarding the relative positions of the victim and the vehicle at the time of accident. Moreover in *'hit and run'* cases, the nature of injuries and collection of trace evidence from the decedent will help to connect the suspect vehicle with the crime. It may also be possible to give an opinion as whether the vehicle had run over the victim.[1-4]

The trauma sustained in transportation accidents can be classified according to the victim involved into:[1-8]

- Trauma sustained by pedestrian
- Trauma sustained by cyclist/motor-cyclist
- Trauma sustained by occupants of a vehicle.

Trauma Sustained by Pedestrians

Pedestrians struck by a vehicle may sustain three groups of injuries (Figs 18.1A to C), *primary impact injuries, secondary impact injuries* and *tertiary Injuries.*

Primary Impact Injuries

When a moving vehicle hits a person walking along the street, parts of the vehicle will come into forceful contact with the body of the pedestrian during the impact thus producing patterned injuries and these constitute the primary impact injuries. The pedestrian can be hit from the front, behind or sides (Figs 18.1B and C). As the vast majority of impacts are with the front of the vehicle and parts of it, usually injuries produced are patterned injuries, and are due to the bumper, radiator grill, headlights, etc. A hit by the bumper of a car produces fracture—sometimes wedge-shaped—of the bones of leg, commonly known as *bumper fractures*. In *hit and run* cases, in which such an injury has occurred, the height of the bumper from the ground can be discovered by measurements taken from the heel to the fracture site. This should be taken at the time of the accident, – usually this is little less than the actual height of the bumper from the ground, as the front of the car and the bumper dip down on applying the brakes suddenly.[1-4,6] Radiator grills and headlight rims may produce patterned abrasions and contusions. Pieces of glass and particles of paint recovered from clothing or the body will help to connect the vehicle with the crime[6,7] (Figs 18.2A to G). The detection of a primary impact injury will help to find out the relative position of the pedestrian and the vehicle. This is very important either to corroborate or refute the version of the eyewitnesses of the incident.

Secondary Impact Injuries

After the primary impact if the initial blow lies below the centre of gravity of the person – which for all intents and purposes is the umbilicus – the feet of the victim slide away and the whole body is lifted forcibly off the ground and thrown onto the vehicle into its path of movement. Thus with children the pattern of injury is often different as their centre of gravity is placed lower in their body.[1-4]

The head may then strike the windshield or a pillar of the vehicle, and the torso may strike the bonnet or other parts of the car. During this impact, a set of injuries can be produced (Fig. 18.1B). The particles of the broken wind-shield, parts of the bonnet, etc. can produce contusions, lacerations, etc. After the secondary impact the victim will be forcibly thrown onto the ground.

Tertiary Impact Injuries (Synonyms: Secondary Injuries)

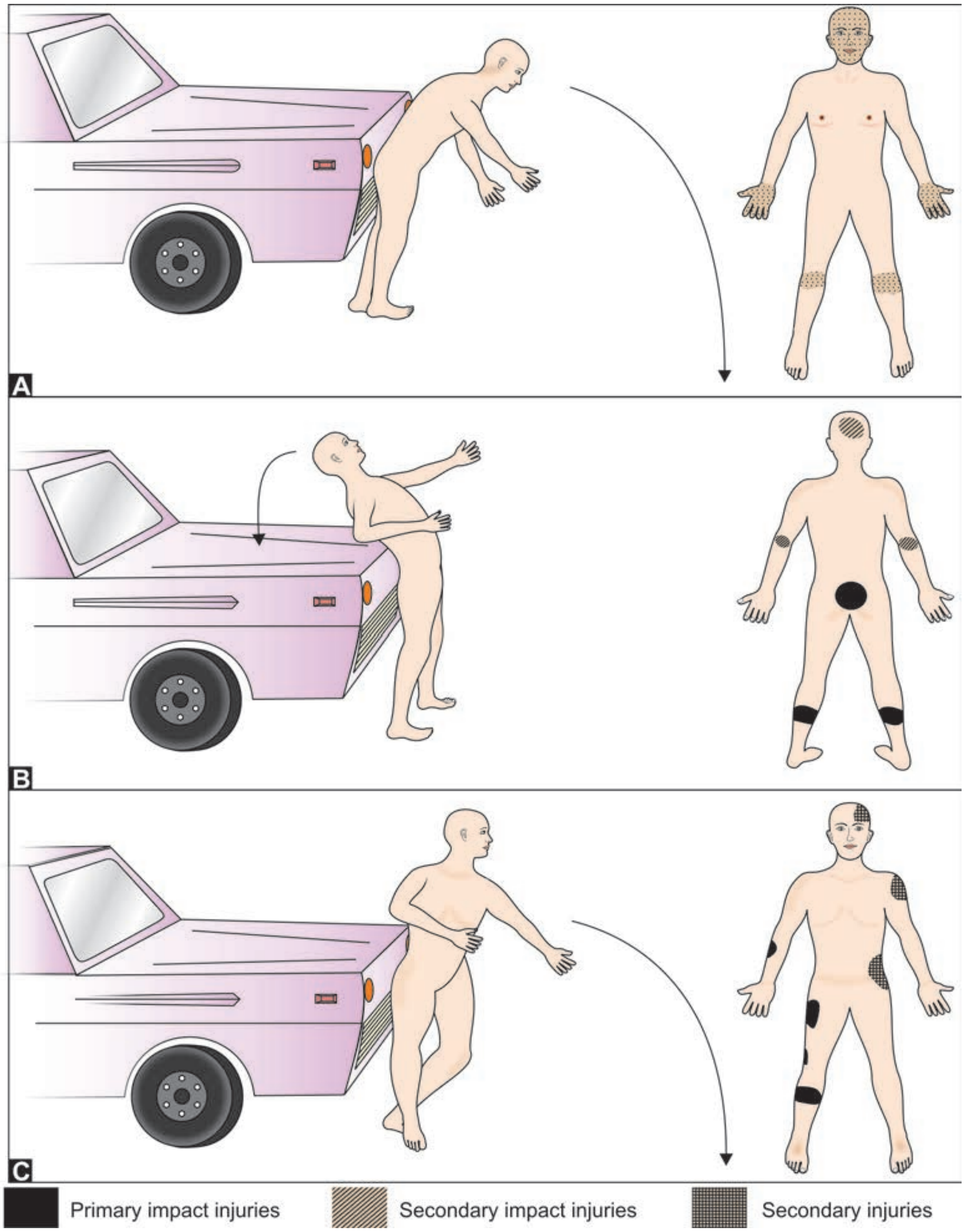
These injuries arise when the victim's body then strikes the ground after the secondary impact[2-4,6] (Figs 18.1A and C). Head injuries are the most common injuries of this group and death may be as a result of the later ground-impact injuries.

The victim may also be sometimes run over by the vehicle producing crush injuries.[2,4,6] *Tyre marks* may be seen on the body if he or she is run over and if the thread of the tyres is not worn out. If tyre marks are seen, the exact measurements and patterns have to be noted. A photograph of the tyre mark with a scale included is to be taken and will help in appropriate comparisons with the marks on the body.

Trauma Sustained by Cyclist/Motorcyclist

The injuries will be similar to those sustained by the pedestrian, but the primary impact will be usually against some part of the cycle or motorcycle. Secondary impact and tertiary injuries are more severe. Examination of the cycle is also necessary for the reconstruction of the accident. Trace evidence should be collected from the cycle and from the body of the victim.

The incidence of injury and death amongst motorcyclists is far higher than among car drivers particularly in young adults



Figs 18.1A to C: Dynamics of transportation pedestrian injuries

than in the elderly. This has been decreased to some extent by the compulsory wearing of protective gear especially helmets in a number of countries.[1,2,4-6,8,9,12]

Trauma Sustained by Occupants of Vehicles

During a collision, the occupants of a speeding vehicle will sustain different types of injuries on various parts of the body depending upon the force of deceleration and position in the vehicle as listed.

Injuries can be sustained by:

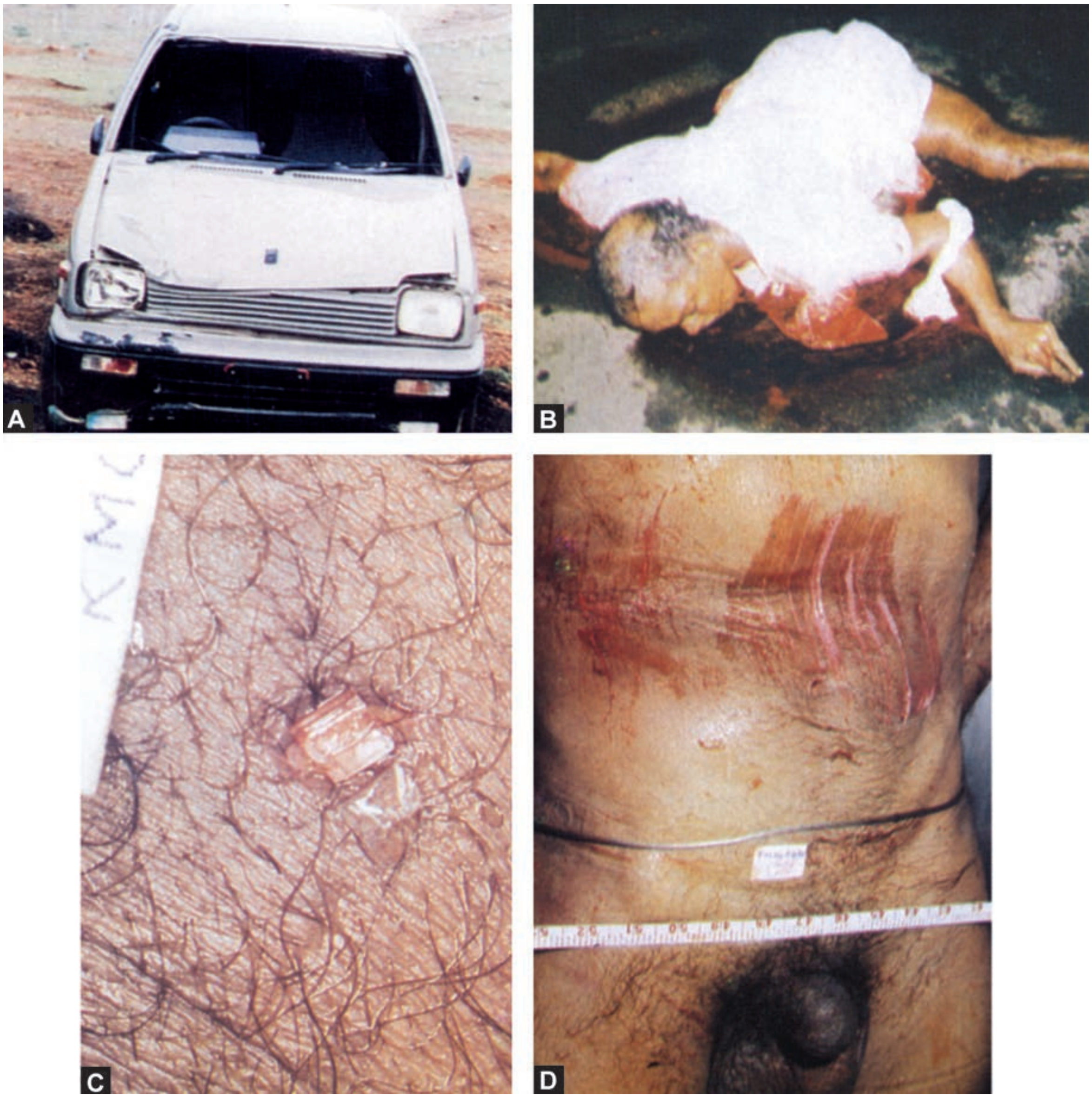
- Driver
- Front seat occupants
- Back seat occupants.

Driver

Driver may sustain an impact on to front of the chest against the *steering column*, especially the *horn boss* resulting in several fractures of the ribs and sternum, contusions and lacerations to lungs and heart, rupture of the aorta, etc. The head may strike against the windshield producing head injury. Force will be transmitted upwards from the pedals causing fracture of leg and thighbones.[2-4,8] It is the experience of the author that dislocation of the hips joint is also observed in such accident due to the transmission of upward force through the pedals.[6,7] Sometimes the vehicle's occupant may be thrown out of the vehicle, if the door of the vehicle opens during the collision.[1-8]

Front Seat Occupants

The front seat occupants can sustain head and chest injuries by striking against the windshield and dashboard. Due to the sudden deceleration, acute flexion or extension of the neck can occur resulting in the dislocation of cervical vertebrae and injury to spinal cord. This is called a *whiplash injury*.[1-8] These injuries have been greatly reduced by the *compulsory* wearing of *seat belts* in a number of countries.[2-6]



Figs 18.2A to D: *Pedestrian injuries in a hit and run case*—(A) Offending vehicle with damage on the bumper, headlight, bonnet on right side, (B) Scene of crime—position of the victim, (C) Primary impact injury above the right knee with glasspiece embedded in it, (D) Secondary impact injuries over the abdomen

Back Seat Occupants

The back seat occupants can sustain head and chest injuries by striking against the front seat. *Whiplash injuries* are also possible. The passengers can also be *thrown out* of the car. *Seat belts* have also been made compulsory for other car occupants and have also reduced the rate and severity of injuries sustained in collisions.[2-6]

RAILWAY DISASTER TRAUMA

Ever since the railway engine was invented, a railway disaster has been associated with numerous fatalities. Thousands of people have been killed over the years in railway incidents, as this constitutes a common mode of public transport system all over the world, especially in India, having a large railway network with unprotected railway crossings.[5-8]

Classification

Railway disaster trauma is classified into:[2-8]

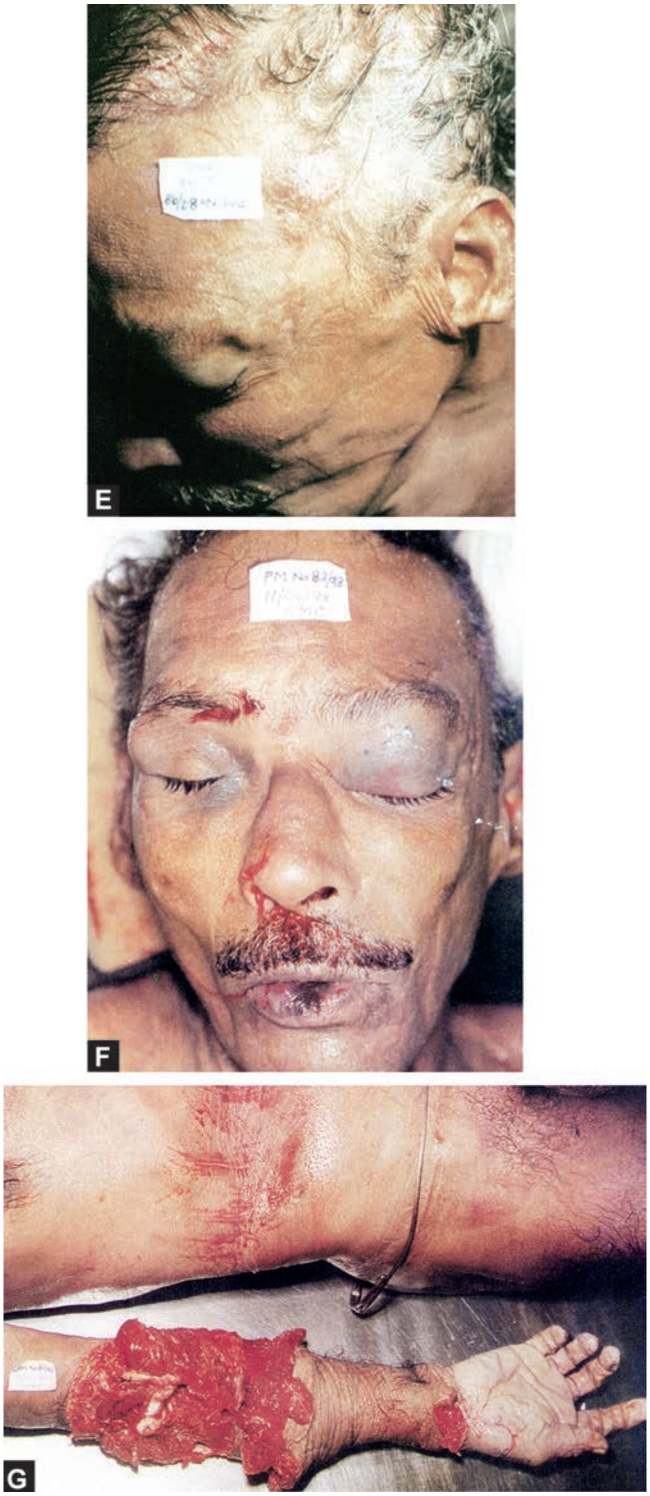
- Collision and derailment trauma
- Falling from a speeding train
- Hit by a speeding train (Figs 18.3A to F).

Collision and Derailment Trauma

The victim may show injuries to head, spine and legs similar to those found in automobile accidents. Persons sleeping on bunks may be thrown forward and may sustain dislocation or fractures of the vertebral column. Broken fragments of the carriage may produce penetrating injuries.

Falling from a Speeding Train

The victim may sustain multiple injuries of different types in various parts of the body. The bodies will be recovered from

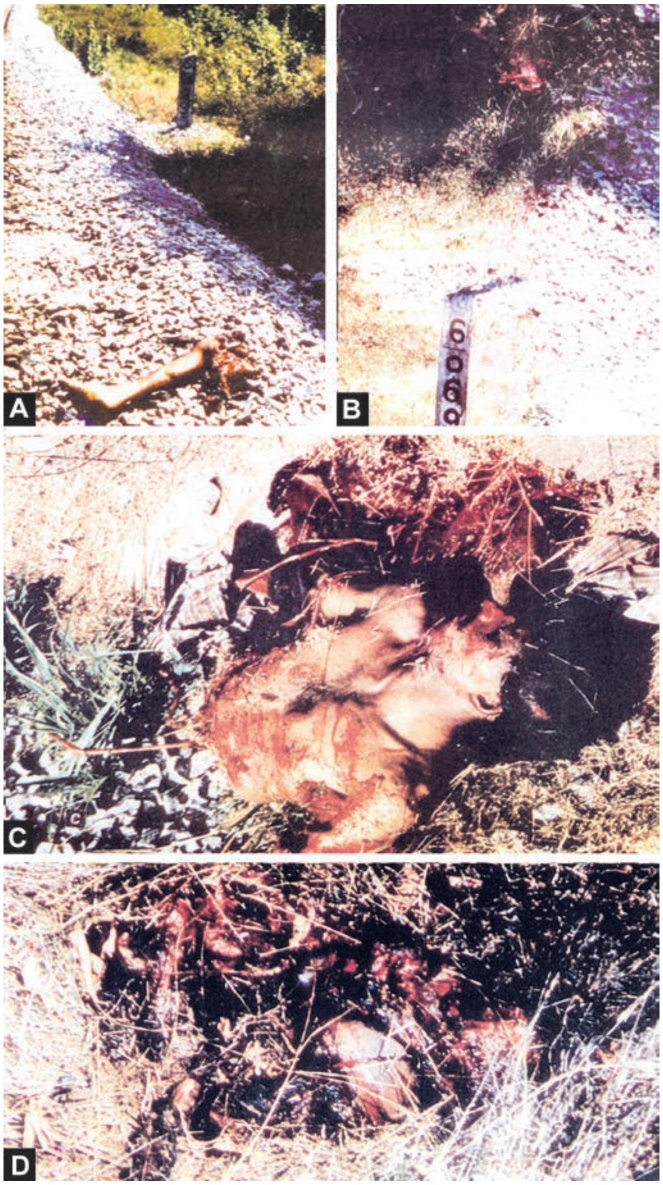


Figs 18.2E to G: *Pedestrian injuries in a hit and run case:* (E) Secondary impact injury over the scalp on left side, (F) Secondary injuries over the face, and (G) Run over injury of the right arm and forearm

the outside of the track unless the victim fell in between the carriages.

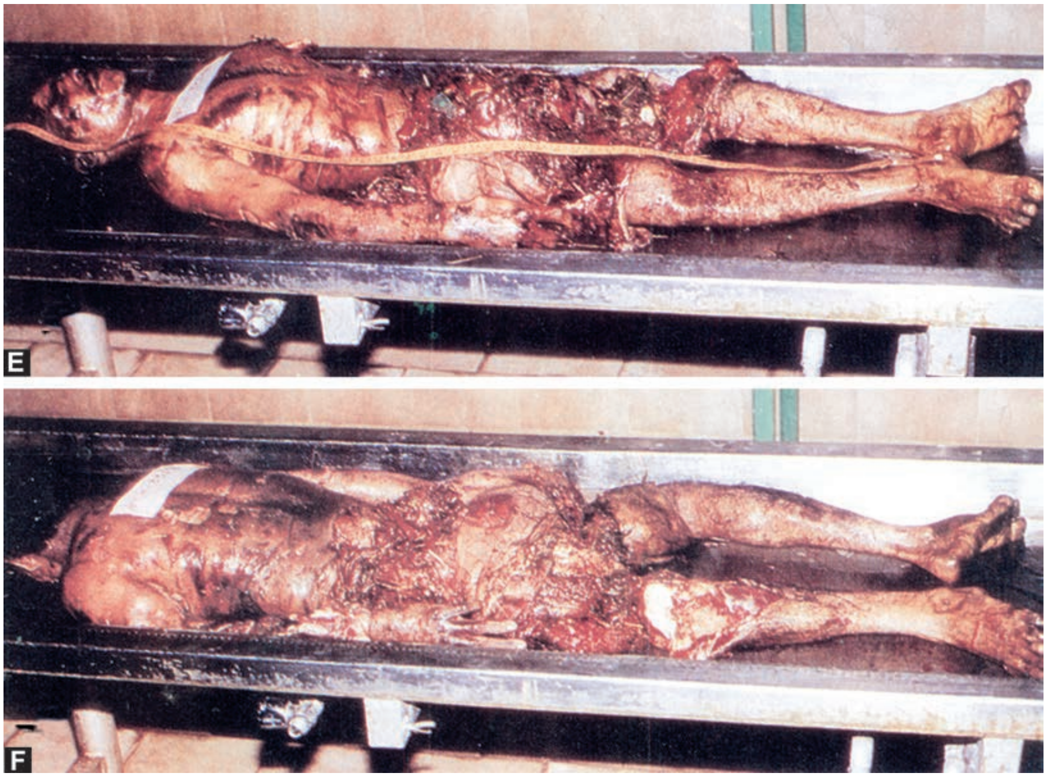
Hit by a Speeding Train

A speeding train will hit the victim while crossing the track or walking along the railway line or jumping in front of it. The engine of the train may have a front bumper known as cattle trap. During the primary impact, the victim will sustain injuries caused by the contact with the cattle trap and other parts on the front of the engine. These injuries will be similar to those seen in very severe automobile accidents. During the autopsy, the distance from the heel to each injury is to be measured for future reconstruction. Immediately after the primary impact the victim will be thrown forward and run over. In such cases, the injuries sustained will be of a dismembering nature. The decapitation and amputation of torso or limbs, etc. with local tissue destruction, usually with grease, rust or other dirt soiling of the damaged area (Figs 18.3A to F), etc. are obvious features of hit by speeding train. A wide band of contusion collar on both side of the amputation area is the frequent finding.[2,6]



Figs 18.3A to D: *Railway trauma:* Scene of crime—amputated torso and limbs of the victim with massive local tissue destruction, soiling of damaged area with grease, rust, dirt etc, and scattered on either side of the railway track suggestive of hit by a speeding train

Track workers may be run down and die from electrocution by usually accidental contact with high voltage overhead cables. Pathology of all these is no different from accident elsewhere,



Figs 18.3E and F: *Victim of run over by speeding train:* Remains of the victim. Reconstruction of the body in the mortuary

the interest lying in the occupational epidemiology and preventive aspect. Remains of the victim scattered on both sides of railway tract (Figs 18.4A to D) are seen, the distance between these two are also to be measured and compared with the breadth of the railway track. In the absence of other injuries this indicates that the victim was lying across the track. In such cases, the body has to be examined for signs of violence, intoxication, natural diseases, etc.[2,5,6,8]

Medicolegal Importance of Railway Disaster Trauma

A careful autopsy has to be conducted on a body recovered from the railway track to determine whether the injuries are antemortem or not. The majority of the deaths may be accidents or suicides, but the dead body of homicide victims may also be placed on the rails to simulate suicide. As the death is *instantaneous* in run-over cases, the *vital reaction* in the injuries will be minimal or non-existent.[1,2,4,5,7]

Postmortem injuries are usually dry and parchmentised with little, if any, evidence of bruising around them or erythematous changes of their edges, and there is no profuse bleeding that would be expected from the more severe injuries.[2,4,5] As in the case of automobile accidents, patterned injuries may be discernible due to the primary and secondary impact but they will be of a very severe nature.[2,7]

A fairly common railway fatality is the suicide with the victim laying himself or herself in front of an approaching train. A clean decapitation (Figs 18.5 and 18.6) with no other injuries elsewhere is suggestive of suicide.[2,5,7,8] In all such case it is essential that the past medical and social history of the decedent is looked in to, to find out if there is any known immediate cause for the person wishing to take their own life.

AIR DISASTER TRAUMA

Between the two *World Wars* physicians made little investigation of aircraft accidents. Early in *World War II*, however, scientific approach to the pathologic and medical investigation of aviation accidents was developed and since then it has grown into a specialty of its own.[4,6]

Incidence

Most of the aircraft accidents occur either during landing (55%) or take-off (22%) with all rest of the instances actually in air during flight (23%).[2]

Causes

Common causes are bad weather, poor visibility, engine trouble, striking by lightning strokes to aircraft, bomb explosions or fire accidents within aircraft, etc.[2,6] By far the commonest cause is *human error*, usually in combination with the other problems.[5,6]

Mechanism

Sudden *deceleration* on crashing, resulting in break-up of the aircraft and injuries to occupants.

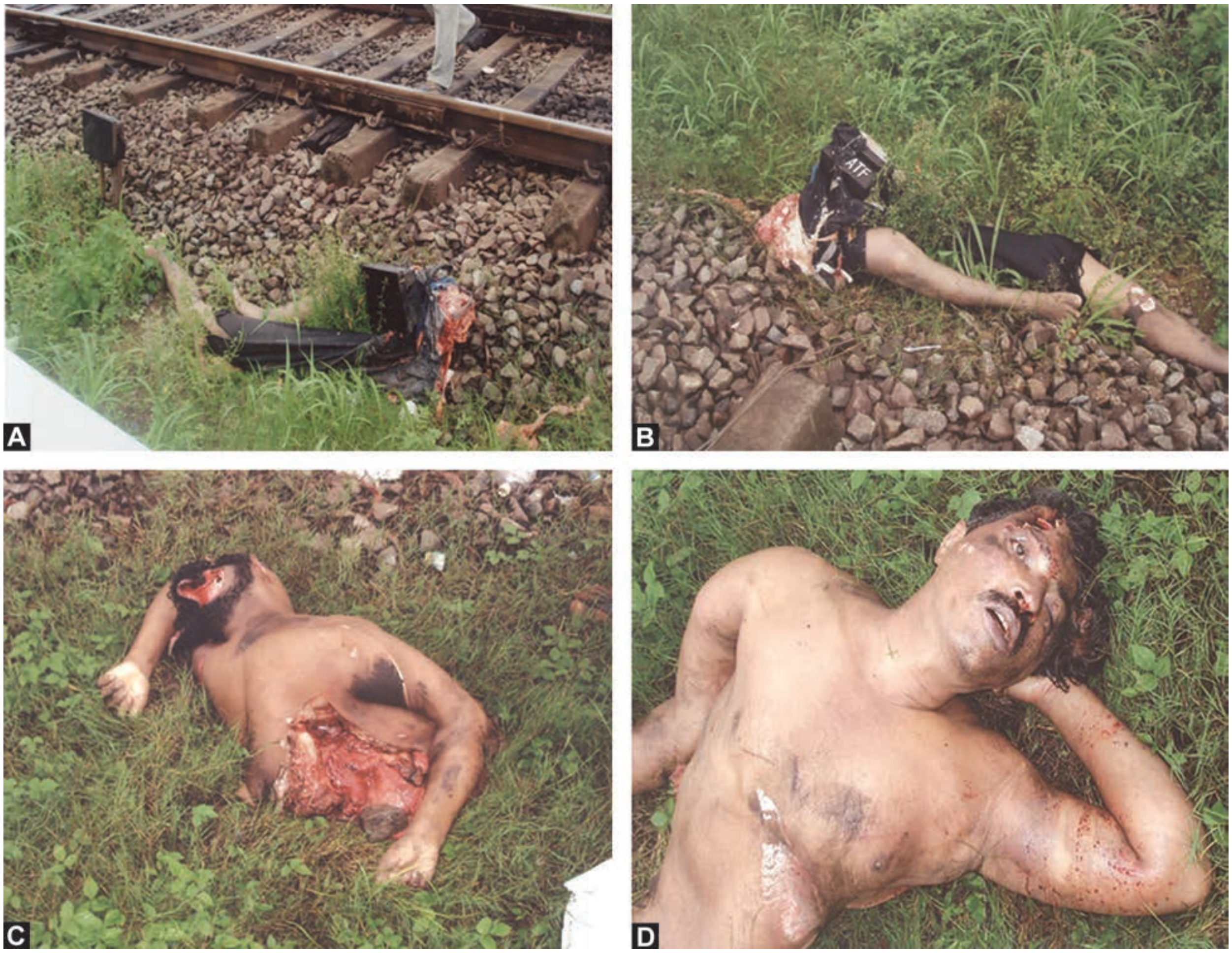
Classification

Aircraft injuries are classified into two types.[2-4,6]

Crash Accident Injuries

Usually during landing or take-off the passengers are secured to their seats by the seat belts. The forward momentum developed due to crashing pushes the head and torso of the passenger on to the backrest/back of the seat in front and results in various injuries such as:

- Fracture of skull, cervical vertebrae (due to *hyperextension*).
- Laceration of abdominal viscera (liver, spleen, kidney, etc.).
- Laceration of abdominal major blood vessels (aorta).



Figs 18.4A to D: *Railway Trauma:* Scene of crime-amputated torso and limbs with massive local tissue destruction, soiling of damaged area with grease, rust, dirt etc and scattered on either side of the railway track, suggestive of run over by speeding train (*Courtesy:* Dr Ritesh G Menezes, Associate Professor of Forensic Medicine, KMC, Mangalore)

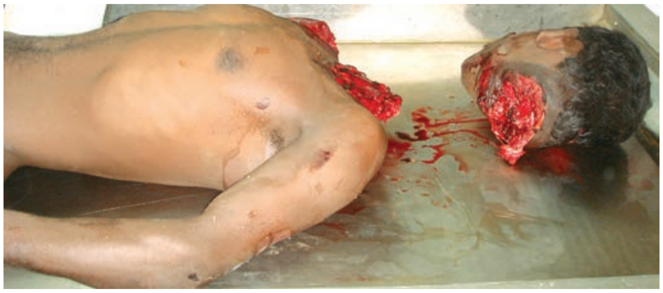


Fig. 18.5: *Railway Trauma:* A clean decapitation wound, with wide band of contusion collar on either side of severed area of the neck, suggestive of *suicide*

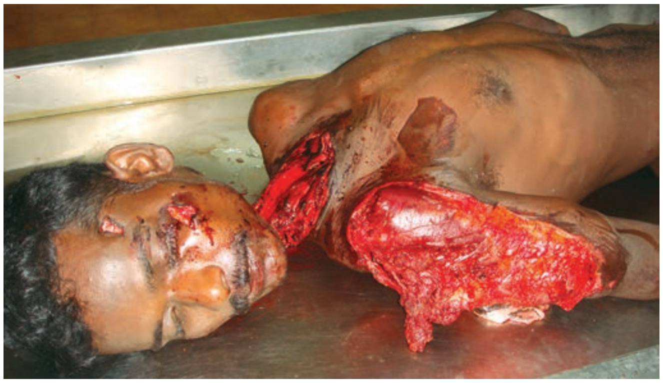


Fig. 18.6: *Railway Disaster Trauma:* Decapitation wound, with wide band of contusion collar on either side of severed area of the neck, and injury to other part of the torso suggestive of *non-suicide*

- Burns injuries (usually about 20% of fatal crashes result in fire and conflagration).

Flight Accident Injuries

The flight cabins are usually pressurised to prevent anoxia. If a door or window breaks, the pressures inside cabin drops down and results in anoxia leading to death. The air that gets sucked out in such occasions can blow away the passengers from cabins tearing them into pieces.

Medicolegal Importance

- All injuries are considered as examples of *mass disaster injuries.*

- Identification of the deceased is a great problem as most of the bodies in aircraft injuries are totally mutilated or burnt.[5-8]

INTOXICATION

In all transportation incidents it is essential to exclude *intoxication* with *alcohol*, medicinal compounds such as *antidepressants, hypnotics and antipsychotic drugs,* and the illicit use of drugs e.g. *cannabis, heroin*. No transportation death investigation including aircraft crashes is completed without an examination of the victim's *blood* and *urine* for such compounds. *Drivers* of any vehicles involved in such accidents should also be tested for alcohol and other drugs.[2,3,6]

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Chapter 19

Effects of Injury

The direct and secondary effects of injury are often referred under several synonymous terms—*trauma and fatalities, death from injuries, injury and complications,* etc.

Fatalities or death due to trauma are not uncommon and can arise immediately or swiftly due to the direct and immediate causes thereof or because of delayed and late effects related to the traumatic episode.

Following are the effects/complications of injuries, which can lead to death:[1-4]

- Haemorrhage *(exsanguinations)*—internal and/or external
- Injury to vital organs
- Neurologic shock
- Vagal inhibition
- Infection and septicaemia
- Embolism
- Adult respiratory distress syndrome
- Suprarenal haemorrhage.

HAEMORRHAGE

Haemorrhage could be *external* through the infliction of lacerations or incised wounds, or *internal*, into the body cavities or organs. In extensive haemorrhage, the *rate of loss* is as important as much as the *total volume* lost. The age of the patient and the presence of any pre-existing medical ailments such as *anaemia, cardiovascular* and *respiratory conditions* are also very important points to consider.

External Haemorrhage

External haemorrhage can result in loss of blood with consequent hypovolaemic shock and death. Loss of 2 litres of blood, i.e. about *two-fifths* of the blood volume is usually fatal in an adult. A smaller proportion of blood volume loss can cause death in the *previously ill* and in the *extremes* of ages.

Internal Haemorrhage

Internal haemorrhage can be encountered in:

The Respiratory Tract

The respiratory tract, i.e. into the lumen of the tracheobronchial tree (Fig. 19.1A) resulting in mechanical asphyxia with external additional features thereof, e.g. cut-throat injury, wherein the blood gets *aspirated into the respiratory tract* producing a condition called *drowning in one's own blood* leading to death due to mechanical asphyxia. This can take place in *partially conscious, unconscious* and in *intoxicated individuals*. The *cough* and *gag reflexes* will otherwise protect the conscious individual.

The Pericardial Sac [Haemopericardium]

Accumulation of blood more than 250 ml into the pericardial sac can result in cardiac standstill due to inability of myocardial muscle to function. This is also called *cardiac tamponade. Pericardial tamponade* (Figs 19.1B and C), is a medical emergency in which pericardial pressure is elevated and this puts significant pressure on the heart, causing a decrease in diastolic filling of the ventricles, and hence in stroke volume. The end result is ineffective pumping of blood, shock and often death.[5,6]

Intracranial Haemorrhages

Intracranial haemorrhages is bleeding within the skull or bleeding into the cranial cavity[7] is not always dangerous because of the actual blood loss into this site, but because of the space-occupying effect it can produce by its physical presence and direct pressure on to the brain *(refer Head Injuries);* it will also obstruct, if partially clotted, the foramina through which the CSF flows.[7]

Bleeding into Serous Cavities

Bleeding into serous cavities—litres of blood can accumulate within the pleural (Figs 19.2A and B) and peritoneal cavities (Fig. 19.2C).[8] The source of such haemorrhages should be looked for carefully.

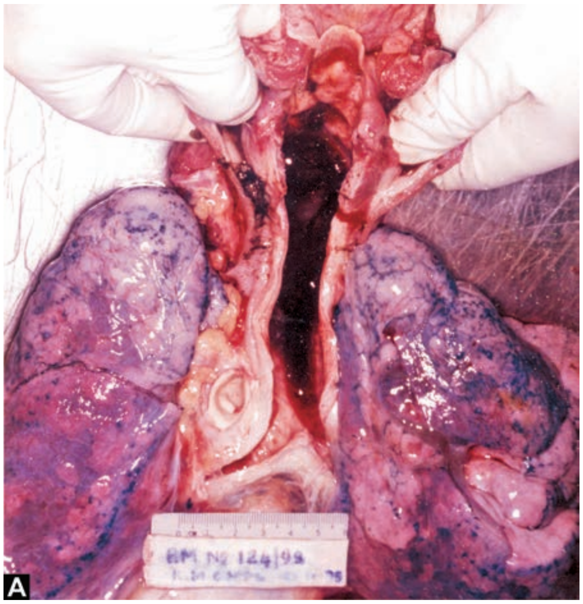


Fig. 19.1A: Haemorrhage into the trachea, resulting in asphyxiation

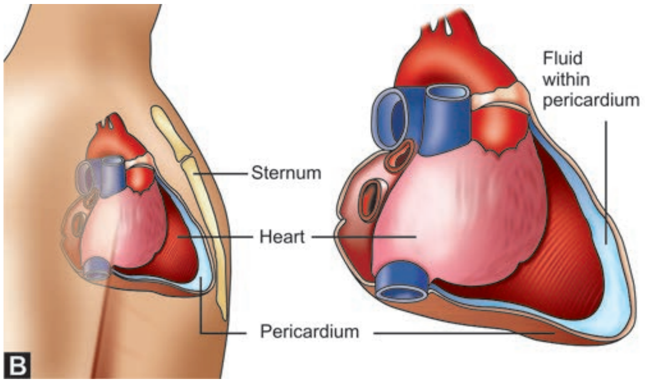

Fluid
within
pericardium
Sternum
Heart
Pericardium
B

Fig. 19.1B: *Cardiac tamponade:* Anatomical consideration—heart and pericardial sac

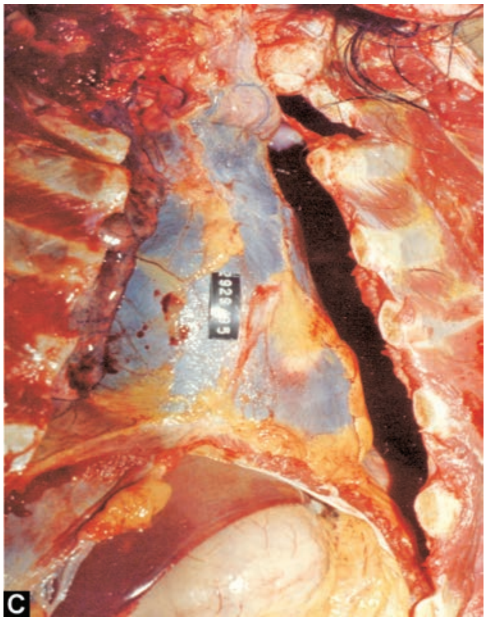


Fig. 19.1C: *Cardiac tamponade:* Removal of sternal plate shows the pericardial sac distended with blood, compressing the heart (*Courtesy:* Dr B Santhakumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

Bleeding into the Gastrointestinal Tract

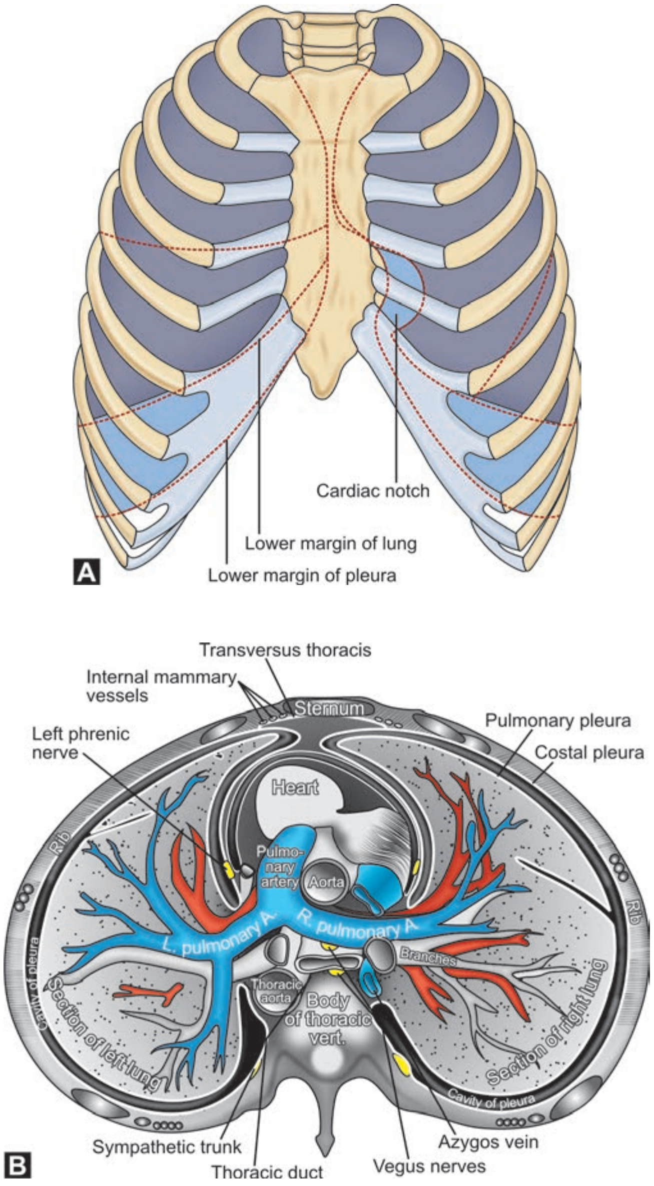
Bleeding into the gastrointestinal tract the accumulation of blood within the stomach and intestines is usually due to pre-existing medical conditions viz: oesophageal varices, peptic ulceration, etc. but on occasions may be the result of traumatic damage—sometimes surgical.

Bleeding into Soft Tissues

Bleeding into soft tissues—on occasion there is no specified accumulation of blood, but extensive bruising and haematoma formation may occur throughout the body. This can cause *hypovolaemia* and *death* on occasions, e.g. with *severe beatings, kicking, flogging,* etc (Fig. 19.3).

Other Practical Points

- It may be also noted that the completely severed artery may bleed less than the one that is partially transected, as the former may retract the muscular coats sealing the lumen partly or completely. If the damage is partial irregular clots are formed more easily.


Cardiac notch
Lower margin of lung
Lower margin of pleura
A
Transversus thoracis
Internal mammary
vessels
Sternum
Pulmonary pleura
Costal pleura
Left phrenic
nerve
Heart
Rib
Pulmo-
nary
artery
Aorta
L. pulmonary A.
R. pulmonary A.
Branches
Rib
Cavity of pleura
Section of left lung
Thoracic
aorta
Body
of thoracic
vert.
Section of right lung
Cavity of pleura
Sympathetic trunk
Thoracic duct
Vegus nerves
Azygos vein
B

Figs 19.2A and B: *Pleural cavity:* Anatomical considerations

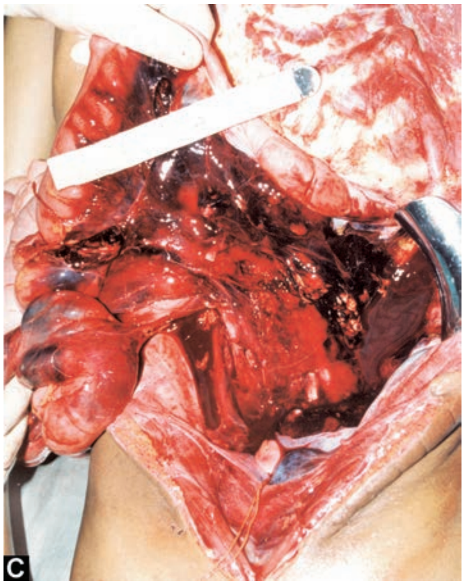


Fig. 19.2C: Intraperitoneal and mesenteric haemorrhage

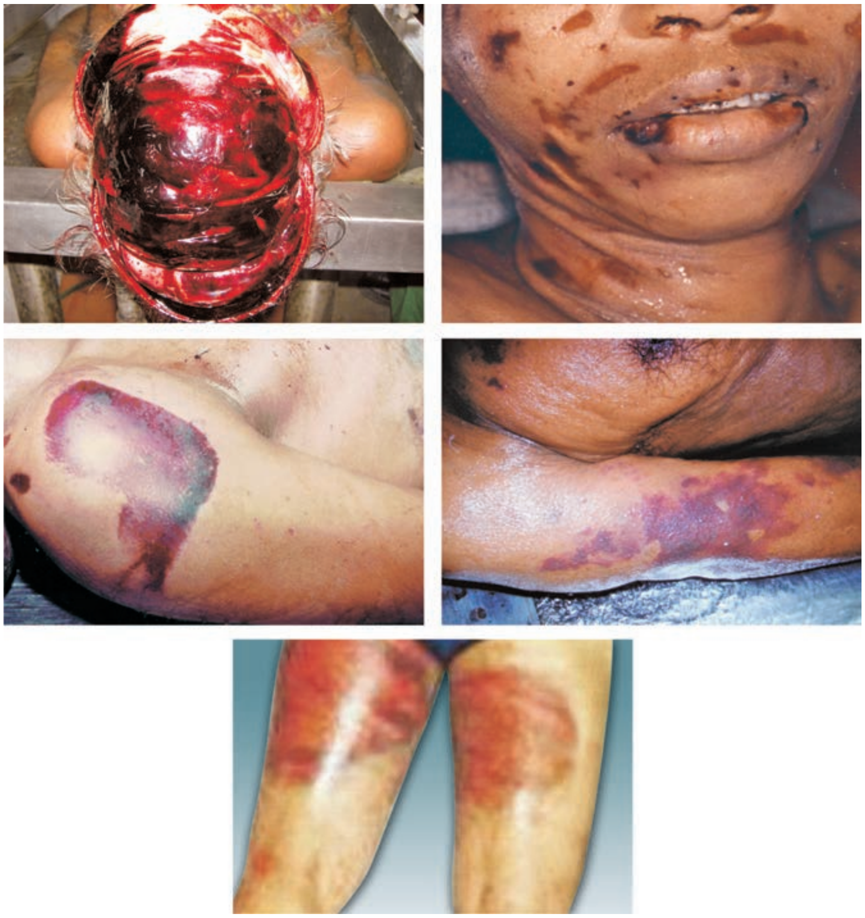


Fig. 19.3: Extensive bruising of subscalp region, face and neck, in the right shoulder and right upper arm, and back of both thighs, etc may lead to fatality

- It is better to exercise caution in giving opinions about the amount of blood found at the scene. Wounds can continue to bleed copiously after death due to passive leakage and postmortem fibrinolysis, especially if the injured part of the body is dependent, i.e. lower than the rest of the body. Scalp wounds are usually prone to bleed passively after death.[9] Blood can also accumulate in large quantities within pleural and peritoneal cavities after death, so it is impossible to assess accurately the volume of blood that was due to true intravital bleeding.
- Projectile spurting of blood is only possible from arteries cut during life. A major vessel such as the common carotid can spray blood for a maximum distance of the order of half metre or more depending on the systolic blood pressure. Venous bleeding such as from the jugulars cannot spray but only well out and ooze over a period in considerable volume, but without projection.
- If the patient is on anticoagulants—and remember that alcohol and aspirin both act pharmacologically as anticoagulants—the bleeding will be out of proportion to the trauma. This is also true of any other congenital or acquired coagulation problem/factor or platelet disease.

INJURY TO VITAL ORGANS

(Refer: Regional Injuries)

VAGAL INHIBITION

Vagal inhibition is due to stimulation of trigger areas *(refer Thanatology)*.

INFECTION AND SEPTICAEMIA[2,3,4,10-12]

Wound infection is a common phenomenon when the wound is not properly treated and debrided at presentation, especially if heavily contaminated and contains within it foreign substances such as soil, grit, bits of clothing, and can result in death. Surgical debridement and the use of antiseptics, sterile dressings and antibiotics have revolutionised the prognosis of open injuries, but infective consequences can still occur, especially in situations where rapid and effective treatment is not available.

Commonly encountered organisms like *staphylococci, streptococci*, coliforms, *Pseudomonas aeruginosa* and *anaerobes* such as *Clostridium perfringens* and *tetani* colonize wounds.

In addition to frank sepsis, in recent years the phenomenon referred to as *"septic shock"* has become apparent. This is due to *toxins* (now known as *'Aggressins')* and bacterial colonisation of the blood. It may be due to gram-positive bacteria where death is due to exotoxins originating in unsuspected foci of infection, such as vaginal tampons harboring *Staphylococci*. Much more commonly it is due to gram-negative bacteria mainly coliforms with a source in the gastrointestinal tract, the urinary tract, the genital tract and the biliary tract, being some of the most important tracts.

Common complications of septic shock are the *adult respiratory distress syndrome (ARDS)* and *DIC (Disseminated Intravascular Coagulopathy)* in which there is consumption of platelets, fibrin and blood clotting factors with myriad of small thrombi arising in small peripheral blood vessels with consequent haemolysis and coagulation problems due to toxin-induced damage to these blood vessels.

EMBOLISM

Embolism is most commonly overlooked as a cause of death by both clinicians and inexperienced medicolegal experts.

Injury and Embolism[1-4,13,14]

Embolism is a mechanism wherein there is an introduction of undissolvable foreign material – gaseous or solid within the living circulation which is then distributed in the blood stream, and may cause decrease in blood supply (*ischaemia*) or impaction in end-arteries or veins with consequent ischaemic *necrosis (infarction)* of the tissues involved.

Embolism due to injuries of medicolegal importance is of four main types:

- *Fat embolism*
- *Air embolism*
- *Amniotic fluid embolism*
- *Thrombotic embolism or thromboembolism.*

Fat Embolism

Its ***source*** could be of intrinsic/extrinsic origin.

Intrinsic or body fat — the actual origin of the fat is either from adipose tissue or from bone marrow, and only occasionally from the lipids in the plasma. Fat embolism occurs in almost every case-involving trauma to the bone or subcutaneous tissue, especially where there is a crushing effect on areas rich in adipose tissue, such as buttocks or breasts. Fracture of the pelvis and long bones can lead to release of some bone marrow fat into the venous circulation as well as globules of bone marrow, but the lung capillaries will filter this off. If lung histology, specially stained for fat (*Sudan dyes, Osmium Tetroxide*), is examined after almost any tissue trauma, a few fat globules will be found (Fig. 19.4). However, when the fat load in the lungs reaches a certain threshold, globules penetrate the anatomic vessels and appear in the *systemic circulation*, and then embolize to vital organs and tissues like brain, kidneys, skin and myocardium. *Microinfarcts* are caused in many organs; and an infarct in *brainstem* can be *fatal*. If there has been active *cardiorespiratory resuscitation [CPR]* associated with fracturing of the sternum and ribs, this finding may have an *iatrogenic causation*. In case of *multiple injuries*, early *fulminant post-traumatic fat embolism syndrome* masked by haemorrhagic shock is reported in the literature recently, highlights the risk of masked fat embolism shortly after trauma.[13]

Extrinsic or Injected fat, e.g. injecting oily substances in radiography and in total parenteral nutrition.

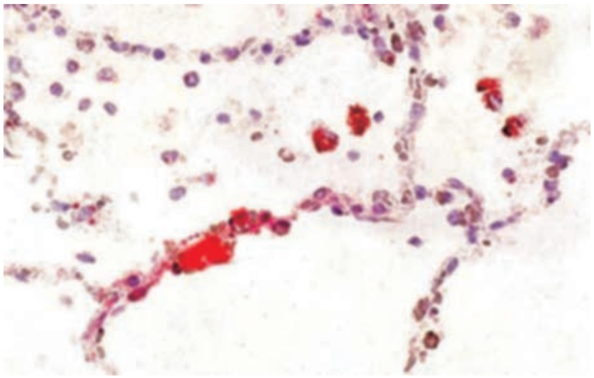


Fig. 19.4: *Fat embolism lung histopathology changes:* An oil red O stain for neutral fat shows a homogeneous red globule of fat in the capillary wall. Several alveolar macrophages are also seen which are granular, browner lipochrome pigments, may be mistaken for fat globule

Air Embolism (Gas Embolism)

Source Extrinsic only (200-250 ml of air in circulation is lethal in an adult; in the extremes of ages. In the sick individuals it is much less).

Varieties – Types of air embolism:

- Pulmonary embolism
- Systemic embolism.

Pulmonary Air Embolism (via Right Side of Heart)

Causes

- I/V administration – lack of taking precautions such as releasing the air in the tubing used for intravenous administration prior connecting the drip to the vein. This is much more dangerous with *Hickmann's lines* and other forms of prolonged *catheterisation* of blood vessels.
- *Cut-throat injuries* or *surgery* to the *neck* [thyroidectomy, parathyroidectomy]–air can be sucked into the jugular veins.
- Superior sagittal sinus and other cerebral venous sinuses– due to *injury* or *brain surgery.*
- Induction of *criminal abortion* by pumping *air* into the uterus.
- *Rubin's fallopian tube test* for its patency [rarely performed nowadays].
- *Artificial pneumothorax* in chest disorders or *pneumo-peritoneum* induction in *'keyhole' surgery/ laporoscopy*.

Systemic Air Embolism

Causes–as with penetrating injury or other wounds of chest.

Gas Embolism and SCUBA Diving

Gas embolism is one of the diving disorders SCUBA divers sometimes suffer when they receive pressure damage to their lungs following a rapid ascent where the breath is inappropriately held against a closed glottis, allowing pressure to build up inside the lungs, relative to the blood. It is termed "gas" because the diver may be using a diving breathing gas other than air. The gas bubbles can impede the flow of oxygen-rich blood to the brain and vital organs. They can also cause clots to form in blood vessels.

Gas embolism and decompression sickness (DCS) may be difficult to distinguish, as they may have similar symptoms, especially in the central nervous system. The treatment for both is the same, because they are both the result of gas bubbles in the body. In a diving context, the two are often called decompression illness (DCI).[14]

Amniotic Fluid Embolism

Amniotic fluid embolism is a complication of childbirth; in complicated labor, there may be an escape of amniotic fluid into maternal circulation. It is not known whether this can occur on a small scale without causing symptoms, but it can certainly prove fatal by an allergic response, by producing disseminated intravascular coagulopathy and by diffuse blocking of the pulmonary capillaries. On autopsy apart from the fluid, fetal debris such as fetal lanugo, squamous cells, mucin, vernix lipid and even meconium can be found (Fig. 19.5). Histologically Aguilera et al reported foetal epithelial squamous cells obstructing 80 per cent of pulmonary capillaries and fetal epithelial squamous cells in alveoli on autopsy.[15] A blood sample from a central venous catheter also showed foetal squames. Marcus et al found focal interstitial haemorrhages in the kidneys, the left ventricle, and the interventricular septum.[16,17] Alcian blue periodic acid-Schiff (PAS) stain was positive for mucin in the vasculature and *oil red O stain* for lipid was positive in the lungs. Special staining methods may have to be opted on multiple sections of lung for a positive diagnosis.

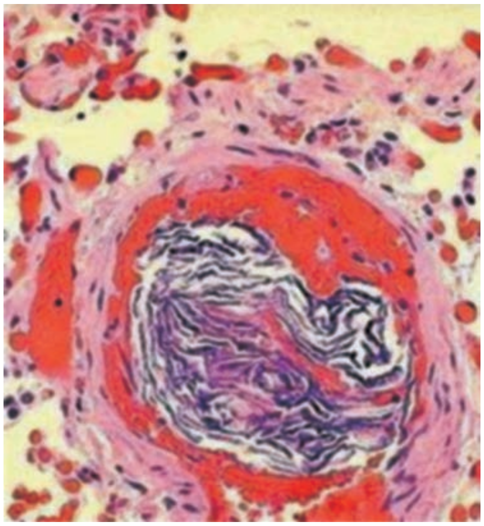


Fig. 19.5: Lung histopathology showing foetal squamous cells in the blood vessels suggestive of amniotic fluid embolism

Thrombotic Embolism

Following all forms of trauma including surgery, the body reacts by increasing the number of platelets in the bloodstream and rendering these more sticky and ready to aggregate together. There is also embolisation after trauma due to the lethargy, malaise and physical immobility that results due to pain, etc. and the use of a general anesthetic and drugs used for pain relief and sedation. There may also be dehydration or a diminished blood volume, which render the blood more viscous. This sets the scene for thrombosis particularly in those blood vessels mainly veins, which have been injured at the site of trauma or surgery, e.g. pelvic veins after surgery to the hip, rectum or perineum, postpartum, or elsewhere e.g. calf veins in an immobile patient (*Deep Vein Thrombosis—DVT*). These thrombi often produce embolism, which by blockage of the pulmonary arteries is often fatal; indeed, it is still one of the most important post traumatic and postoperative causes of death.

ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)[2,4,19-22]

The incidence of ARDS has been difficult to determine but various published estimates have ranged from 1.5 to 75 cases per 100,000 populations.[19] Adult respiratory distress syndrome (ARDS) is a complication of a whole range of traumatic or stressful incidents, including aspiration of gastric contents, near-drowning, blast injuries to the chest, heavy impacts on thorax, inhalation of irritant gases, infections, acute pancreatitis, burns and systemic shock.[2,4]

The lungs become oedematous and stiff due to inter-alveolar wall oedema and later intra-alveolar oedema leading to marked respiratory failure due to poor gas exchange. The chest radiograph reveals characteristic diffuse alveolar-interstitial infiltrates in all lung fields (Fig. 19.6). Clinically there is dyspnoea orthopnoea, hypoxaemia, CO_2 retention and progressive respiratory insufficiency. There is difficulty in ventilating the lungs artificially and more and more pressure is required due to the decrease of the *intrinsic elasticity* or *compliance* of the pulmonary parenchyma

At autopsy, stiff lungs are noted on naked eye inspection, which have a very fleshy liver–like appearance and retain their shape, and histologically, intra–alveolar exudation of fibrin rich fluid, hyaline membranes and haemorrhage are seen initially, and these later give way to cellular proliferation, which becomes fibrotic if survival is long enough.

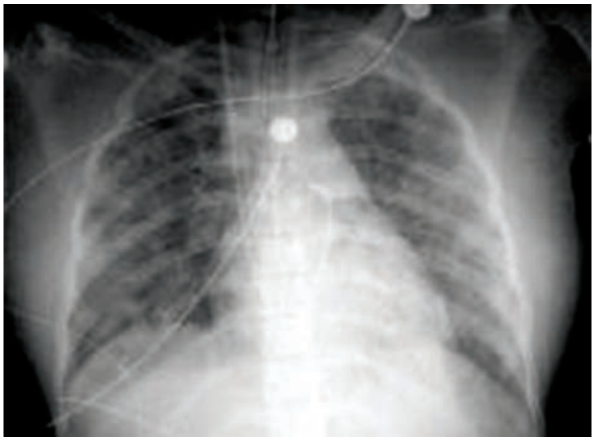


Fig. 19.6: Chest radiograph showing adult respiratory distress syndrome (ARDS)

A similar condition occurs with certain poisons such as paraquat (weed killer) and drugs e.g. busulphan used in oncology.

SUPRARENAL HAEMORRHAGE[1,2,4]

An uncommon complication of injury is haemorrhage into substance of the adrenal glands, sometimes bilaterally. It tends to occur a few days after severe trauma, preceded by cortical depletion of yellow lipoid, which is a common sequel to stress. The bleeding sometimes massive enough to expand the glands to several times their normal size occurs in the medulla, the cortex being stretched around the haematoma. The bleeding is not due to direct mechanical damage, but appears to be systemically mediated due to direct damage by toxins of the medullary blood vessel. It is seen *par excellence* in *meningococcal* septicaemia (Waterhouse *Frederickson syndrome*) and in gram-negative shock.

ANTEMORTEM AND POSTMORTEM INJURIES

Differentiating the antemortem wound from postmortem wound is one of the major problems. It is so important in forensic medicine that it can many a time help in deciding the appropriate outcome of a criminal case, either proving a suspected person guilty or not. Table 19.1 enumerates the differences between antemortem and postmortem injuries.

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Table 19.1: Differences between antemortem and postmortem wounds[1-4,6]

Criteria	*Antemortem wound*	*Postmortem wound*
Naked eye examination		
1. Haemorrhages		
• Quantity	Copious	None/very slightly
• Origin	Arterial	Capillary/venous
• Tissue staining	Deeper	Not so
• Washing the stain	Cannot be washed	Can be washed
• Spurting marks	+ve	–ve
2. General appearances	Wet, red and have the typical appearances of incised or lacerated wounds	Dry, parchment- like, yellow and *do not* have the typical appearances
3. Blood clot in their bases		
• Clotting	+ve	–ve/+ve
• Colour	Variegated	Yellow**/Red***
• Consistency	Firm and rubbery	Soft and loose
• Friability	–ve	+ve
• Adherence to endothelium	Firm and rubbery	Weakly
4. Wound edges		
• Gaping	+ve	–ve (may be +ve) ~
• Eversion	+ve	–ve
• Swelling	+ve	–ve
• Bruising	+ve	–ve
5. Vital reaction ~~		
• Signs of inflammation and repair	+ve	–ve
• Secondary infection with pus and slough	+ve	–ve
• Granulation tissue	+ve	–ve
6. Microscopy		
Pavementation and infiltration of RBC and WBCs between muscle fibres	+ve	–ve
7. Enzyme histochemistry tests		
A. Enzymes reported to be present are:		
• Adenosine triphosphatase	+ve	Nil
• Acid phosphatase	+ve	Nil
• Alkaline phosphatase	+ve	Nil
B. Wound zone reporting of:		
• –ve vital reaction	Wound edge, central/ superficial zone	Nil Nil
• +ve vital reaction	Peripheral zone	Nil
8. Wound biochemistry		
• Presence of serotonin [5HT]	May be positive	Nil

~~If death has been delayed sufficiently after infliction, * Postmortem clots, ** Chicken fat, *** Black currant's jelly, ~ If injury occurs within 2 hours of death

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Chapter 20

Firearms and Explosive Injuries

INTRODUCTION

Firearms are barreled weapons of any type or description from which a shot, bullet or other missile can be discharged with some velocity and momentum, and which in the appropriate circumstances can cause injury and death. This chapter mainly deals with basics on firearms and ammunitions, their identification and injuries produced by them under three headings, i.e. historical aspect, ammunitions and firearm wounds.

HISTORICAL ASPECT

Man has been fascinated with the idea of launching a projectile at animals, developed more efficient ways of doing so. The invention of gunpowder led to the development of firearms.[1-3]

Gunpowder first appeared in use in China over a thousand years ago, but was used primarily in firecrackers and only sparingly in weapons for military use. Dissemination of the knowledge of gunpowder manufacture to Europe in the 14th century did not at first lead to military usage. However, once the effectiveness of projectiles impelled by the force of gunpowder against both the armor of knight-soldiers and fortifications was known, the use of firearms proliferated rapidly.[2] The greatest stimulus for firearms development was and continues to be military usage. The important needs, militarily speaking, for a firearm included the following: reliability of firing, accuracy of projectile, force of projectile, speed of firing. The reliability issue sparked the development of a number of mechanisms to ignite the powder. Primitive matchlock weapons employed a burning wick on a spring that was "locked" back and released into a pan of powder upon pulling a trigger. The powder in the pan then ignited, sending flame through a small hole into the barrel chamber of the weapon, igniting a larger powder charge in the chamber and sending the projectile (bullet) forward.[3-5] More details on historical aspects of different firearms and ammunitions are given in boxes below:

IMPORTANT TERMS

Some of the common terminologies under firearms and ammunitions are listed below and discussed in brief.[3-20] They are also accessible in detail under relevant areas in this chapter.

Bullets: The projectile. They are shaped or composed differently for a variety of purposes and enumerated below (Fig. 20.1):

- "*Round-nose*" — The end of the bullet is blunted.
- "*Hollow-point*" — There is a hole in the bullet that creates expansion when a target is struck, creating more damage.
- "*Jacketed*" — The soft lead is surrounded by another metal, usually copper, that allows the bullet to penetrate a target more easily.
- "*Wadcutter*" — The front of the bullet is flattened.
- "*Semi-wadcutter*" — Intermediate between round-nose and wadcutter, has the features of both semi-wadcutter and hollowpoint.

Butt or buttstock: The portion of the gun which is held or shouldered.

Calibre: The diameter of the bore of a gun/shotgun.

Cartridge: Also called a "round". Made up of a case, primer, powder, and bullet.

Centrefire: The cartridge contains the primer in the centre of the base, where it can be struck by the firing pin of the action.

Chamber: The portion of the "action" that holds the cartridge ready for firing.

Choke: A constriction of a shotgun bore at the muzzle that determines the pattern of the fired shot.

Double-action: Pulling the trigger both cocks the hammer and fires the gun.

Double barrel: Two barrels side by side or one on top of the other, usually on a shotgun.

Gauge: Refers to the diameter of the barrel of a shotgun really refers to calibre, but is worded as such to refer to a shotgun.

Hammer: A metal rod or plate that strikes the cartridge primer to detonate the powder.

Ignition: The way in which powder is ignited. Old muzzle-loading weapons used flintlock or percussion caps. Modern guns use "primers" that are "rimfire" or "centrefire".

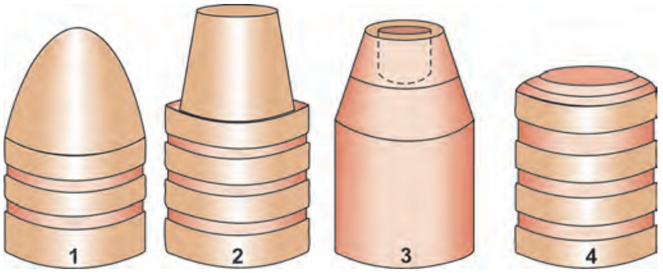


Fig. 20.1: Types of bullets presenting variety of modification: (1) Round nose, (2) Semi-wadcutter, (3) Jacketed semi-wedcutter (Hollow prints), (4) Wadcutter

Historical Aspects of Muskets, Revolvers, Guns, Rifles and Ammunitions[1-5]

Since the introduction of the **flintlock musket** in the 17th century, military small arms have gone through a series of significant changes.

Muskets

Puckle Gun – 1718: In 1718, **James Puckle** of London, England, demonstrated his new invention, the "**Puckle Gun**," a tripod-mounted, single-barreled flintlock gun fitted with a multishot revolving cylinder. This weapon fired nine shots per minute at a time when the standard soldier's musket could be loaded and fired but three times per minute. Puckle demonstrated two versions of the basic design. One weapon, intended for use against Christian enemies, fired conventional round bullets, while the second variant, designed to be used against the Muslim Turks, fired square bullets, which were believed to cause more severe and painful wounds than spherical projectiles. The "*Puckle Gun*" failed to attract investors and never achieved mass production or sales to the British armed forces. One newspaper of the period observed following the business venture's failure that "those are only wounded who hold shares therein." According to the Patent Office of the United Kingdom, "In the reign of Queen Anne, the law officers of the Crown established as a condition of patent that the inventor must in writing describe the invention and the manner in which it works." James Puckle's 1718 patent for a gun was one of the **first** to provide a description.

Revolvers

History of the Colt Revolver: **Samuel Colt** invented the first revolver - named after its revolving cylinder. He was issued a U.S. patent in 1836 for the Colt firearm equipped with a revolving cylinder containing five or six bullets with an innovative cocking device. See also - Samuel Colt and the Revolver

Rifles

- **The breech-loading rifle** was invented by Captain Patrick Ferguson of Pitfours, Scotland.
- **John Moses Browning** - Winchester Rifle: *John Moses Browning* was the prolific gun designer who invented the Winchester rifle (30/30), the pump shotgun, and the Colt 45 automatic. He is best known for his automatic pistols and was the first one to invent the slide, which encloses the barrel of a pistol and the firing mechanism.
- **Modern Assault Rifles - M16 History:** The history of the modern assault rifle begins with the German Sturmgewehr used during WW2, the first rifle that could fire a medium size bullet at high rates of fire. In response the U.S. military began developing their own assault rifle; the result was the M16 assault rifle, first issued to American Soldiers in Vietnam in 1968 and designed by Eugene Stoner, a Marine Corps Veteran.
- **John Garand - M1 Semiautomatic Rifle:** Canadian, John Garand invented the M1 semiautomatic rifle in 1934.
- **The Johnson Rifle:** The Johnson Model 1941 Rifle one of the most innovative rifles of its time period. The Johson rifle was invented by Melvin M. Johnson Jr.
- **Samuel Gardiner:** Samuel Gardiner, Jr. received a U.S. Patent in 1863 on a "high explosive rifle bullet" in .54, .58, and .69 calibers. Fused to explode 1 1/4 seconds after firing, it ensured that any soldier hit by the projectile with a range of 400 yards faced the danger of the bullet exploding within the impact wound. The U.S. Government purchased 110,000 rounds of such ammunition for issue during the Civil War. Criticising the use of similar ammunition by the Confederates, General Ulysses S. Grant complained that "their use is barbarous because they produce increased suffering without any increased advantage to using them."
- **Rifle Scopes:** A rifle scope is a refractor telescope used on a rifle. In 1880, August Fiedler (Stronsdorf), forestry commissioner of Prince Reuss, managed to build the first telescopic sight that really did work.
- **Silencers:** Hiram Maxim (born 1853) invented the Maxim Silencer or Suppressor that attached to the front of the barrel of a pistol and allowed the firearm to be fired without a loud bang. Invented in 1909, the Maxim Suppressor was the first commercially successful silencer.

Lands and grooves: Lands are the metal inside the barrel left after the spiral grooves are cut to produce the rifling.

Magazine: This is a device for storing cartridges in a repeating firearm for loading into the chamber. Also referred to as a "clip".

Magnum: An improved version of a standard cartridge which uses the same caliber and bullet, but has more powder, giving the fired bullet more energy. Magnum shotgun loads, however, refer to an increased amount of shot pellets in the shell.

Muzzle: The end of the barrel out of which the bullet comes.

Pistol: Synonym for a handgun that does not have a revolving cylinder.

Powder: Modern gun cartridges use "smokeless" powder that is relatively stable, of uniform quality, and leaves little residue when ignited. For centuries, "black powder" was used and was quite volatile (ignited at low temperature or shock), was composed of irregularly sized grains, and left a heavy residue after ignition, requiring frequent cleaning of bore.

Primer: A volatile substance that ignites when struck to detonate the powder in a cartridge. "Rimfire" cartridges have primer inside the base, while "centrefire" cartridges have primer in a hole in the middle of the base of the cartridge case.

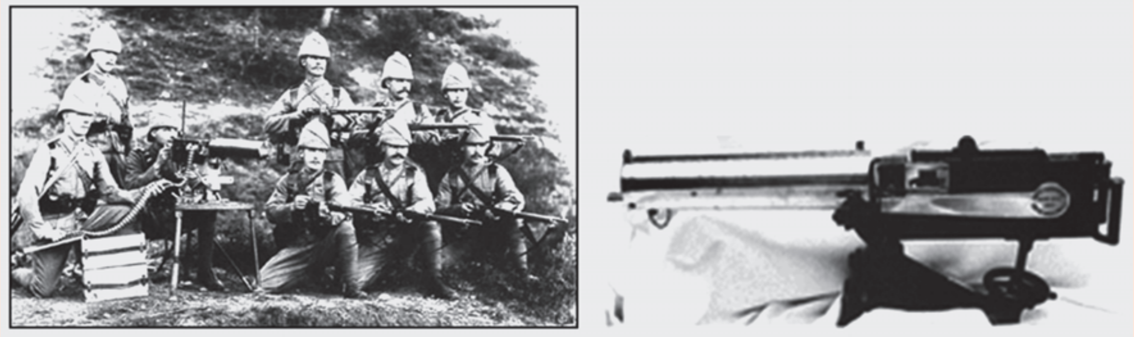
Revolver: Handgun that has a cylinder with holes to contain the cartridges. The cylinder revolves to bring the cartridge into position to be fired. This is "single-action" when the hammer must be cocked before the trigger can fire the weapon. It is "double-action" when pulling the trigger both cocks and fires the gun.

Rifling: The spiral grooves cut inside a gun barrel that gives the bullet a spinning motion. The metal / elevations between the grooves is called a "land".

Historical Aspects of Machine Guns[1-5]

Machine guns

- **Gatling Gun – 1861:** Doctor Richard Gatling patented his design of the “Gatling Gun”, a six-barreled weapon capable of firing a (then) phenomenal 200 rounds per minute.
- **Thompson Submachine Gun - Tommy Gun:** The Thompson submachine gun or Tommy gun was invented by **General John T** and it was the first hand held machine gun. It was driven with the thought of creating a hand held machinegun that would help end the **First World War**. However, “the first shipment of prototype guns destined for Europe arrived at the docks in New York city on November 11, 1918, the day the War ended.”
- The idea of a gun that would keep up a continuous stream of fire attracted inventors early in the development of firearms. In 1718 **James Puckle** invented what he called his **Defence Gun**. Placed on a tripod it was a large revolver with a cylinder behind its single barrel. Although the cylinder had to be turned manually it could fire 63 shots in seven minutes.
- The **American Civil War** provided an incentive to inventors and **Wilson Agar** was able to sell 54 of his **Coffee Mill guns** to the Union Army. The **Billinghurst-Requa** was also used by Union forces in the war. The gun comprised a wheeled frame carrying 24 rifle barrels. Once the gun was loaded a single percussion cap was placed on a nipple on the iron frame and fired by a hammer, the flash passing through the frame to ignite all 24 cartridges.
- In 1861 **Richard Jordan Gatling**, a trained ***dentist*** from North Carolina produced an effective mechanical gun. **The Gatling Gun** consisted of six barrels mounted in a revolving frame. The United States Army purchased these guns in 1865 and over the next few years most major armies in Europe purchased the gun. The British Army tested it at Woolwich in 1870, and found that the 0.42 Gatling Gun fired 616 shots in two minutes. Of these, 369 hit their intended targets.
- In 1879 the **Gardner Machine Gun** was demonstrated for the first time. The gun fired 10,000 rounds in 27 minutes. This impressed military leaders from Britain and the following year the British Army purchased the gun. It also adopted the ten-barrel Nordenfelt Machine Gun.
- Maxim Machine gun – 1885: In 1881 the American inventor, **Hiram Maxim**, visited the Paris Electrical Exhibition. While he was at the exhibition he met a man who told him: “If you want to make a lot of money, invent something that will enable these Europeans to cut each other’s throats with greater facility.” Maxim moved to London and over the next few years worked on producing an effective machine-gun. In 1885 he demonstrated the world’s first **automatic portable machine-gun** to the British Army. Maxim used the energy of each bullet’s recoil force to eject the spent cartridge and insert the next bullet. The **Maxim Machine-Gun** would therefore fire until the entire belt of bullets was used up. Trials showed that the machine-gun could fire 500 rounds per minute and therefore had the firepower of about 100 rifles. The Maxim Machine-Gun was adopted by the British Army in 1889. The following year the Austrian, German, Italian, Swiss and Russian armies also purchased Maxim’s gun. The gun was first used by Britain`s colonial forces in the Matabele war in 1893-94. In one engagement, fifty soldiers fought off 5,000 Matabele warriors with just four Maxim guns (Fig. 20.2).
- The success of the Maxim Machine-Gun inspired other inventors. The German Army’s **Maschinengewehr** and the Russian **Pulemyot Maxima** were both based on Maxim’s invention. **John Moses Browning** produced his first machine-gun in 1890 and five years it was adopted by the US Navy. An Austrian, **Count Odkolek**, worked with the French company, **Hotchkiss,** to produce an effective gun that was adopted by the French Army in 1897.
- By the outbreak of the **First World War**, the British Army had adopted the **Vickers Machine-Gun**. Fitted with interrupter gear, the Vickers was also standard armament on all British and French aircraft after 1916. During the war the British also used the **Lewis Gun**. Easier to produce and far lighter than the Vickers, it was used by soldiers on the Western Front and on armoured cars and aircraft.



Figs 20.2A and B: (A) The King’s royal rifles in 1895 with a maxim machine gun and Lee-Enfield rifles. (B) Maxim machine gun – magnified view[1]

Rimfire: The cartridge has the primer distributed around the periphery of the base.

Safety: A mechanism on an action to prevent firing of the gun.

Shotgun: A gun with a smooth bore that shoots cartridges that contain "shot" or small metal pellets (of lead or steel) as the projectiles.

Sights: The device(s) on top of a barrel that allow the gun to be aimed.

Silencer: A device that fits over the muzzle of the barrel to muffle the sound of a gunshot. Most work by baffling the escape of gases.

Single-action: The hammer must be manually cocked before trigger can be pulled to fire the gun.

Smokeless powder: Refers to modern gunpowder, which is really not "powder" but flakes of nitrocellulose and other substances. Not really "smokeless" but much less so than black powder.

Stock: A wood, metal, or plastic frame that holds the barrel and action and allows the gun to be held firmly.

FIREARMS AND AMMUNITIONS—BALLISTICS

Ballistics is the science of the motion of projectiles.[4-6] The term ballistics refers to the science of the travel of a projectile in flight (Ballistics gr. ba'llein, meaning "throw"). The flight path of a bullet includes: travel down the barrel, path through the air, and path through a target. The wounding potential of projectiles is a complex matter.[7] Ballistics is often discussed under five subheads[5,6] and they are:

- Internal/Interior/Initial ballistics.
- External ballistics.
- Elementary ballistics.
- Terminal ballistics.
- Wound ballistics.

INTERNAL/INTERIOR/INITIAL BALLISTICS (Within the Gun)[5]

This comprise of the study of projectiles/bullets used for firing from firearm weapons. Bullets fired from a rifle will have more energy than similar bullets fired from a handgun. More powder can also be used in rifle cartridges because the bullet chambers can be designed to withstand greater pressures (70,000 psi vs. 40,000 psi for handgun chamber). It is difficult in practice to measure the forces within a gun barrel, but the one easily measured parameter is the velocity with which the bullet exits the barrel (muzzle velocity) and this is what will be used in examples below.

The controlled expansion of burning gunpowder generates pressure (force/area). The area here is the base of the bullet (equivalent to diameter of barrel) and is a constant. Therefore, the energy transmitted to the bullet (with a given mass) will depend upon mass times force times the time interval over which the force is applied. The last of these factors is a function of barrel length. Bullet traveling through a gun barrel is characterised by increasing acceleration as the expanding gases push on it. Up to a point, the longer the barrel, the greater is the acceleration.

EXTERNAL BALLISTICS (FROM GUN TO TARGET)[5]

This comprise of study of projectiles in the air. The external ballistics of a bullet's path can be determined by several formulae, the simplest of which is:

$$\text{Kinetic Energy (KE)} = 1/2\ MV^2$$

Velocity (V) is usually given in feet/second (fps) and mass (M) is given in pounds, derived from the weight (W) of the bullet in grains, divided by 7000 grains per pound times the acceleration of gravity (32 ft/sec) so that:

$$\text{Kinetic Energy (KE)} = W(V)^2/(450{,}435)\ \text{ft/lb}$$

This is the bullet's energy as it leaves the muzzle, but the ballistic coefficient (BC) will determine the amount of KE delivered to the target as air resistance is encountered.

$$\text{Ballistic coefficient (BC)} = SD/I$$

SD is the sectional density of the bullet, and 'I' is a form factor for the bullet shape. Sectional density is calculated from the bullet mass (M) divided by the square of its diameter. The form factor value 'I' decreases with increasing pointedness of the bullet (a sphere would have the highest 'I' value).

Forward motion of the bullet is also affected by drag (D), which is calculated as:

$$\text{Drag (D)} = f\ (v/a)\ k \text{ and } \rho d\ 2v^2$$

'f (v/a)' is a coefficient related to the ratio of the velocity of the bullet to the velocity of sound in the medium through which it travels. The 'k' is a constant for the shape of the bullet and is a constant for yaw (deviation from linear flight). The 'ρ' is the density of the medium (tissue density is >800 times that of air); 'd' is the diameter (calibre) of the bullet, and 'v' the velocity. Thus, greater velocity, greater caliber, or denser tissue gives more drag. The degree to which a bullet is slowed by drag is called retardation (r) given by the formula:

$$r = D/M$$

Since drag (D) is a function of velocity, it can be seen that for a bullet of a given mass (M), the greater the velocity, the greater the retardation. Drag is also influenced by bullet spin. The faster the spin, the less likely a bullet will "yaw" or turn sideways and tumble. Thus, increasing the twist of the rifling from 1 in 7 will impart greater spin than the typical 1 in 12 spiral (one turn in 12 inches of barrel).

ELEMENTARY BALLISTICS

This comprise of study of the anatomy of the firearm and ammunitions. Elementary Ballistics includes basic study on firearm.[3-5] Accordingly a firearm is the weapon used for firing or shooting of a projectile, and ammunition refers to the projectile that is being fired, i.e. the cartridge and its ingredients, i.e. bullets, pellets, missiles, etc. In the field of forensic science, forensic ballistics is the science of analysing firearm usage in crimes. It involves analysis of bullets and bullet impacts to determine the type 2.

Firearms and ammunition can be studied under the two heads, namely

- General considerations
- Types of firearms.

GENERAL CONSIDERATIONS

These comprise of knowing about the basic construction and structure of firearms and that of the chemical constituents of firearm ammunition.[5-13] Each one is discussed below separately.

Structure of the Firearms in General

Basically a firearm is made up of a hollow metallic tube known as barrel of some length (variable). One end of this is closed and at this end the ammunition (cartridge with bullet, pellet, etc.) is loaded for shooting out of the firearm, this end being modified accordingly for the type of weapon and it is called the chamber or breech end. This end also has a special device known as trigger, by the operation of which there is detonation of a charge at the lower end of the cartridge, with the ammunition being thus forcefully fired and ejected out through the other end of the barrel, which is free and open end and is known as the muzzle end (Figs 20.3A and B).

Chemical Constituents in Firearm Ammunitions

The important chemical ingredients used in firearm ammunitions are namely-igniting powder and gunpowder.

Igniting mixture: Also known as detonator and often contains two chemical ingredients, namely—mercuric fulminate and lead oxide. These chemicals undergo a thermogenic reaction on being percussed or hit by the ignition hammer.

Gunpowder: Usually two types of gunpowders are used, smokeless powder and black gunpowder. The former contains the chemical nitrocellulose. On ignition it burns without producing any smoke. The latter contains three chemicals,

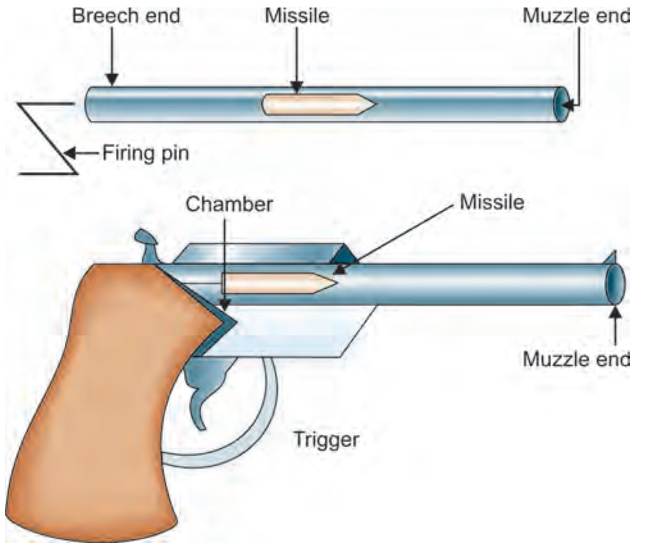


Fig. 20.3A: Rifled firearm structure in general

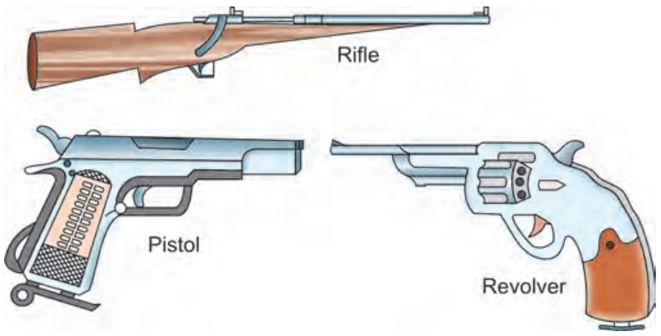


Fig. 20.3B: Diagram of rifle, pistol and revolver

namely charcoal (15%), potassium nitrate (75%) and sulfur (10%). On ignition, this is incapable of complete combustion and thus produces smoke.

TYPES OF FIREARMS

Firearms are basically of two types:

1. With rifled barrels – Rifled Firearms (Guns)
2. With smooth barrels – Smooth bored firearms (Shot guns).

RIFLED FIREARMS (GUNS)

These are weapons with elevations on the inside of the barrel that spirals round from one end of the barrel to the other, which can fire missiles or bullets (Fig. 20.4E) capable of spinning on its own axis thus rendering their aim more accurate and their muzzle velocity higher.[4,8,9,12,14-19] Learning about the following helps in better understanding of guns and its mechanisms:

- Rifling of a gun
- Rifling marks
- Calibre of a gun/rifled firearm
- Classification of guns/rifled firearms
- Cartridge of a gun/rifled firearm.

Rifling of a Gun

The barrel in a gun of any type is cut into spiral grooves, longitudinally from the chamber to the muzzle end on the inner aspect of its wall. These spiral grooves are called rifling of a gun. Thus, in a cross-section and longitudinal section (Figs 20.4A to D) of a gun barrel, this is seen as elevated areas called lands and depressed areas called grooves.

Purpose of Rifling

Rifling of a gun helps the bullet to achieve a gyroscopic movement, which in turn gives a steadiness, during its flight. It gives the bullet greater power of penetration, enabling it to cover longer ranges, improves accuracy of hitting the target aimed at and also prevents wobbling of bullet due to wind/air resistance.

Pattern of Rifling

Rifling in guns is not uniform. It varies with such factors as direction of the grooving, which could be clockwise or anticlockwise (Fig. 20.4B), magnitude of the rifling spin (i.e. the distance covered for one complete turn of the spin within the barrel) and width and depth of rifling groove. These factors make the rifling marks on the outer casing of the cartridge of the bullet acquire unique characteristics to the gun just like fingerprints are to a hand. In its rotational passage at speed through the barrel, heat is generated and this renders the metal on the outside of the bullet softer and thus more easily marked on forceful pressures against the lands of the barrel.

Rifling Marks

Rifling marks are important markings developed on the surface of the bullet (Fig. 20.4D) during its transport through the barrel when fired due to the friction or rubbing against the lands and grooves. A detailed microscopic study of rifling marks using a comparison microscope comparing a bullet retrieved from a scene or body with another bullet from another scene or body and one test fired from the suspect gun, has proved beyond doubt that they are unique and typical for a particular gun, because no two guns (even of same make) can induce rifling marks, which are absolutely identical. Thus, rifling marks are considered as fingerprint marks of a gun or a rifle.

Medicolegal importance — Rifling marks can help in identification of the weapon used in a crime. It is essential that all spent bullets be handled with great care to avoid adding other marks to them. They should be handled with gloved hands and with plastic (NOT metallic) forceps.

Calibre of a Gun

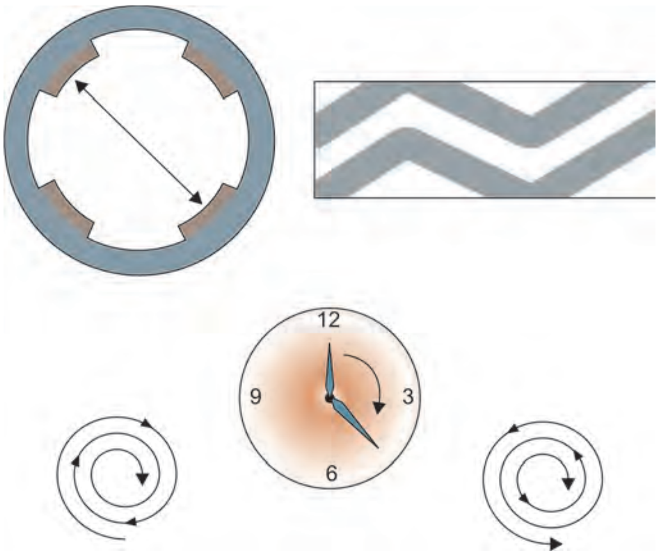
Calibre of a gun is the diameter of the barrel or bore. It is measured as the distance between two lands (Fig. 20.4A) of the rifling. Thus, a 0.303 rifle means a rifle, which has the caliber of 0.303 of an inch (it is an Indian military rifle). Calibre of gun or rifle is usually given in inches (as 0.22, 0.25, 0.303, 0.32, 0.38, 0.45, etc inch) or in millimetres (as 6.35, 7.62, 7.65, 9.0, etc millimeters).

Classification of Guns/Rifled Firearms

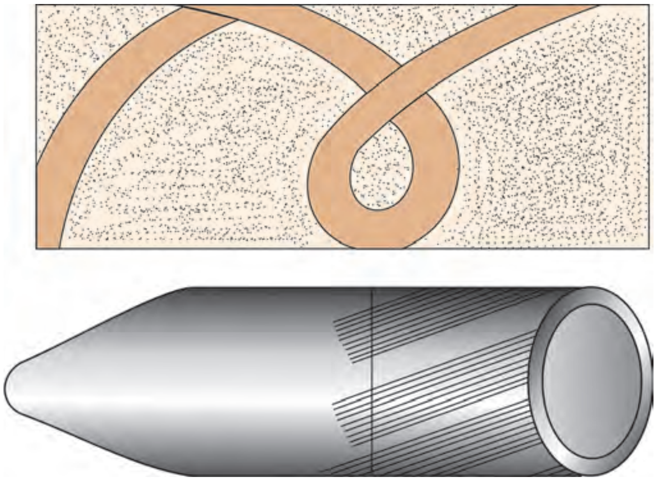
Guns or rifled weapons could be classified into the following six varieties:[15,16] (i) air/gas operated guns/rifles, (ii) 0.303 and 0.22, 0.25, 0.32, 0.39, 0.45, etc. rifles, (iii) pistols (single shot), (iv) revolvers, (v) automatic pistols, and (vi) machine guns (true automatic weapons).

Cartridge of a Gun

Gun cartridge is rifled firearm ammunition, which can be loaded into the chamber of the gun and can discharge the bullet on being fired (Fig. 20.4E). It is made up of two segments—cartridge case and bullet.



Figs 20.4A and B: *Illustrate rifling particulars:* (A) Cross section and longitudinal section of the barrel of a rifled firearm showing lands (elevations) and grooves (depressions), distance between two lands determine the caliber of rifled firearm. (B) Represents clockwise and anticlockwise direction of spinning



Figs 20.4C and D: Illustrates rifling particulars: (C) Magnitude of rifling, (D) Bullet showing rifling marks

Cartridge case – It is an elongated metallic cylinder made up of cupronickel alloy, and its length varies with each type of gun. This has two ends, a flat and closed end, base with a depression in its centre called percussion cap (primer cup) and this accommodates the primer mixture (detonator) next to it within the case. The terminal end of the gun cartridge – holds tightly the bullet in it. Gunpowder is placed in between the detonator and bullet within the cartridge case.

Bullet – It has got a conical shape and four components, namely (i) a body made of cupronickel alloys, (ii) a flat base, which is held in the terminal end of cartridge case, (iii) a pointed tip called the nose, and (iv) a lead core within — which provides the weight of the bullet and therefore the steadiness for the bullet in its flight from muzzle to the target.

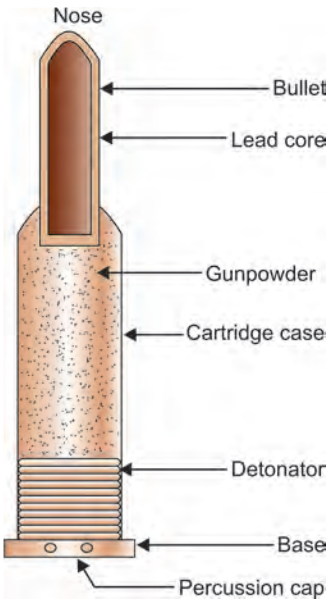


Fig. 20.4E: Diagram of longitudinal section of a rifle cartridge

SMOOTH BORE FIREARMS (SHOTGUNS)

Shotguns are smooth bore firearm weapons, which can fire missiles or cartridges, which contain multiple small lead pellets at the same time.[4,8,9,12,14,16-21] Learning about the following helps in better understanding of guns and its mechanisms:

- Calibre of a shotgun
- Chocking of shotguns
- Classification of shotguns
- Cartridge of a shotgun.

Calibre of a Shotgun

The barrel in a shotgun of any type is perfectly smooth on the inner aspect of its cylindrical wall, and this is the source of the term smooth bore firearm for a shotgun. Caliber or bore of a shotgun is the inner diameter of the barrel. Caliber of the shotgun is usually measured by the two methods: direct method and indirect method.

Direct method — Here the caliber is determined by measuring the inner diameter of the cylindrical barrel directly (Fig. 20.5A) in decimals of an inch or millimetres, e.g., 0.410 shotguns, which means the barrel diameter (inner) is 0.410 of an inch. Thus, among the shotguns, caliber of 0.5 inch (1.25 cm) is maximum.

Indirect method — Here the caliber or bore is determined indirectly by finding out the number of spherical balls of uniform size, prepared from 1 lb (454 gm) of pure lead and each one fitting exactly in the barrel. Thus, there are variety of shotguns ranging from 4 bore to 32 bore depending on the number of such lead spheres which they can accommodate.

Explanation and diagram (Fig. 20.5B), highlights how 6 bore and 12 bore shotguns are calibered.[9]

- *6 Bore shotgun:* In this, barrel of the shotgun exactly fits with each of the 6 spherical balls of uniform size prepared out of 1 lb. of pure lead (i.e. each is 1/6th lb) and the number of balls prepared, i.e. 6 gives the caliber and type of the shotgun as 6 bore shotgun.
- *12 Bore shotgun:* In this, 12 spherical balls of uniform size prepared out of 1 lb of pure lead (each 1/12th lb.) exactly fit the barrel of the shotgun. From the number of balls thus

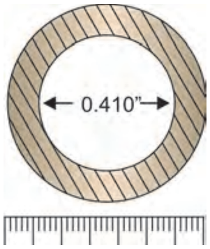


Fig. 20.5A: *Calibre of shotgun:* Direct method

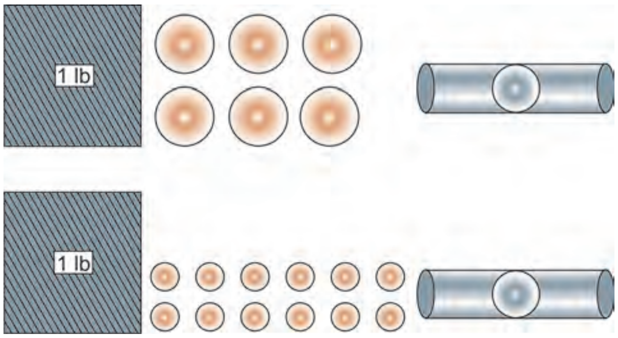


Fig. 20.5B: *Calibre of shotgun:* Indirect method

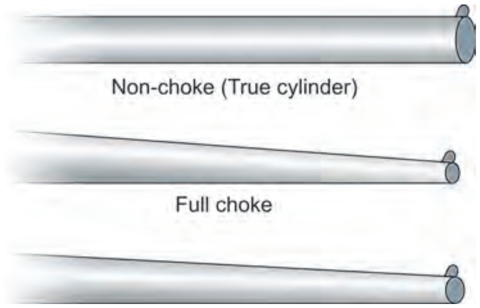


Fig. 20.5C: *Calibre of shotgun:* Choking of shotgun

fitting the barrel (i.e. 12) derives the caliber and type of the shotgun, as 12 bore shotgun. Thus, here the rule is that, as the bore number increases, the inner diameter of the shotgun barrel decreases and vice versa.

Chocking of Shotguns

Normally when a shotgun is fired, the lead pellets spread out soon after their exit from the muzzle end and the degree of dispersion (or area of spread) increases with the range of firing. Choking is a mechanical narrowing mechanism or constriction device at the muzzle end of the shotgun (Fig. 20.5C), which can control to some extent the area of dispersion of the pellets.[4,8,9,12,14-20]

Types of Chokes

Various chokes are devised depending on fraction of an inch by which the muzzle end of the firearm is constricted. Thus, we have shotguns of following types:

- Full choke shotgun (with constriction of 40 thousandth's of an inch)
- 3/4th choke shotgun (with constriction of 30 thousandth's of an inch)
- 1/2 choke shotgun (with constriction of 20 thousandth's of an inch)
- 1/4 choke shotgun (with constriction of 10 thousandth's of an inch).
- Improvised cylinder (with constriction of 3-5 thousandth's of an inch)
- True cylinder is a shotgun with no choking (any constriction at all).

Table 20.1: Advantages of choking in spread area of pellets of a fixed range

Shotgun-extent of choking	*Percentage of shots found in 30" circle at 40 yards (120 ft)*
Full choke	70
Modified/ half choke	60
1/4th choke	50
Improvised cylinder	40

Advantages of Choking for a Shotgun

- It enables the lead pellets or shot to remain together for long distance during their flight from muzzle end to the target.
- It can reduce spread area of the pellets, on the target (victim) aimed (Table 20.1).
- It makes the weapon more lethal.

Classification of Shotguns

Shotguns are classified depending on length and number of the barrels, and on the route through which they are loaded and on their end of barrel choking.[8-14,20]

- Depending on length of barrel there are two types — a short barrelled shotgun and a long barrelled shotgun. Sometimes the barrel is partially sawn off to render the shot gun less conspicuous and more easily carried and concealed; this is quite frequent in illegal use of such forearms.
- Depending on number of barrels there are again two types — a single barrelled shotgun and a double-barrelled shotgun.
- Depending on loading route there are two varieties — a muzzle loading shotgun and a breech-loading shotgun.
- Depending on choking there are several types of shotguns and they are full choked, modified (3/4, 1/2, 1/4) choked, non-choked, etc.

Cartridge of a Shotgun

Shotgun cartridge is the ammunition for smooth bore firearm, which can be loaded into the chamber of the shotgun and can discharge the pellets on being fired.

Structure The shotgun cartridge (Fig. 20.5D) is made up of — cartridge case, gunpowder, wad and pellets.

Cartridge case—It is an elongated cylinder, made up of cardboard or special paper or plastic composition, length of which varies with type of shotgun. It has two ends. Flat closed end known as the base; this is metallic and has a central depressed area called percussion cap (primer cup), which accommodates the primer mixture (detonator) next to it within the case. The second flat end, which is the closed terminal end, and is comprised of a cardboard disk and holds the pellets under it.

Gunpowder—It is placed in the compartment immediately next to the detonator at the base of the cartridge and the pellets at the upper end.

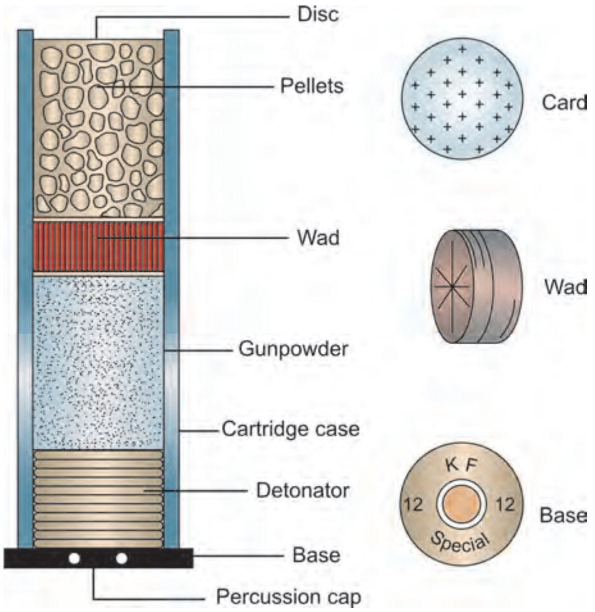


Fig. 20.5D: *Calibre of shotgun:* Longitudinal section of shotgun cartridge with its structure

Wad—It is a rounded disk of some thickness (usually a few millimetres) and made up of compressed paper, felt or plastic material, and it physically separates the gunpowder and pellets.

Pellets—These comprise multiple uniform spherical balls of lead. The cardboard disk and the wad help to keep these pellets as a compressed core in one mass within the cartridge case.

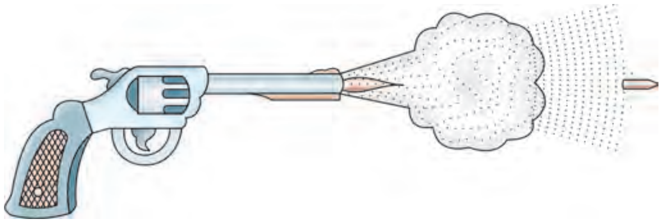


Fig 20.6A: Line drawing representing the various components, which eject on firing from a rifled firearm/ gun, namely - gun flame, gun smoke, soot particles, gunpowder, gases and bullet

WOUND BALLISTICS (Firearm Wounds)

Wound ballistics is the study of tissue penetration of the projectile. This is very important in order to decide several objectives. Though the main objective of study of a firearm wound is to establish the range and direction of firing, the ultimate purpose is to determine the weapon used for firing. Ballistics is the science that involves a detailed study of firearm wounds. It includes injuries caused by the bullet and pellets, shot into or through the body. Firearm wounds are fundamentally of two mains types: wounds of entry and wound of exit. The wound of entry is the wound through which the bullet or pellets enter the body, and the wound of exit is the wound through which the bullet emerges out of the body. As firearms are of two types, wounds produced by each one are dealt separately, under the subheadings:[8-24]

- Rifled firearm (gunshot) wounds
- Smooth bore firearm (shotgun) wounds.

RIFLED FIREARM WOUNDS (Gunshot Wounds)

Rifled firearms wounds are wounds produced by rifled firearms and also known as gunshot wounds.

General Consideration

On firing a gun, there are several components that emerge out along with the bullet or missile, each of which has its own role to play in imparting a specific feature to the wound produced. The components, which are ejected on firing, are the bullet or missile, smoke, which contains soot particles, unburnt gunpowder particles, and hot combustion gases and gun flame. Figures 20.6A and B represent the same. Though the bullet is responsible for the injury produced, each of the other components in the firing discharge can also impart certain specific effects depending on range of firing[9, 16, 20-24] (Table 20.2).

Each of these specific effects mentioned in the Table 20.2 are enumerated and discussed individually in detail:[16-24]

- Heat combustion effect
- Soiling of the wound:
  - Smudging
  - Blackening
  - Tattooing
- Blast effects
- Facial distortion effects
- Cherry red discolouration
- Abrasion and grease collar

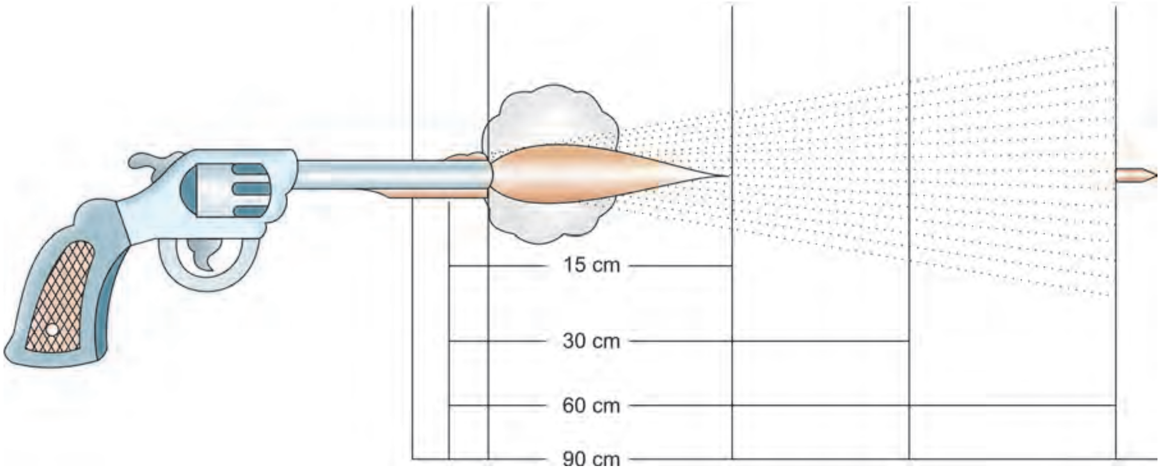


Fig. 20.6B: Line drawing presenting the distance traveled by various components ejected on firing

Table 20.2: Distance travelled by the components of rifle firearm discharge and the special effects produced by them

Components	*Distance travelled*	*Specific effects*
Gun flame	15 cm (6")	Heat combustion effect
Gun smoke		
Soot particles	30 cm (12")	
Gun powder		Smudging
Fine particles	60 cm (24")	Blackening *
Course particles	90 cm (36")	Tattooing *
Gases	Contact shot	Blast effects Facial distortion Cherry red discolouration
Bullet	Variable	Abrasion collar Grease collar Wound of entry Wound of exit

* Collectively called soiling of the wound

- Wound of entry
- Wound of exit.

Heat Combustion Effects

The flame emerging out of the muzzle end of the gun on firing is responsible for producing a heat combustion effect and the effect of the high temperature of these gases is usually seen on the garments and skin's hair around the wound of entry.[8-15]

Effects on garments—These comprise of three characteristic changes, namely

- *Ironing effect*—wherein the cloth looks as if freshly pressed with hot iron.
- *Melting effect*—especially if the clothing is of synthetic origin.
- *Burning effect*—particularly with non-synthetic natural fibres.

Effects on hair—The changes observed are described as ***singeing*** and it is a common finding with contact/close shot range firing. Singed hair will be grossly swollen, curly, fragile (mere touching makes these hairs fall off), blackish (in blondes), and emits a peculiar odor (due to keratin burning). The tip of the hair is swollen and resembles the bulb/root of the hair.

Microscopy—shows increase in width of the hair, as well as vacuolisation within the shaft.

Medicolegal importance—from the heat combustion effects the following can be inferred:

- Helps to distinguish the wound of entry from wound of exit
- Can assist with the assessment of the range of firing (contact shot, near contact shot, and close shot).

Soiling of the Wound

Gunsmoke from burnt powder and gunpowder are solely responsible for the soiling effect (Figs 20.6A and B) and it is usually seen around the wound of entry (on the skin or the clothing worn) 18-24 and includes.

Smudging/blackening—It is due to the deposition of smoke particles, lead or metal of which the missile is made of.

Tattooing/peppering—It is due to deposition of fine or coarse gunpowder particles, which become truly impregnated into and mark the skin, and cannot be washed or scrubbed off.

Medicolegal importance—Smudging and tattooing effects can help to assess the range of firing, as below.

- If both are present, range of firing is close or near shot.
- If both are absent, range of firing is a distant shot.
- If both are found deeper, i.e. on the inner aspect of the wound, range is contact shot.
- If only tattooing is present, range is near shot.
- Thus as the range increases, blackening fades first, followed by tattooing, the reason being their causative agents cannot travel beyond a fixed distance (Table 20.2).

Blast Effect

Blast effects are basically due to the hot gases formed on combustion of the gunpowder and are seen around the wound of entry. These are also under marked pressure and, in accordance with the known physical properties of hot gases; this disrupts tissues at the entry site.

Mechanism—When the firing is done with the muzzle end held firmly pressed against the site aimed at (i.e. contact shot range), the gases evolved (namely — CO, CO_2, etc.) on combustion of the gunpowder liberate huge pressure. These gases accumulate between the skin and subcutaneous tissues below and in other natural tissue planes, particularly over bony prominences as the skull and induce a blast effect[9-16] (Fig. 20.7A) such as:

- Tearing of skin — due to efforts made by the gases accumulated to come out of the wound of entry.
- Eversion of wound edges — as the gases try to flow out of the wound of entry, the wound edges become everted.
- Muzzle end imprint abrasion — due to the skin being pushed forwards with force and this impinging against the muzzle end of the weapon by the gases accumulated underneath.
- Formation of blood and gunpowder pocket - when the wound of entry is not sufficiently large, blood and gunpowder can get accumulated under the skin and subcutaneous tissue.
- Pressure extrusion — fabric fibres, subcutaneous soft tissues, blood, fat, etc. can extrude out of the wound along with the gases that are emerging out of the wound of entry.
- Soiling of bones (periosteum) and garments (inner aspect) — are due to the soot particles reaching inside of the wound at a contact shot range.

Facial Distortion Effects

In contact shot firing anywhere on the head, the gases from the firing discharge can enter into the skull, while then tries to accommodate this increase in its contents. These results in an acute building up of intracranial pressure with the rigid cranium attempting to accommodate it by expansion.

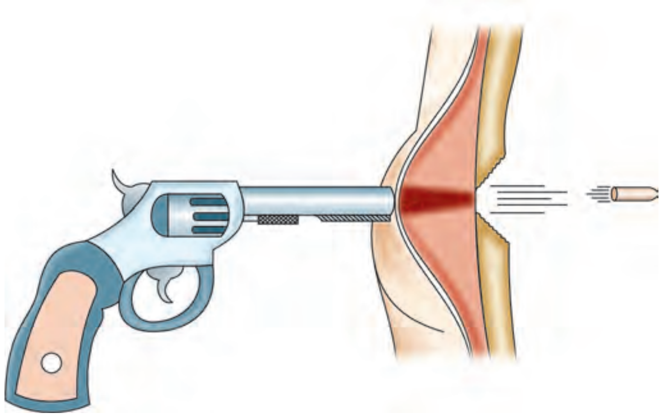


Fig 20.7A: Mechanisms of blast effect

Mechanism — Gases under pressure accumulate inside the cranium and in the process of attempting to release this build-up of pressure, fractures will arise with outward expansion with outwards pushing out of the fracture fragments and consent tears (lacerations) through the skin of the face as these sharp and tough bony fragments are pushed outwards. This is seen especially on the face and this produces facial distortion effects[8, 9-16] such as:

- Over expansion or swelling of tissues of the face
- Tears of the skin can occur in front of the ears and along the creases on either side of the mouth and nose, along the inner canthus of the eyes, etc.
- Fractures of the skull and facial skeleton.

Medicolegal Importance

- The changes can help in location of wound of entry
- Disfigurement resulted, however, can create difficulty in establishing identity of the deceased.

Cherry Red Discolouration

Carbon monoxide gas evolved on burning of gunpowder is the causative factor for this effect. It is usually seen in the tissues around and beneath the wound of entry.

Mechanism — Carbon monoxide gas can combine with the hemoglobin and myoglobin in the blood/muscle tissues respectively resulting in the formation of carboxyhemoglobin and carboxymyoglobin, which are cherry red in colour.[9, 13, 16, 22] This results in cherry red colour in muscles around the wound of entry.

Medicolegal Importance

- The change can help to identify the wound of entry
- It can also help to establish the range of firing.

Abrasion and Grease Collar

Gyroscopic movement of the bullet, the skin's elasticity, the grease and dirt particles on bullet's surface are the major causative factors of abrasion and grease collar. They are seen on the skin around the wound of entry at all ranges[9-16, 22] (Fig. 20.7B).

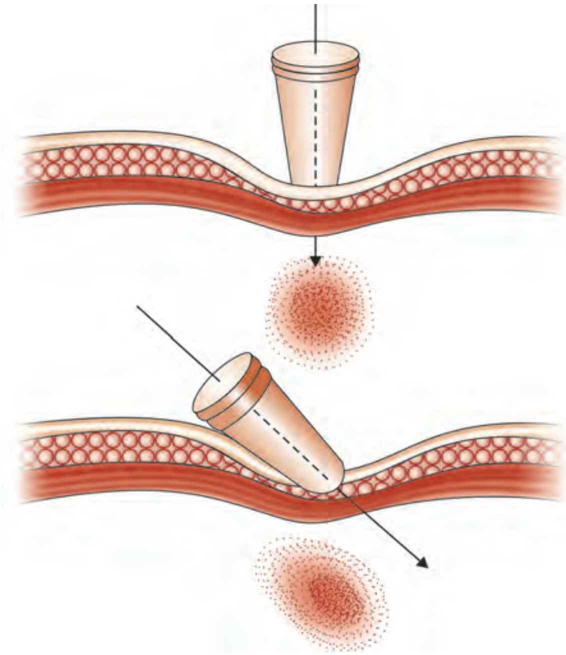


Fig.20.7B: Abrasion collar in a bullet entry wound

Mechanism — When a bullet comes in contact with the skin in the process of penetrating into it against its elasticity, the axial spinning action of the bullet develops great friction with skin and produces a collar of abrasion all around the wound of entry. The garment or skin, while yielding to the gyroscopic movement of the bullet, is perforated; the bullet wipes off all the dirt or grease particles (used to lubricate the barrel) lying on the bullet, which get deposited around the wound of entry, producing the grease collar.[9,13-22] These partakes are warm and this are easily impregnated into the skin.

Appearance — Each one is discussed separately.

Abrasion collar — seen as a marginal abrasion around the wound of entry. Depending on the angle of entry made by the bullet, i.e.

- A perpendicular entry of the bullet develops the abrasion collar of rounded shape.
- An acute angular entry of the bullet develops the abrasion collar of an oblique or oval shape.

Grease collar — Mostly seen on the clothing and is seen as a black deposit, sharply out-lined as if printed (better-seen on light colour clothes and on fair-skinned individuals).

Medicolegal importance — Helpful in deriving information such as wound of entry and direction of firing.

Wound of Entry

Wound of entry depends on range of firing (Figs 20.8A to J) and usually for a rifle firearm, four different ranges are observed[9-16,22] and they are: (i) contact shot range, (ii) close shot range, (iii) near shot range, and (iv) distant shot range (Fig. 20.8A).

Contact Shot Range

Here the muzzle end of the firearm is held in—touch or in contact with skin. The appearances of this wound are as depicted in (Fig. 20.8A and E).

Size — largest among all the four ranges

Shape — crater form/stellate

Blast effects — only two effects observed, they are muzzle end imprint abrasion and eversion of wound edges

Blackening — usually not seen around the wound on outside, but may be seen on its deeper or inner aspects.

Tattooing — usually not seen around the wound on outside, but may be seen on its deeper or inner aspects.

'*Cooking*' *and/or cherry red discolouration* of tissue inside wound — present.

Singeing and/or scorching — absent

Abrasion collar — absent

Grease collar — absent.

Close Shot Range

Here the muzzle end of the gun is held about 1" to 3" (2.5-7.5 cm) away from the target aimed at. The appearance of the wound is as depicted in Figures 20.8B and 20.8F.

Size — smaller than a contact shot wound

Shape — circular or oval

Blackening — usually seen around the wound covering a smaller spread area

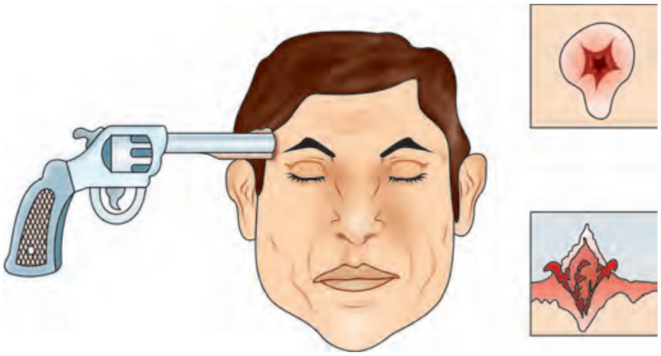


Fig 20.8A: Line drawing illustrating the wound of entry by a rifled firearm: Contact shot range

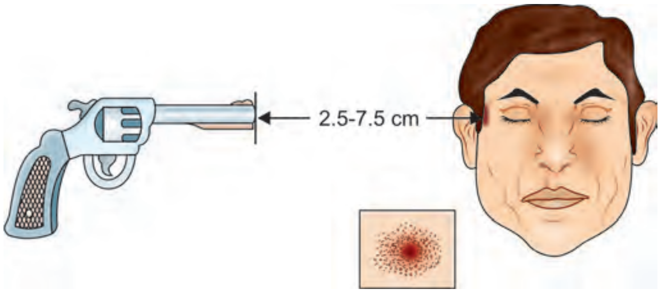


Fig 20.8B: Line drawing illustrating the wound of entry by a rifled firearm: Close shot (2.5-7.5 cm) range

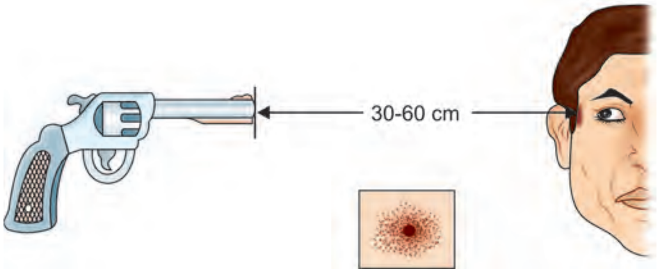


Fig 20.8C: Line drawing illustrating the wound of entry by a rifled firearm at various range of firing: near shot (30-60 cm) range

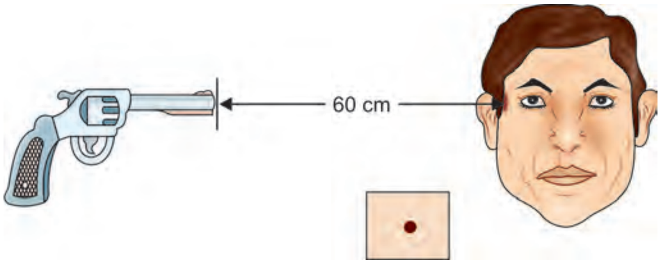


Fig 20.8D: Line drawing illustrating the wound of entry by a rifled firearm at various range of firing: distant shot (> 60 cm) range

Tattooing — usually seen around the wound covering a smaller spread area

Singeing and/ or scorching — present

Abrasion collar — present

Grease collar — present.

Near Shot Range

Here muzzle end of the gun has been held at about 12" to 24" (30-60 cm) away from the target. Appearance of the wound is as depicted in Figures 20.8C and 20.8G.

Size — smaller than bullet.

Shape — circular or oval.

Blackening — usually seen around wound with large spread area (max 30 cm).

Tattooing — usually seen around wound with large spread area (maximum 60 cm).

Singeing — absent.

Abrasion collar — present.

Grease collar — present.

Edges — usually inverted, but could be everted also. Whenever the site of injury has abundant adipose tissue, it makes an attempt to flow out of the wound and this can turn the edges everted.

Distant Shot Range

Here the muzzle end of the gun is held beyond 24" (60 cm) away from the target. Wound apparently resembles a near shot wound. Appearance of the wound is as depicted in Figure 20.8D and 20.8H.

Size — smaller than near shot wound.

Shape — circular.

Blackening — absent (on both outer and inner aspect of the wound).

Tattooing — absent (on both outer and inner aspect of the wound). However, it may be seen around the wound of entry to a maximum range of 36" or 90 cm.

Singeing — absent.

Abrasion collar — present.

Grease collar — present.

Edges — inverted.

The details of the wound of entry for a rifled firearm at different ranges and differences between entry wound and wound of exit are summarised in Tables 20.3A and B respectively.

Wound of Exit

Wound of exit is described under four heads — site, size, shape, and other findings[9-24] (Figs 20.8I and J).

Site — opposite to the wound of entry

Size — four possibilities are observed and they are:
- At closer range — smaller than wound of entry
- As the range increases — exit wound size increases
- With a high velocity bullet — both entry wound and exit wound are of equal size
- With deformed bullet — a big exit wound is produced.

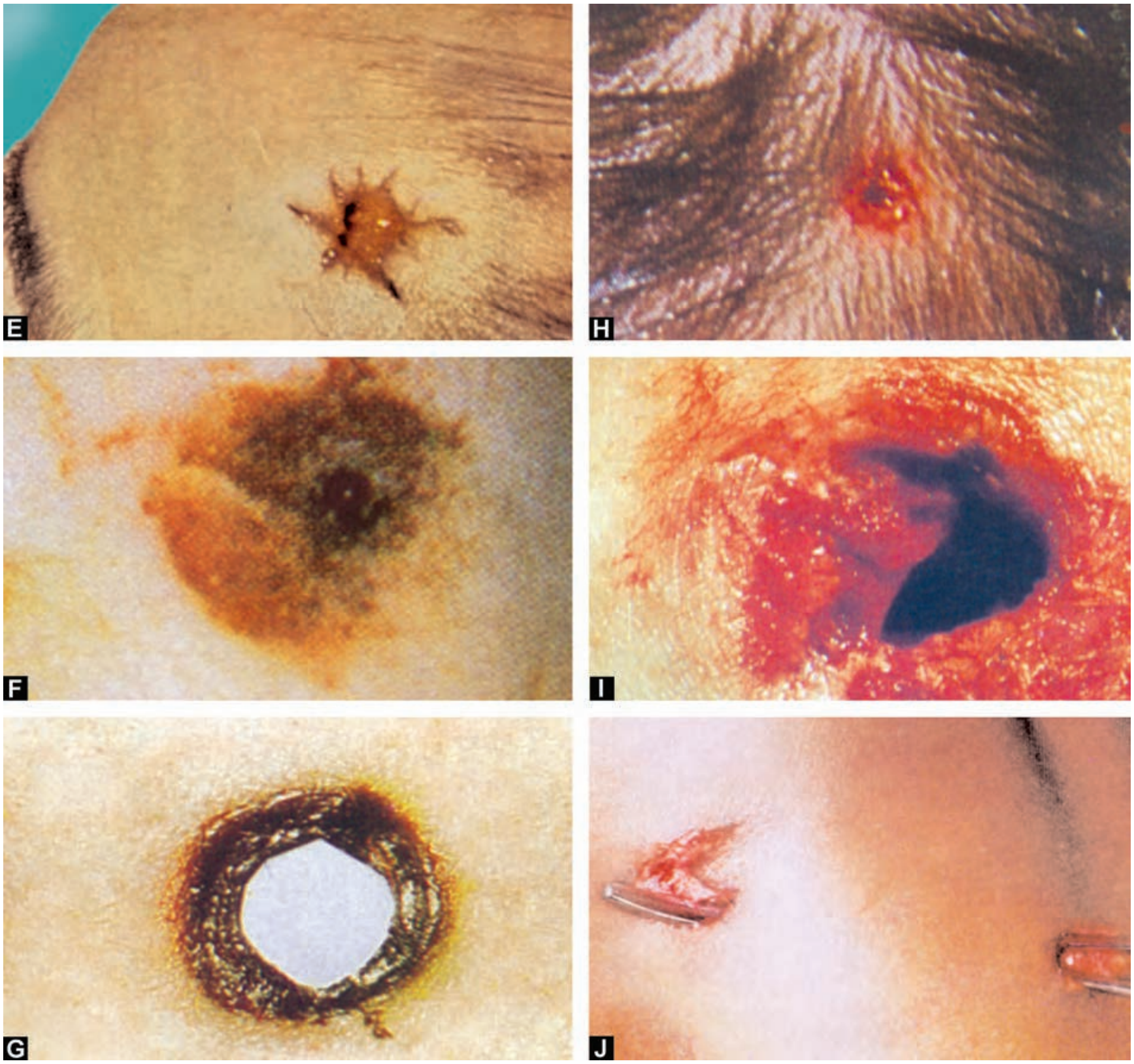
Shape — irregularly circular.

Other Findings

Edges — puckered/torn/everted

Blackening — absent

Portion of fat and other soft tissue - may be present



Figs 20.8E to J: Photographs illustrating entry wound of rifle firearm at various ranges of firing: (E) contact shot, (F) close shot, (G) near shot; note—abrasion collar (piece of the skin with the wound), (H) distant shot on the hair partition of the forehead, (I) exit wound; note—it is large and has everted, irregular margins, showing no abrasion or grease collar, (J) an unusual entry and exit wound in a superficial injury over back. The circular appearance of entry wound, contrasts well with irregular, lacerated bigger exit wound. This could be due to 'wobble' imparted by contact of the bullet with underlying rib

Tattooing — absent

Singeing — absent

Abrasion collar — absent

Grease collar — absent.

PECULIAR EFFECTS OF RIFLED FIREARMS/ GUNSHOT WOUNDS

Rifled firearm (gunshot) wounds though mostly remain typical, at times may exhibit peculiar changes, which are enumerated and described.[9-34]

1. Atypical entrance wound such as:
    - Large atypical entrance wound
    - Bullet bruise
    - Bullet graze/slap
2. Ricochet bullet
3. Single entry wound and multiple exit wounds
4. Multiple entry and exit wounds, single bullet recovered
5. Entry wound present, but no bullet/no exit wound
6. Gutter fracture of skull
7. Tandem/piggy back bullet effect
8. Frangible bullet effect
9. 'Dumdum' bullet effect
10. Artifacts
    - Rayalaseema phenomena
    - Souvenir bullet
    - Magic bullet
    - Concealed firearm wounds.

Large Atypical Entrance Wound

Usually entrance wound is the largest at contact shot range firing. Size of the wound gets reduced as the range increases. When the size of the wound increases to an unusually larger size, more than that of contact shot range or at any other known range, it is considered as large atypical entrance wound.[9,16,20]

Table 20.3A: Wound of entry for rifled firearm at different ranges — Summary of details

Characteristics	*Contact shot*	*Close shot*	*Near shot*	*Distant shot*
Range	In-touch with skin	2.5-7.5 cm	30-60 cm	>60 cm
Size	Largest size	Bullet size	Smaller	Smallest
Shape	Stellate	Circular	Circular	Circular
Blast effect				
• Muzzle end imprint	+ve	–ve	–ve	–ve
• Edge eversion	+ve	–ve	–ve	–ve
Other findings				
• Blackening	–ve	+ve	–ve	–ve
• Tattooing	–ve	+ve	+ve	–ve
• Singeing	–ve	+ve	–ve	–ve
• Abrasion collar	–ve	+ve	+ve	–ve
• Grease collar	–ve	+ve	+ve	–ve

Table 20.3B: Difference between wound of entry and wound of exit

Characteristics	*Wound of entry*	*Wound of exit*
Size	• Small if near shot • Large if distant shot	• Larger when near shot • Smaller if distant shot
Margin	Inverted	Everted
Soiling of wound	+ve	–ve
Abrasion collar	+ve	–ve
Contusion collar	+ve	–ve
Grease collar	+ve	–ve
Bleeding	Less	More
Foreign bodies	+ve	–ve
Dispersion of pallets (Shot gun)	+ve	–ve
Protrusion of fat	–ve	+ve
Bright red (carboxy Hb)	+ve (Near shot)	–ve
X-ray Metallic Ring	+ve	–ve
Muzzle impression	+ve (contact shot)	–ve
Blast effect	+ve (contact shot)	–ve

Causes—Probable causes are enumerated:

- Use of bullets of poor quality
- Wobbling of the tail of the bullet after leaving the muzzle end.

Mechanism—Both causes mentioned above could reduce the penetration power of the bullet, which therefore produces greater tissue damage at the wound of entry, making it unusually large irrespective of range of firing.

Medicolegal importance—May pose difficulty in assessing the range of firing by examining entry wound.

Bullet Bruise

Bullet here just hits the skin only and drops down to the ground, without any penetration or wound of entry, producing a bruise alone at the site of impact.[9] Gunshots can also produce bruise patterns on persons who wear soft body armor when shot even though the armor stops the bullets.[26]

Causes—Probable causes are:

- Use of unjacketed bullets
- Worn out of gun barrel
- Bullet used is smaller than gun calibre.

Mechanism — Any of the causes mentioned above can lead to the decrease in velocity of the bullet which in turn leads to loss of the power of penetration making it act like any other blunt object, producing just bruising only.

Appearance — Injury produced is a linear bruise of the size of the bullet or a little more, showing all typical colour changes as any other bruise.

Medicolegal importance — It may be mistaken for any other blunt weapon injury. Colour changes can give clues regarding time since injury.

Bullet Graze (Bullet Slap)

Here the bullet just hits the skin only at an angle and goes off from the site, without any penetration or wound of entry, producing an abrasion or a laceration depending on the thickness of the skin involved[9, 16, 27] (Fig. 20.9A).

Causes—Probable causes are:

- Poor quality bullet
- Poor aiming

Mechanism—Any of the causes mentioned above can make the bullet just touch the skin only and then go beyond the wound.

Appearance—Injury could be a scratch mark or a lacerated furrow or an incised like wound.

Medicolegal importance—Wound may be mistaken for a blunt (or rarely sharp) weapon injury.

Ricochet Bullet

Ricochet bullet is a peculiar effect on the wound of entry due to the ricocheted or deflected or rebounded bullet[9-16] (Fig. 20.9B).

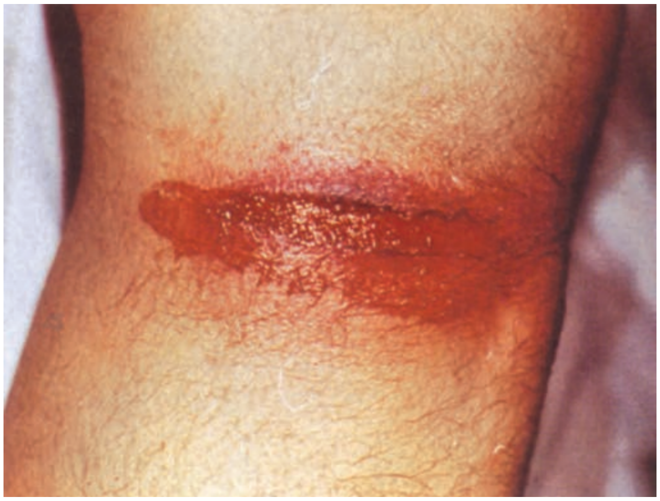


Fig. 20.9A: Peculiar effects of rifled firearm: Bullet graze—on right leg just below the knee (outer aspect)

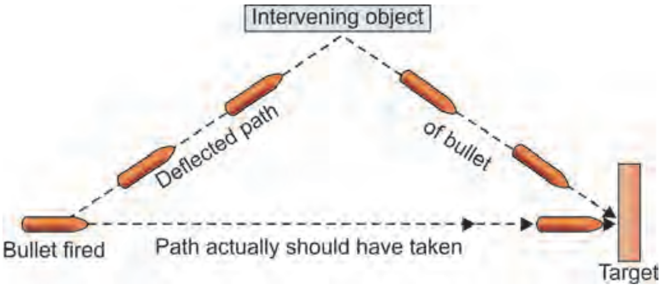


Fig. 20.9B: Peculiar effects of rifled firearm: Ricochet bullet, mechanism

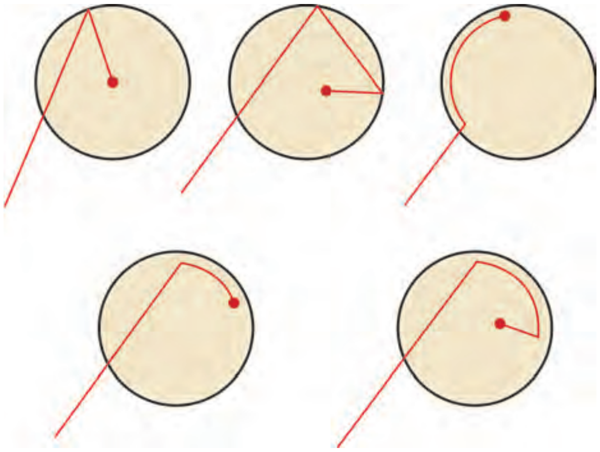


Fig. 20.9C: Peculiar effects of rifled firearm: Ricochet bullet inside the skull—mechanism and types

Causes — Probable causes are:

- Using a firearm of inferior quality
- Using low-velocity bullet.

Appearance — The wound of entry will show peculiar effects such as:

- *Size* — large (irrespective of range)
- *Shape* — keyhole like/elongated
- *Edges* — ragged.

Examination of the bullet — Bullet might be:

- Deformed/flattened bullet
- Carry the paint/dirt or any such other things from the deflected object on its surface.

Mechanism — Any of these cases mentioned above can bring about loss of gyroscopic movement, resulting in deviation in its path slightly, hit some other object, get deformed and then penetrate a victim/target that may or may not be aimed at.

Examples

Intracranial bullet injuries are quite often ricochet bullet injuries. Bullet which enters cranium may bounce with inner table of the skull, and undergo ricochet effect and travel in a deflected direction inside the skull, getting lodged in an area away from the usual track a bullet can take when not undergoing ricochet effect (Fig. 20.9C). There are five different possible ricochet mechanisms inside the cranial cavity[9,10,15,16] as given below:

1. Single ricochet — bullet rebounds only once.
2. Double ricochet — bullet rebounds twice.
3. Inner tangential of entrance side — bullet glides on entering into the skull of the wound of entry.
4. Inner tangential at contralateral side — bullet on hitting the opposite side to wound of entry glides on it and travels to some distance.
5. Inner tangential at contralateral side and ricochet — bullet after gliding rebounds into another direction.

Medico legal importance — It might explain the path taken by the bullet in the deceased and cause of death.

Single Entry and Multiple Exit Wounds

Explanation — A bullet after entering into victim's body may be broken into several pieces by hitting against bony structures and then each fragment ejects out like an individual missile (secondary missile) thus producing multiple wounds of exit[9,16] (Fig. 20.10A).

Multiple Wounds of Entry and Exit, but a Single Bullet Recovered

Explanation — The body of the victim may be in such a position while receiving the crime bullet that in its course of transit, it will enter into and leave the body several times producing several wounds of entry and wounds of exit successively, e.g. a bullet passing through a victim sitting with knees touching the chest, wounds of entry and exit of the bullet may be seen on back, chest, knees, etc. respectively (Fig. 20.10B). Such possibilities have to be very carefully considered in reconstructing the scene in a fatal shooting. This is of particular importance when more than one firearm is involved and several bullets have been fired in the same incident.[9,16]

Entry Wound Present, But No Exit Wound or Bullet Recovered

Explanation — The bullet after entering into the body might enter one of the natural passages and may be lost either being coughed out or vomited out or passed out through anus. It may not be recovered if it gets lodged in the bones (Fig. 20.10C). It may also enter a large blood vessel and be embolised to distant place far from expected site of lodgement (bullet embolus). X-ray survey of whole body is essential in all deaths by shooting to locate the bullet and this should take place before the body is subjected to an internal examination.[9,16]

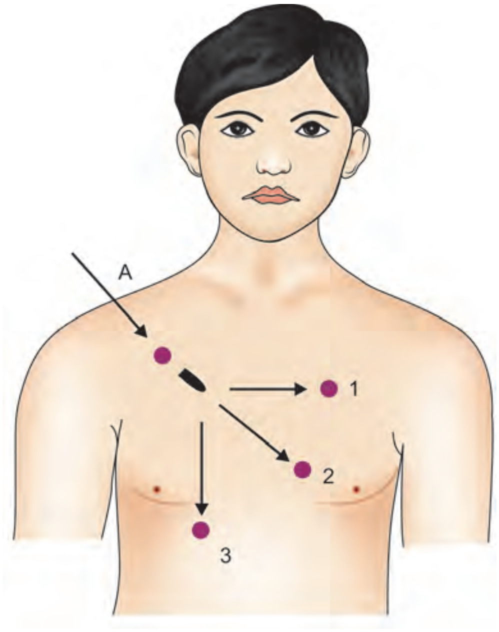


Fig. 20.10A: Peculiar effects of Rifled firearm-single wound of entry, multiple exit wounds

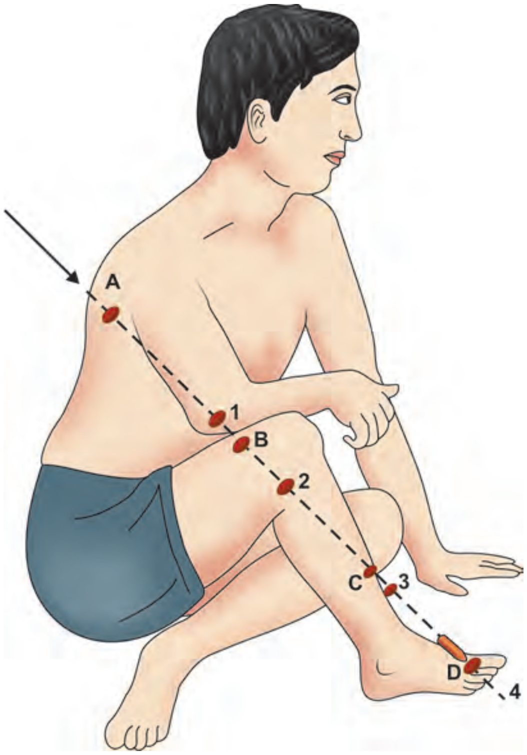


Fig. 20.10B: Peculiar effects of rifled firearm—multiple wound of entry (A, B, C, D) and exit (1, 2, 3, 4), but single bullet recovered

GUTTER FRACTURE (Refer Fracture Skull)

Tandem (Piggy Back) Bullet Effect

Here the wound of entry in its depth or along its track shows more than one bullet arranged exactly one behind the other in a row. This is particularly of importance if an inferior, old, unused firearm is discharged without prior proper cleaning of its barrel. Here every possibility is there that the bullet fired earlier might not have been ejected from the barrel, but remained lodged in the barrel itself, and is just carried away in the course of a second discharge of the firearm along with the bullet fired afresh; both get lodged together into the wound of entry.[28]

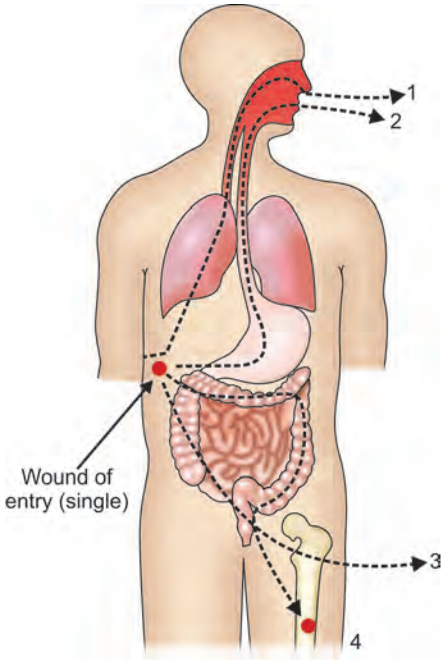


Fig. 20.10C: Peculiar effects of Rifled firearm - wound of entry present, but no wound of exit or bullet detected. 1, 2, 3-Possible routes through which the bullet might have flown out and lost 4-Bullet got lodget in the bone

Frangible Bullet Effect

Frangible bullet effect is an inferior bullet, which on penetrating into the body gets broken up into several fragments and produces severe injuries, by each fragment. When a frangible bullet impacts a solid surface such as a steel plate or backstop, complete fragmentation occurs with the bullet disintegrating into small pieces.[29,30]

Dum Dum Bullet Effect

This effect is due to the special modifications in the bullet rendering it more lethal. (The name has been coined from a town in India where such bullets were first used.) Here a bullet has a special device at its tip (nose) which, on coming in contact with the target aimed at, bursts open into several fragments, each of which can act like an individual missile and produce fatal injury, over a larger area.[31]

Rayalaseema Phenomenon

This is an artifact. Here a victim was first killed by stab injury and then to mislead the investigating officer, the criminal stuffed the stab wound with a bullet manually. This was unraveled only after a meticulous autopsy examination. Since the whole incident occurred in the Rayalaseema district of Andhra Pradesh State in India, the terminology for the condition was coined accordingly[9,10] (Fig. 20.10D).

Souvenir Bullet

This refers to a condition where in a bullet is preserved for a long time, e.g. >25-30 years in the body. A dense fibrous tissue

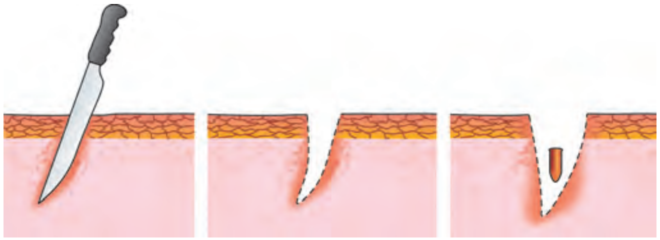


Fig. 20.10D: Peculiar effects of rifled firearm: Rayalaseema phenomena

capsule usually surrounds it. A small scar indicates the original wound of entry. Absence of fresh bleeding in the vicinity and inability to locate the recent track from site of lodgement of bullet to wound of entry confirms long presence of bullet inside the body. Bullets form the two world wars keep turning up with some regularity on CT scans carried out for different conditions. Lead poisoning may also result very rarely from lead bullet lodged for longtime in the body, when there is a rich vascularity in the tissue surrounding the bullet, or there is prolonged bathing of the bullet with synovial fluid.

Magic Bullet

The single bullet theory, called the magic bullet theory by critics, was introduced by the Warren Commission to explain how three shots made by Lee Harvey Oswald resulted in the assassination of United States President John F. Kennedy.[33,34] It is also referred to as ***Kennedy phenomena.***[9]

The theory, generally credited to Warren Commission staffer Arlen Specter (a US Senator), posits that a single bullet, (known as "Warren Commission Exhibit 399" / CE399"), caused all of the non-fatal wounds in both President Kennedy and Texas Governor John Connally. The fatal head wound to the President was caused by another bullet.

According to the single bullet theory, a one-inch long, copper jacketed, lead core 6.5 millimeter rifle bullet fired from the sixth floor of the Texas School Book Depository passed through President Kennedy's neck, Governor Connally's chest and wrist and embedded itself in the Governor's thigh. In doing so, the bullet traversed 15 layers of clothing, 7 layers of skin, approximately 15 inches of tissue, struck a tie knot, removed 4 inches of rib and shattered a radius bone.

The bullet that is supposed to have done all this damage was found on Governor Connally's stretcher in the corridor at the Parkland Memorial Hospital in Dallas. It became a key Commission Exhibit, identified as CE399. Its copper jacket was completely intact. The bullet's nose appeared normal, but the tail was compressed laterally on one side.

In its conclusion, the Warren Commission found persuasive evidence from the experts that a single bullet caused the President's neck wound and all the wounds in Governor Connally. It acknowledged that there was a "difference of opinion" among members of the Commission "as to this probability" but stated that the theory was not essential to its conclusions and that all members had no doubt that all shots were fired from the sixth floor window of the Depository building.

The 1978 House of Representatives' Select Committee on Assassinations agreed with the Single Bullet Theory but differed on the time frame. The Single Bullet Theory has been staunchly defended by those who believe the Warren Commission's finding was correct and roundly criticised by those who disagree.

Concealed Firearm Wound

At times due to heavy bleeding from wound of entry a large blood clot may cover the wound of entry and conceal it, till the clot is washed off. Hence, it is essential that the actual site of bleeding is meticulously identified in all bodies in whom exsanguinations appear to be the cause of death.[9,16,21]

SHOTGUN WOUNDS
(Smooth Bore Firearm Wounds)

Shotgun wounds are wounds produced by shotguns or smooth bored firearms.[8-20, 35-42] Shotguns are popular world wide and more of these weapons exist than rifled types.[38] With an increasing incidence and prevalence of gunshot wounds it is important for traumatologists to be familiar with shotgun wound ballistics. Shotgun wounds differ from those of other missiles because the spectrum of wound severity is large owing to the fact that the pellets scatter as they travel.[31] Like gunshot wounds the various aspects of the shotgun wounds are better studied under following sections:[8-21,28,31]

- General considerations of shotgun wounds
- Wound of entry
- Wound of exit.

General Considerations of Shotgun Wounds

1. A shotgun when fired discharges the components such as gun flame, gun smoke containing soot particles and gunpowder particles, cards, wads, gases and lead shot. Like the various parts of gunshot components each of these can produce a specific effect to the wound produced. However, prior to knowing the various effects produced by them, it is important to know the range through which each component particle can travel. The maximum distance travelled by each of these components and the specific effects produced by them at the wound of entry are given below in Table 20.4.
2. Thus, though most of the effects mentioned above resemble those of rifled firearm (gunshot) wound a significant difference is observed with wound of entry and exit.

Wound of Entry

Appearances vary depending on several factors[9,13,16,18,20] such as:

a. Range or distance of firing
    - Contact shot range
    - Other ranges such as:
        - – Close range (less than 15 cm, at 15 cm, at 90 cm)
        - – Near shot range (at 2 m)
        - – Distant range (at 4 m and at 20 m).

b. Size of pellets
    - Smaller pellets—produce wounds which are smaller
    - Bigger pellets—produce wounds, which are bigger.

c. Types of gunpowder
    - Smokeless—produces no soiling effect of the wound
    - Black gunpowder—produces a clear soiling effect on the wound.

Wound of Entry at Different Ranges

As the pellets remain together up to a distance of 90 cm. usually the wound of entry produced will be single. However, when the range of firing is beyond 90 cm the pellets begin to spread and each one acts as an independent missile producing multiple smaller wounds over a fixed area. Appearances of wound of entry at various ranges are given in Table 20.5 (Figs 20.12 to 20.15).

Table 20.4: Distance travelled by components of the shotgun firing discharges with the specific effects produced by them

Components	*Distance*	*Specific effects*
Gun flame	15 cm (6")	Heat combustion effect on garments, hair (singeing), etc
Gun smoke		
1. Soot particles	60 cm (24")	Blackening*
2. Gunpowder – Fine particles	30 cm (12")	Blackening*
– Coarse particles	90 cm (36")	Tattooing*
Cards	2 m (96")	Minor injuries
Wads	1.25 m (60")	Minor injuries
Gases	Contact and close shot ranges	Blow back effect, cherry red colouration around wound of entry
Lead shots	Varies ball ricochet effect.	Wound of entry and exit, billiard

* Collectively called as soiling of the wound

Table 20.5: Wound of entry by a shotgun (smooth bore firearm) at different ranges — summary of details

Characteristics	*Range/distance of firing*						
	Contact shot	Close shot ranges			Near shot	Distant shot	
Range	Contact range	Close range	Short to Mid range			Mid to distant range	
	In touch	< 15 cm	15 cm	90 cm	2 m	4 m	20 m
Number of wounds	Single	Single	Single	Single	Multiple	Multiple	Multiple
Spread area	—	—	—	—	5-7 cm	10-14 cm	> 14 cm
Site	Vital parts	Any where on the body					
Size	Largest (> 2.5 cm)	Smaller	Smallest*	2.5-4 cm	—	—	—
Shape	Irregular**	Circular	Circular	Rat hole*	Central big wound with smaller wounds around	Wider spread	Spread is not measurable
Edges:	Scorched and contused	Well defined and inverted		Irregular and lacerated	—	—	—
Blackening	+ + +	+ + +	+ +	+	—	—	—
Tattooing	+ + +	+ + + +	+ + +	+ +	—	—	—
Singeing	+ + +	+ + + +	+ +	—	—	—	—

* *Both are called as close range*
** *Irregularly circular with crenated and scalloped edges*

Note:
1. *Close-range shotgun wounds can be as destructive as those from a high-velocity rifle, however, longer the weapon-victim range-injury produced is minimal.*
2. *The type of shot (size and weight of pellets) used also determines the type of injury, usually more serious injuries are produced by the larger type of buckshot (greater than 0.14 inches in diameter).*

Blow Back Effect

It may be compared to the blast effect of a gunshot wound. Mechanism mainly involved is that in contact shot or close shot range firing, the gases evolved may get accumulated under the skin resulting in a bigger wound of entry, muzzle end imprint abrasion, distortion of the face when shot with muzzle end kept pressed against the roof of the mouth, etc.

Billiard ball ricochet effect — This is a peculiar effect wherein though the firing is done at close range, the appearance of the

John F Kennedy Assassination — Autopsy[33,34]

John F Kennedy, was the Thirty-Fifth President of USA, Term: 1961-1963, was born on May 29, 1917 in Brookline, 32 Massachusetts, died on November 22, 1963, was killed by an assassin's bullet in Dallas, Texas. He was married to Jacqueline Lee Bouvier Kennedy. The autopsy of President John F. Kennedy was performed, beginning about 8 p.m. and ending about midnight EST, on Nov. 22, 1963, the day of his assassination, at the then Bethesda Naval Hospital in Bethesda, Maryland. The choice of autopsy hospital in the Washington, D.C. area was made at the request of Mrs. Kennedy, on the basis that John F. Kennedy had been a naval officer.

Earlier Testimony from Dallas Doctors

The back wound

The death certificate, signed by the President's personal physician Dr. Burkley, an Admiral in the U.S. Navy, gave a location for the back wound lower than found by the later autopsy (either its photographs or measurements). Dr. Burkley believed a bullet to have hit Kennedy at "about" the level of the third thoracic vertebra. Supporting the location of Dr. Burkley is a diagram from the autopsy report of Kennedy, which shows a bullet hole in the upper back. However, this diagram is freehand and not drawn with any attention to landmarks — a criticism made of it by the later HSCA analysis.

Burkley's location at T3 is also about the same location of the bullet hole in the President's shirt and the bullet hole in the suit jacket worn by Kennedy· which shows bullet holes between 5 and 6 inches below the top of Kennedy's collar. However, again there has been controversy on the matter of whether or not the holes in the president's clothing should be expected to correspond to the location of his back wound, since he was sitting with a raised arm at the time of the assassination, and multiple photographs taken of the motorcade show his suit jacket bunched at the back of his neck and shoulder, so that it did not lie closely against his skin.

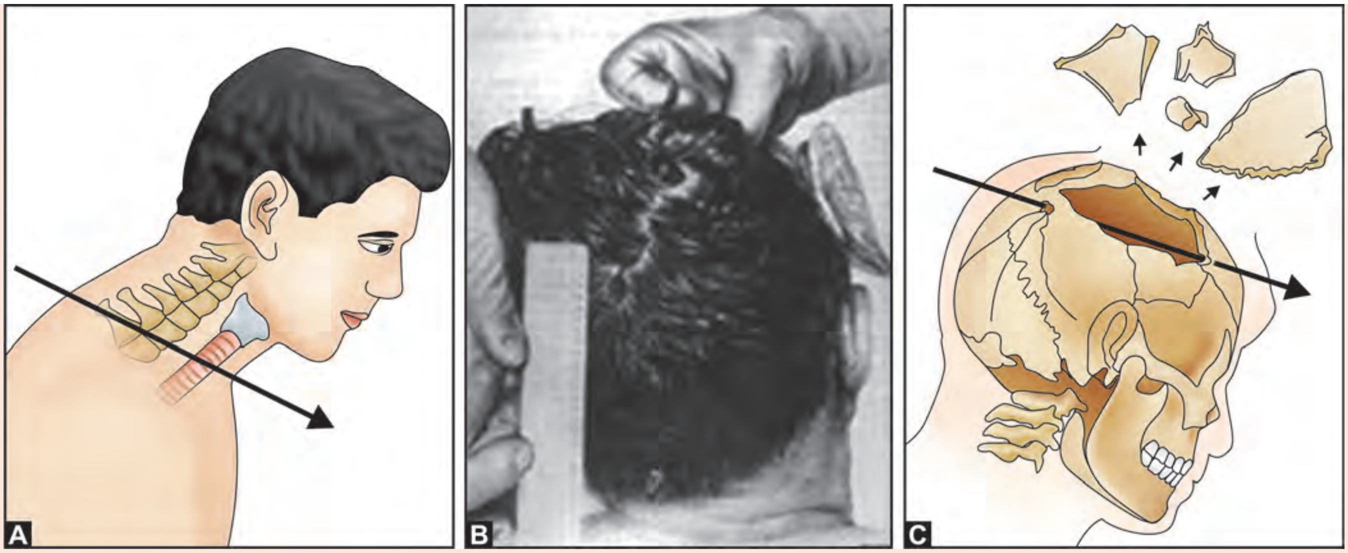
Official Findings of the Autopsy

The missile wound in the back

1. The Bethesda autopsy physicians attempted to probe the bullet hole in the base of Kennedy's neck above the scapula, but were unsuccessful as it passed through neck strap muscle. They did not perform a full dissection or persist in tracking, as throughout the autopsy, they were unaware of the exit wound at about the same level, at the front of the throat. Emergency room physicians had obliterated it when they performed the tracheostomy.
2. At Bethesda, the autopsy report of the president, Warren Exhibit CE 386 described the back wound as being oval, 6 × 4 mm, and located "above the upper border of the scapula" [shoulder blade] at a location 14 cm (5.5 in) from the tip of the right acromion process, and 14 cm (5.5 in) below the right mastoid process (the bony prominence behind the ear).
3. The concluding page of the Bethesda autopsy report, states: "The other missile [the bullet to the back] entered the right superior posterior thorax above the scapula, and traversed the soft tissues of the supra-scapular and the supra-clavicular portions of the base of the right side of the neck.
4. The report also reported contusion (bruise) of the apex (top tip) of the right lung in the region where it rises above the clavicle, and noted that although the apex of the right lung and the parietal pleural membrane over it had been bruised, they were not penetrated, indicating passage of a missile close to them, but above them. The report noted that the thoracic cavity was not penetrated.
5. This missile produced contusions of the right apical parietal pleura and of the apical portion of the right upper lobe of the lung. The missile contused the strap muscles of the right side of the neck, damaged the trachea, and made its exit through the anterior surface of the neck."
6. The ***single bullet theory*** of the Warren Commission Report places a bullet wound at the sixth cervical vertebra of the vertebral column, which is consistent with 5.5 inches (14 cm) below the ear. The Warren Report itself does not conclude bullet entry at the sixth cervical vertebra, but this conclusion was made in a 1979 report on the Kennedy assassination by the ***House Select Committee on Assassinations (HSCA)***, which noted a defect in the C6 vertebra in the Bethesda X-rays, which the Bethesda autopsy physicians had missed and did not note.
7. Even without this information, the original Bethesda autopsy report, included in the Warren Commission report, concluded that this bullet had passed entirely through the president's neck, from a level over the top of the scapula and lung (and the parietal pleura over the top of the lung), and through the lower throat.
8. Claims that anyone on the commission "moved the wound" are subject to discussion, because Gerald Ford publicly admitted to re-naming the location of the wound, so as "to make things clearer". The Bethesda autopsy had merely noted that JFK was hit in the upper thorax above the scapula (this is in the soft area at the top of the shoulder) and Ford changed this to "the base of the neck.
9. The Commission report, as amended by Ford, then found the bullet to have passed through the base of the neck, and not to have been in the back. However, Ford's change is consistent with a bullet hit in the shoulder at the C6 vertebral body, where the HSCA and the photograph placed the wound on the basis of X-damage of the vertebrae and tiny lead fragments in that location. The neck formally begins (and thorax ends) at the level of C7, the first cervical vertebral body above the thorax, and thus the original autopsy report is technically in error (Figs 20.11A to C).

The missile wound to the head

1. The wound to the back of the head is described by the Bethesda autopsy as being a laceration measuring 15 × 6 mm, situated to the right and slightly above the external occipital protuberance. In the underlying bone is a corresponding wound through the skull showing beveling (a cone-shaped widening) of the margins of the bone when viewed from the interior of the skull.
2. The large, irregularly shaped defect in the right side of the head (chiefly to the parietal bone, but also involving the temporal and occipital regions) is described as being about 13 cm (5 inches) wide at the largest diameter.
3. Three fragments of skull bone were received as separate specimens, roughly corresponding to the dimensions of the large defect. In the largest of the fragments is a portion of the perimeter of a roughly circular wound presumably of exit, exhibiting beveling of the exterior of the bone, and measuring about 2.5 to 3.0 cm in diameter. X-rays revealed minute particles of metal in the bone at this margin.
4. Minute fragments of the projectile were found by X-ray along a path from the rear wound to the parietal area defect.



Figures 20.11A to C: (A) Medical drawing of a cross-section of President Kennedy's neck and chest, showing the trajectory of the projectile from back to throat; (B) Photograph depicting the posterior head wound of President Kennedy; (C) Diagram showing the trajectory of the missile through President Kennedy's skull

Later Government Investigations[33,36]

Ramsey Clark Panel Analysis (1968)

At the request of The Honorable Ramsey Clark, Attorney General of the United States, four physicians (hereafter sometimes referred to as **The Panel**) met in Washington, DC and reviewed the original autopsy records, photos, and X-rays, as well as clothing, films, motion pictures, and bullet fragments. They also reviewed and discussed the Warren Commission report and presented the following conclusions:

Examination of the clothing and of the photographs and X- rays taken at autopsy reveal that President Kennedy was struck by two bullets fired from above and behind him, one of which traversed the base of the neck on the right side without striking bone and the other of which entered the skull from behind and exploded its right side. The photographs and X-rays discussed herein support the above-quoted portions of the original Autopsy Report and the above-quoted medical conclusions of the Warren Commission Report. Major differences with, and support of, conclusions in the Bethesda autopsy and Warren Report:

- The Clark report places the head bullet wound 100 mm (4 inches) above the reported occipital protuberance wound of the Bethesda report. This is important, because it is consistent with a high angle rear entry wound to the skull.
- The Clark report places the back wound squarely in the neck above the scapula and passing through the throat, passing over the TOP of the right lung, in keeping with the Bethesda conclusions. However, this finding is bolstered by additional findings of metallic fragments along the higher bullet trail.

Rockefeller Commission Analysis (1975)

The five-member Rockefeller Commission, investigation was limited to the movements of the President's body associated with the head wound that killed the President."

The Commission examined the films, the autopsy report, the autopsy photographs and X-rays, President Kennedy's clothing and back brace, the bullet and bullet fragments recovered, the 1968 Clark Panel report, and other materials. The five panel members came to the unanimous conclusion that President Kennedy was struck by only two bullets, both of which were fired from the rear, including one that struck the back of the head. Three of the physicians reported that the backward and leftward motion of the President's upper body following the head shot was caused by a "violent straightening and stiffening of the entire body as a result of a seizure-like neuromuscular reaction to major damage inflicted to nerve centres in the brain."

HSCA Analysis (1979)

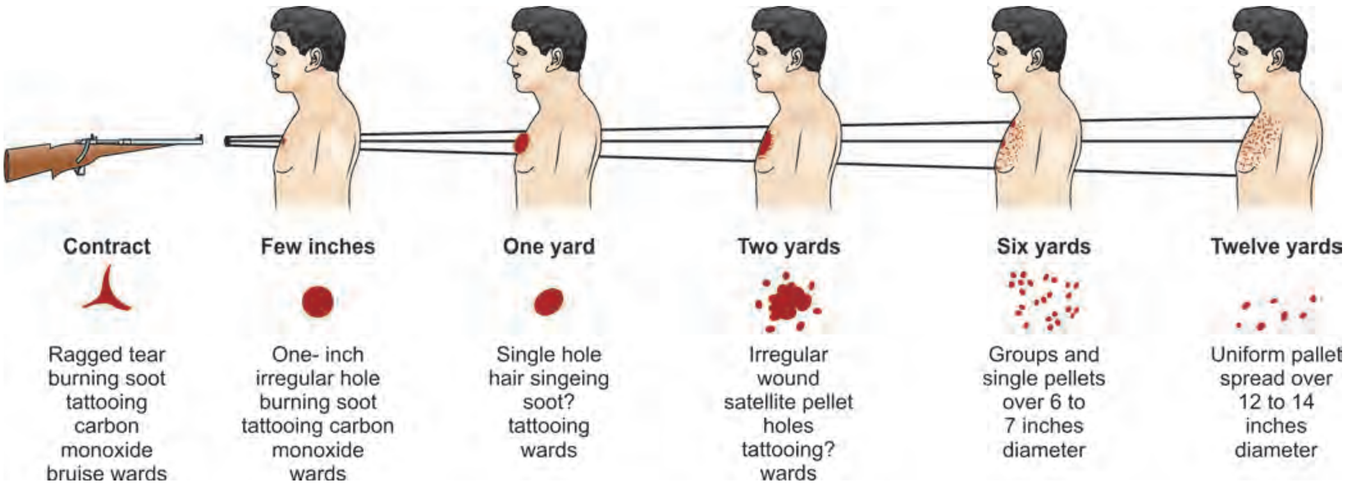
The **United States House of Representatives Select Committee on Assassinations** (HSCA) contained a forensic panel which undertook the unique task of reviewing original autopsy photographs and X-rays and interviewed autopsy personnel, as to their authenticity. The Panel and HCSA then went on to make medical conclusions based on this evidence. The HSCA's major medical-forensic conclusion was that *"President Kennedy was Struck by Two Rifle Shots Fired from Behind Him"*. Two questions were put to the experts in HSCA:

1. Could the photographs and X-rays stored in the National Archives be positively identified as being of President Kennedy?
2. Was there any evidence that any of these photographs or X-rays had been altered in any manner?

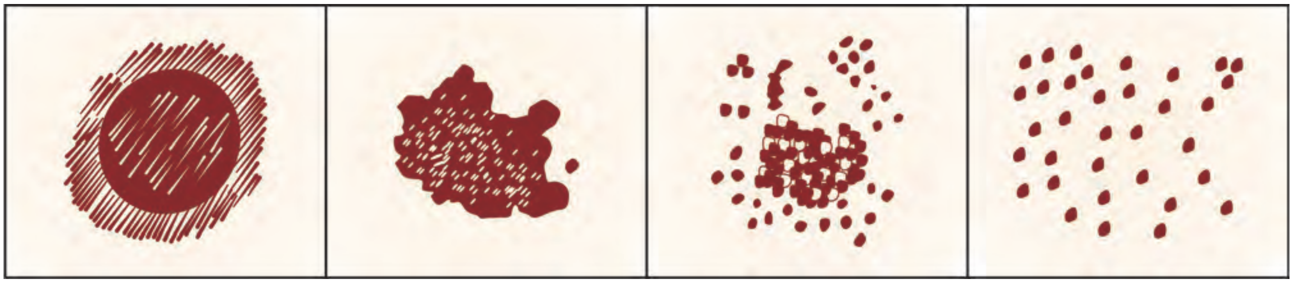
- To determine if the photographs of the autopsy subject were in fact of the President, forensic anthropologists compared the autopsy photographs with antemortem pictures of the President. This comparison was done on the basis of both metric and morphological features, and depicted as that from same person. The anthropologists studied the autopsy X-rays in conjunction with premortem X-rays of the President. A sufficient number of unique anatomic characteristics were present in X-rays taken before and after the President's death to conclude that the autopsy X-rays were of President Kennedy.
- This conclusion was consistent with the findings of a forensic dentist employed by the committee. Since many of the X-rays taken during the course of the autopsy included the President's teeth, it was possible to determine, using the President's dental records that the X-rays were of the President. Once the forensic dentist and anthropologists had determined that the autopsy photographs and X-rays were of the President, photographic scientists and radiologists examined the original autopsy photographs, negatives, transparencies, and X-rays for signs of alteration. They concluded there was no evidence of the photographic or radiographic materials

having been altered. Consequently, the committee determined that the autopsy X-rays and photographs were a valid basis for the conclusions of the committee's forensic pathology panel.

- *HSCA also concluded that President Kennedy was struck by two, and only two, bullets, each of which entered from the rear***.** The panel further concluded that the President was struck by *one bullet that entered in the upper right of the back and exited from the front of the throat, and one bullet that entered in the right rear of the head near the cowlick area and exited from the right side of the head, toward the front. This second bullet caused a massive wound to the President's head upon exit.*
- Because this conclusion appeared to be inconsistent with the backward motion of the President's head in the film, the committee consulted a wound ballistics expert to determine what relationship, if any, exists between the direction from which a bullet strikes the head and subsequent head movement. The expert concluded that nerve damage from a bullet entering the President's head could have caused his back muscles to tighten which, in turn, could have caused his head to move toward the rear. He demonstrated the phenomenon in a filmed experiment which involved the shooting of goats. However, the HCSA also voiced certain criticisms of the original Bethesda autopsy and handling of evidence from it. These included:
    1. The "entrance head wound location was incorrectly described."
    2. The autopsy report was "incomplete", prepared without reference to the photographs, and was "inaccurate" in a number of areas, including the entry in Kennedy's back.
    3. The "entrance and exit wounds on the back and front neck were *not localised with reference to fixed body landmarks and to each other*".
- These inconsistencies in the original autopsy report resulting in confusion and demanded further investigation and expert analysis of autopsy reports and autopsy material analysis further with discussion etc., though solved direction of firing, the cause of death of President JF Kennedy ultimately, things lead to the evolution of conceptual record in forensic literatures such as *Kennedy Phenomena, Single Bullet Theory* and *Souvenir bullet* for ever.


Contract
Ragged tear
burning soot
tattooing
carbon
monoxide
bruise wards
Few inches
One- inch
irregular hole
burning soot
tattooing carbon
monoxide
wards
One yard
Single hole
hair singeing
soot?
tattooing
wards
Two yards
Irregular
wound
satellite pellet
holes
tattooing?
wards
Six yards
Groups and
single pellets
over 6 to
7 inches
diameter
Twelve yards
Uniform pallet
spread over
12 to 14
inches
diameter

Figs 20.12: Characteristics of shotgun (smooth bore firearm) wounds at various ranges



Figs 20.13: Magnified view of shotgun wounds at 1 yard, 1-2 yards, 2-6 yards and more than 12 yards respectively (left to right)

wound of entry resembles that of firing done at a greater range (beyond 90 cm), e.g. a victim fired at close range, the shots being hit through a wooden partition or window panel, etc.[9,16]

Explanation – The shots/pellets which are bunched at a close range when strike the primary target (wooden partition intervening between firearm and victim) the leading shot gets slowed and hit by the after coming shots causing them to veer off or rebound in a wide pattern, just as the billiard balls do when hit with cue ball at the back, and produce wounds over a wider area on the secondary target (i.e., victim). This constitutes billiard ball ricochet effect (Fig. 20.14A). However, this effect

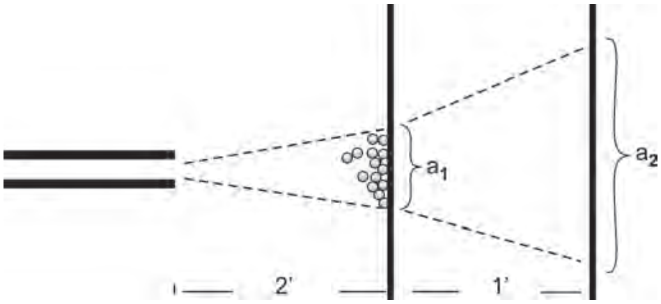


Fig. 20.14A: Billiard ball ricochet effect at close range of firing[16] (Note the bunched shots at primary target (a1)

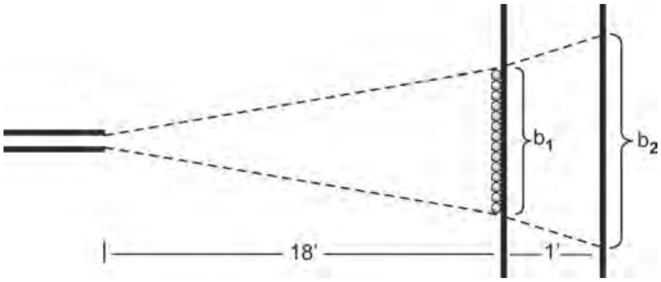
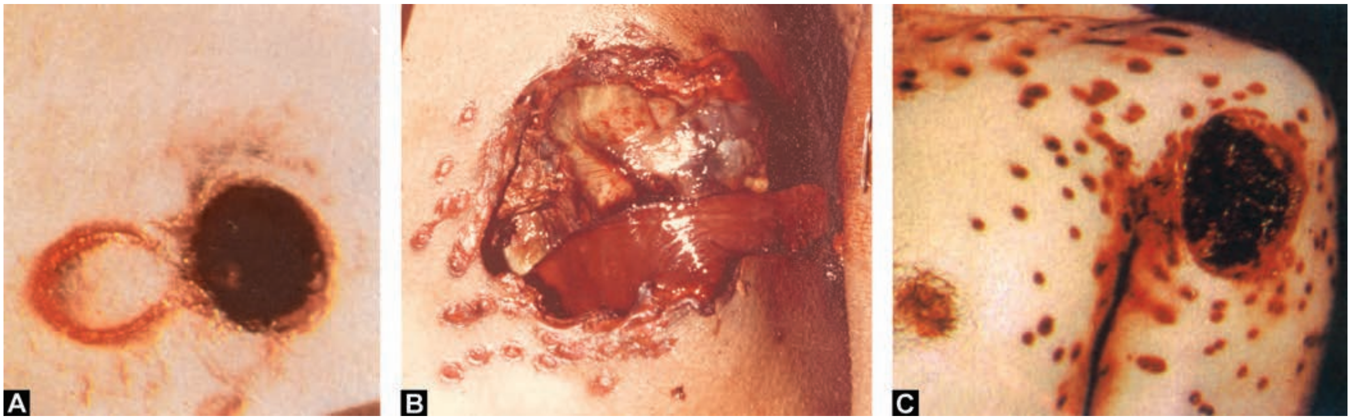


Fig. 20.14B: No Billiard ball ricochet effect as range of firing increases[16] (Note the spreadout shots at primary target (b1)



Figs 20.15A to C: Wound of entry by shotgun (smooth bore firearm) at an approximate range of firing: (A) Contact shot range; note the 'imprint'/'copy' or recoil abrasion of unused barrel, which is pinkish due to carbon monoxide in discharge gases, (B) Fire arm wound: Entrance wound of a shot gun at 2-3 m rage (*Courtesy:* Dr SC Mestri, Professor and HOD, Forensic Medicine, JSS Medical College, Mysore, Karnataka), (C) 4 m

will not be true as the range of firing increase for the simple reason that the shots are already spread widely (Fig. 20.13B).

Medicolegal importance—Difficulty in assessing the range of firing.

Estimation of Range of Firing by Examining Wound of Entry

A rough estimation of range of firing is always possible by examining the wound of entry (and the clothing worn at the time).

Principle—As the range of firing increases, the spread area of pellets also increases in a regular manner.

Thus, for a cylindrical barrel (non-choked) gun:[9,16]

For Ranges up to 3 m

Range multiplied by 3½ roughly gives the spread area diameter in cm. i.e.

Range (r) × 3½ = Spread area (A)
Range (r) = Spread area (A)/3½
= A × 2/7 m

For Ranges beyond 3 m

Roughly the spread area is 25 cm per 1 m range.
Thus

- For a Spread Area is 25 cm = Range of firing will be 1 m
- For a Spread Area is 50 cm = Range of firing will be 2 m
- For a Spread Area is 75 cm = Range of firing will be 3 m, etc.

Exit wound — It occurs only when the shot passes right through and out of the body; the wound will show features like:[9,12-16]

- Margins everted.
- No soiling, singeing, etc.
- May be multiple as each pellet or groups of pellets might pass out independently.

  Usually shotgun pellets do not exit from the body, except:

a. Contact shot wound
b. Tangential wound where some of the pellets have a very short track through the body and
c. Thin parts of the body like neck, extremities etc.[16,20]

AUTOPSY EXAMINATION OF CASES OF FIREARM FATALITIES

External autopsy examination — comprises of two parts:

- Examination of clothing
- Examination of the wounds.

Examination of Clothing

Purpose of examination of clothing is to establish range of firing, whether it is wound of entry or exit and also sometimes to locate the bullet.[9-16,20]

Procedure — Clothing is to be removed layer by layer. List all layers and note their condition, any stains, holes, etc. in each item. Record the number and location of bullet holes. Assign a number to each one and describe them in relation to distance from collar, pockets, etc. Due to creases in clothing, a single bullet can produce more than a single hole. Preferably with a magnifying hand lens try to find out if the fibers of clothes are turned inwards or outwards.[9,16,20]

Further examinations by FSL — The clothes collected and examined as above are then dried up in the shade. They are then preserved carefully in clean brown paper envelopes, bags, etc. and sent to forensic science laboratory (FSL) for further examination, to detect the presence of blood, any other biological fluid stains and gunpowder residues.[12-16,40-41]

Photographing the clothes — Clothes with bullet holes or tracks should be photographed with a scale placed nearby. Infrared photography may be useful in detecting the soot deposits on dark or black coloured garments.[20]

Examination of the Wounds

Bullet wounds must be described with great care for both wound of entry and wound of exit. The various steps include, wound location, and wound description. There may be requirement for excision and preservation of external wound for sending to FSL.[9,16,20]

Wound location — This is done for both wound of entry as well as wound of exit in relation to top of head, the unshod heel, body midline and certain fixed anatomical landmarks.

Wound description — Describe both entry and exit wounds noting the site, size, shape and other details (vide supra).

Excise the wound for further microscopic examination — cut the wound with 2.5 cm. healthy skin around it and a minimum of 5 mm. thickness beneath. This portion of tissue is then put in rectified spirit, labeled and sent to FSL[9,16,20]

Internal Autopsy Examination

Apart from routine examination internally the most important thing comprises of *study track taken by bullet*[9,16,20]—Best method is by taking a radiograph. However, method of study by insertion of a probe is though used restrictedly, a better procedure is by dissection. Box 20.1 highlights autopsy examination of case of fire arm fatality in the form of a checklist briefly.

1. *Role of radiography/X-rays in gunshot wounds:*[49-53] In gunshot wound cases X-rays should "always" be performed to answer following questions:
   - Is the projectile present?
   - If present, where is the projectile located?
   - If the projectile exited, are projectile fragments present and where are they located?
   - What type of ammunition or weapon was used?
   - What was the path of the projectile?

   Problems to be aware of:
   - The last few inches of a.[22] rimfire ammunition wound track may have no associated hemorrhage.
   - In a partial exit wound and occasionally in a "shored" exit wound the projectile is in the body despite the presence of an exit wound.
   - With partial metal jacketed bullets the lead core may exit leaving the jacket (with its ballistically important rifling marks) in the body. Sometimes the opposite occurs. Alternatively the jacket and core may separate in the body and neither exit. Projectiles may be retained in clothing.
   - Components of some projectiles are poorly radio-opaque on routine X-ray, e.g. Aluminium jacket, plastic tip, plastic shotshell wadding.
   - Projectiles may ***embolise***.
   - Projectiles may ricochet within the body off bones, most commonly the inner table of the skull.
   - The spread of shotgun pellets in the body seen on X-ray cannot be used to determine range of fire because of the billiard-ball effect.
   - In contact shotgun wounds to the head, all but a few pellets may exit.
   - Exact projectile calibre cannot be determined due to X-ray magnification effects.

   Diagnostic tips:
   - ***"Lead snowstorm"*** characterizes high velocity centrefire rifle ammunition. Ruled out are full metal-jacketed bullets and lead slugs.
   - C-shaped or comma-shaped subgaleal lead fragment in head wounds characterises[32] or occasionally[38] calibre the revolver bullets.
   - A "pancake" or "disk" with 2 to 4 associated comma-like fragments (no "lead snowstorm") characterises the ***Foster shotgun slug***.
   - The presence of the base screw characterises the ***Brenneke shotgun slug***.
2. Removal, marking and preservation of the bullet — A bullet may be removed at surgery or autopsy, is always removed by a pair of forceps with rubber tips and then dried in shade and preserved in a cardboard box. A mark (initials) of the doctor collecting it may be done at the base or tip of the bullet but never on the body. This is to avoid any damage to rifle markings.
3. If the bullet is lodged in the bone — Cut the bone segment where the bullet may be lodged.
4. ***Kronlein shot***—This is a condition wherein a large portion of brain is thrown out (herniated) of the exit wound but still intact/attached to the wound.[9,16]

GUNPOWDER RESIDUES TESTS

There are specific types of tests which could indicate whether the person examined was contaminated with "***blowback***" residues of gun powder, from having recently fired a weapon.[9,16,20,43-47] Such residues are typically removed from the subject by swabbing the back of the index finger, thumb and the web spaces/areas of the hand with a moistened cotton swab containing a solution of five per cent nitric acid.

Limitations: However, the value of such an examination is questionable since barium and antimony are also found in nature, as well as in a variety of common products, and it possible that the subject being examined might have come in contact with such sources other than firing a gun. Thus most of these tests are inconclusive and are with ambiguity demands the need of additional forensic evidences in confirming the suspect's guilt.[43-47] Some of the routinely performed gun powder residue tests are enumerated and discussed.

- Dermal nitrate test
- Harrison and gilroy test
- Neutron activation analysis (NAA)
- Image analysis of gunshot residue (IA GSR)
- Sodium rhodizonate test

Dermal nitrate test—It is a test for gunpowder particles on the hands of the assailant.

Box 20.1: Checklist for autopsy in firearm deaths

1. Take an X-ray of the deceased prior to removing the clothing.
2. Recover primer residues from hands by acid (10% nitric) moistened swab or adhesive tape.
3. Examine hands for trace evidence, soot and propellant grains, and blood splatter.
4. Examine and remove the clothing without cutting. Use dissecting microscope to examine clothing defects and wounds for soot and propellant.
5. Examine the body, photograph the wounds if appropriate, and correlate with clothing. Take repeat photographs of the body after cleaning the body and describe the wounds. Note following while describing the Wounds:
   - Describe each wound completely, i.e. including the internal wound track revealed by dissection.
   - Describe wound location relative to (a) body landmarks, (b) body midline and heel or top of the head.
   - Describe the wound appearance by size, shape, abrasion collar (width and symmetry), soot and propellant (its presence, distribution and dimensions) and wound entry scorching/searing. Incase of shotgun/smooth bored firearm wound describe shotgun pellet pattern.
   - Describe muzzle end imprint and compare with alleged weapon if available at crime scene/when produced later.
   - Describe the lodged projectile or exit relative to entrance; describe the general direction of wound track.
   - Describe any recovered projectile or fragments. Trace the wound tracks and recover the projectiles. Complete the dissection.
6. Try to recover/collect:
   - Propellant grains from skin surface or wound track.
   - Projectile, taking care not to scratch the surface metal instrumentation such as a toothed forceps while collecting. Use a rubber tipped forceps/gloved fingers in collection of bullets
   - Sample shotgun pellets and all wadding if any.
   - Blood for grouping and blood and tissue for toxicology analysis.

Procedure—Mop the hands of suspected person with gauze soaked in molten paraffin, cool to harden. Then treat its inner surface with diphenyle amine and if it gives blue colour, the test is positive.

Fallacies—Test is positive with any nitrogenous substance, like urine, tobacco, etc., on hand.

Harrison and Gilroy test—This is a test for certain elements or compounds such as antimony, barium, lead, etc. found in firearm discharge residue.

Neutron activation analysis (NAA)—This is a test used for estimation of distance of firing and confirming the hands of those suspected of firing a firearm. The test can detect barium, copper, antimony, etc. from the primer present in the firearm discharge residue, which are activated in a nuclear reactor and then identified by the gamma ray spectrometer.[9,16,46]

Image analysis of gunshot residue on entry wounds—Newer technique and preliminary study have been reported in literature by image analysis of gunshot residues (GSR). In this method an automated image analysis (IA) technique has been developed to obtain a measure of the amount (i.e. number and area) of gunshot residue (GSR) particles within and around a gunshot wound. Sample preparation and IA procedures were standardised to improve the reproducibility of the IA measurements of GSR. However, preliminary results indicated that the decreasing relationship between firing range and the amount of GSR deposited are non-linear, and that for firing ranges of up to 20 cm the amount of GSR deposited from repeated shots fired from the same distance are highly variable.[47]

Sodium rhodizonate test—This test is performed to detect the particulate lead deposited on surfaces as a consequence of a firearm discharge. This has been directed at addressing some of the problems that have hitherto compromised the value of this test to forensic science. In particular, the aqueous solutions of sodium rhodizonate are considerably more stable when stored below pH 3. The rhodizonate dianion is then diprotonated, forming rhodizonic acid, and the half-life of the solution increases from about one hour to about ten hours. By ensuring that the area to be examined is pretreated with tartrate buffer so that its pH is adjusted to 2.8 prior to treatment with rhodizonic acid, the formation of a nondiagnostic purple complex, instead of the desired scarlet complex, is avoided. Whereas the scarlet complex changes to a blue-violet complex, upon secondary treatment with 5 per cent HCl, which is diagnostic of the presence of lead, the purple complex decolourises completely under these conditions and thus its formation represents wastage of lead from within the test area and is associated with the fading problem that has previously plagued the test. The fading of the blue-violet complex can be eliminated by removing excess HCl, by means of a hair drier once the colour has fully developed.[48]

MEDICOLEGAL QUESTIONS ON FIREARM INJURIES

In a firearm injury death case, basically certain questions need to be answered for medicolegal purposes. Certain important questions are discussed here.[8,9,11-13,16]

Kinds of the Firearms

A proper examination of wound of entry and knowledge about the classification of firearms (vide supra) can help in deciding the type of firearm used.

Range of Firing

A proper description of the wound of entry can help in deciding the range of firing for both gunshot and shotgun wounds (vide supra).

Direction of Firing

It can be decided ideally by radiographic examination (vide supra). However, probing the wound is though allowed, method of autopsy dissection procedure is more reliable.

Cause of Death

It is usually due to the vital organ injury in the path taken by bullet.

Accident, Suicide or Homicide?

Death due to injuries from firearms is an important public health problem. As a group, injuries from firearms were the ninth leading cause of death in USA with total number of firearm deaths increased by 130 per cent, from 16,720 in 1962 to 38,505 in 1994. If these trends continue, firearm-related injuries could become the leading cause of deaths, surpassing injuries due to motor vehicle crashes. As reported, the patterns of overall firearm-related mortality are due to homicide, suicide, unintentional death, deaths occurring during legal intervention, and deaths of undetermined origin. Suicide and homicide are usually responsible for most firearm fatalities; and are accounted for 94 per cent of the total deaths in 1994 in USA.[54-58] The fluctuations and overall increase in rates of total firearm-related mortality most closely resembled the pattern of firearm-related homicide. Although suicide rates were high and gradually increasing over time, they varied less than homicide rates. The rates for unintentional death from firearms, deaths during legal intervention, and deaths of undetermined intent are low and generally declined over a period of time recently. Firearm-related mortality affects all demographic groups, but the greatest increases in recent years were among teens 15-19 years of age, young adults aged 20-24, and older adults aged 75 and older. The rates of overall firearm-related mortality for young people aged 15-24 were higher from 1990-1994 than at any other time. For those 15-19, increases in firearm-related homicide, suicide, and unintentional injury deaths were especially great. The increase in firearm-related homicide in this age group occurred among all race-sex groups. For America's elderly, rates of suicide by firearm were particularly high, and increases occurred in all race-sex groups except black females, for whom number of suicides were too small to produce stable rates.[59-63] While these data help characterize the magnitude of the problem and identify groups at risk, there are still gaps in our knowledge.

In India, the rampant proliferation of illicit small arms combined with poor policing, has eroded human rights, weakened democratic institutions and polarised ethnic, religious, economic and political differences between citizens.[64-69] It is difficult for enforcement agencies to keep a check on violence when during elections private armies of politicians carrying illicit firearms roam at large.[68] The problem of intimidation by such criminal elements is compounded by the fact that legal firearm ownership is so limited – making it impossible for a private citizen to effectively defend himself/ herself. The statistics related to suicides and firearm accidents in India for the year 1990-1994[64] is presented in Table 20.6.

Certain facts helpful in deciding whether a particular firearm wounds is of accidental/ suicidal and/homicidal origin needs to be discussed at this stage:[8-16]

Accidental—Wound could be found anywhere on the victim's body.

Suicidal—Wound seen is often singular and noticed on the most vital part of the body, which is usually easily accessible to the victim himself/herself, e.g. temple, left side of chest—precordium, neck (Fig. 20.15D) etc. Also range of firing in a suicidal death is usually at contact shot range, and the victim may leave a suicide note nearby. Weapon used to fire may be held firmly in the hand by the victim, gripped tightly in a cadaveric spasm. It is also observed that a left handed victim will make a firing on the left side of the head. Points enumerated below are observations in suicidal firing deaths as recorded from literature:[9, 12-19, 60-61]

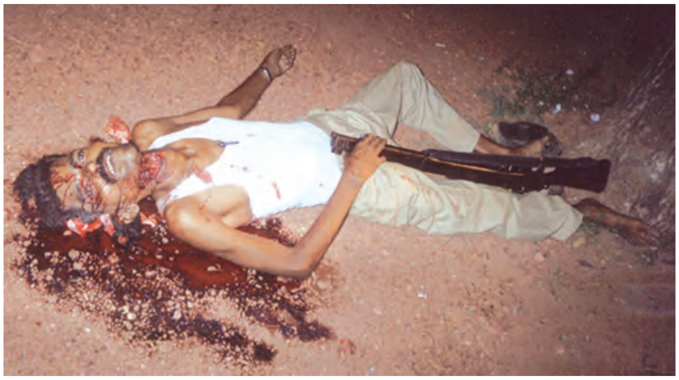


Fig. 20.15D: *Suicide by rifle:* Note the single disruptive wound in the accessible part neck and gripping the muzzle end of the rifle. The victim is a police constable, by profession and alleged to have committed suicide in a sitting posture holding the *butt* of the rifle firmly against the tree trunk and muzzle end under the chin over front of his neck and operated the trigger by the toes of the right foot (*Courtesy:* Dr Mahabalesh Shetty, Assoc. Professor and Head, Dept of Forensic Medicine, KSHEMA, Derla Katte, Mangalore and Dr Suresh Kumar B Shetty, Assistant Professor, KMC, Mangalore)

- The majority of suicides (including gunshot suicides) do not leave any suicide note.
- A contact wound creates a presumption of suicide rather than accident.
- With rifle and shotgun wounds to the trunk the trajectory may corroborate suicide. With the weapon butt on the ground and the body hunched over it the trajectory is downwards (not upwards). Reaching for the trigger with the right hand rotates the body so that the trajectory is right to left side (vice versa if reaching with the left hand).
- Suicide handgun wounds occur primarily in the head (80%), the chest (15%) and abdomen (<5%) being less common. Within the head the common sites, in decreasing order of frequency, are temple, mouth, undersurface of chin and forehead. An unusual location raises a presumption of homicide.
- Suicidal shotgun wounds show the same site preference as handguns. Rifle wounds show a distribution of head 50 per cent, chest 35 per cent, and abdomen 15 per cent.

Table 20.6: Suicides and firearm accidents in India, 1990-1994[64]

	1990	*1991*	*1992*	*1993*	*1994*
Total suicides	73,911	78,450	80,149	84,244	89,195
Firearm suicides	535	571	561	712	586
Firearm suicide rate					0.06
Firearm accidents	2,731	3,169	3,341	1,980	2,375
Firearm accident rate					0.26
Homicidal	*	*	*	*	*

* India reported its homicide statistics as "not reasonably available"

- Suicide by multiple gunshots is uncommon but not rare.
- A suicide victim may "test fire" the weapon before inflicting the fatal shot.
- In about 20 per cent of suicides the weapon is found clutched in the hand. Rarely an orange-brown discolouration of the palm due to iron deposition from the barrel may be seen; the stain cannot be wiped away.
- Occasionally high velocity impact blood spatter will be deposited on the back of the hand steadying the muzzle and the back of the firing hand.
- The hand holding the muzzle may show soot deposition on the radial margin of the forefinger and the adjacent surface of the thumb and the radial half of the palm due to muzzle blast. With revolvers the cylinder blast may cause soot deposition on the ulnar half of the palm.
- Contact wounds to cotton or cotton mixture cloth from medium and large calibre weapons typically produces cruciform tearings; with synthetic materials there are burn holes with scalloped melted margins. Tearing is less prominent with the smaller amounts of gas from.[22] rimfire ammunition. A grey to black rim of "bullet wipe" may be present around the entrance hole.

Homicidal — Wound is usually noticed on the vital parts of the body, which are non- accessible for the victim himself/herself. The range of firing could be of any distance.

Fake Firearm Wounds

Firearm wounds have been inflicted either maliciously or to avoid conscription or for a variety of other reasons not least for implicating others in a crime. These are usually seen on non-vital parts of the body, producing minimum damage to the patient; the motive behind such injuries may have to be unraveled with the assistance of clinical psychologists and psychiatrists. Scotland Yard spokesman said 70 per cent of firearms offences investigated on gun crime amongst London's black communities, involved replica guns which had been converted to fire live ammunition resulting in fatalities. Chris Summers BBC Online News Report 'Killed by Fake Gun' describes one such case which was resolved on confession finally made by the criminal who said that death was unintentional and accidental.[70]

Position of Weapon

Referring to what was discussed above following can be reiterated especially on position of the weapon in cases of firearm fatalities:[9,14,16,20]

- In suicides usually the weapon may be still held firmly in the victim's hand (cadaveric spasm). There are also sites of election for suicidal (sunscald?) wounds, which are mouth, head, and front of the chest. It is still unusual for a woman to commit suicide with a firearm.
- In accidental cases, weapon is found at the scene of crime.
- However, no weapon is detected at the scene of crime if it is a case of homicide.
- It is essential also to be able to assess how the person might have had the access to the gun and to the ammunition for it. In case of long barreled weapons it is essential to discover whether or not the weapon could have been fired by the deceased and measurement of arm span etc is essential.

How Long Victim Survived after Injury?

Bullet wounds of vital parts like brain, heart, etc., can kill the victim instantaneously. However, in gunshot injuries to other parts of the body, victims can live for sufficiently long enough and even may remain ambulant and may perform important acts/reach hospital on their own and then die (volitional activities).[9,16]

Medicolegal Importance of Bullet

A bullet could be crime bullet, test bullet and exhibit bullet.[9-12,71]

Crime Bullet — Crime bullet is a bullet collected at surgery or autopsy, from the body of a victim alive or dead.

Test Bullet — Test bullet is a bullet that is fired from a weapon suspected as being involved in a crime; the test firing is into a gunny bag fixed into a deal wood box. The bullet is then collected for comparison with crime bullet under a comparison microscope, for identifying the crime weapon. This can only be done in relation to rifled weapons.

Exhibit Bullet — Exhibit bullet is a crime/test bullet, which is presented in the court as evidence.

CRIME SCENE INSTRUCTIONS

1. Handle the body as little as possible thus avoiding artifacts and loss of trace evidence.
2. The hands of the victim may be covered inside a paper bag in order to preserve trace evidence.
3. Transport the body in clear plastic sheeting or a body bag to preserve trace evidence and avoid contamination.

EXPLOSIVES AND EXPLOSIVE INJURIES

(*Synonyms:* Explosion injury, explosion, bombing, terrorism, firework injury, fire work injury, industrial fuel eruption, mine explosion, land mine, hand grenade, blast injury, blast injuries, explosive material, radiological contamination, biological contamination, terrorist attacks, primary blast injury, secondary blast injury, tertiary blast injury, blast-related injuries).

Ever since the increase in terrorist attacks, disaster response personnel have to be aware of the pathophysiology of these injuries associated with explosions and be prepared to assess and treat the victims injured. Bombs and explosions can cause unique patterns of injury. Penetrating injuries and blunt trauma predominate among survivors following an explosion. Blast lung is the most common fatal injury among initial survivors. Explosions in confined spaces such as mines, buildings, large vehicles etc. are associated with higher morbidity and mortality.

Explosions have the potential to inflict multi-system, life-threatening injuries on many persons simultaneously. The injury patterns following such events are a product of the composition and amount of materials involved, the surrounding environment, delivery method (if a bomb), the distance between the victim and the blast, and any intervening protective barriers or environmental hazards. Because explosions are relatively infrequent, blast-related injuries can present unique diagnostic and management challenges in administration of emergency care.[72-74]

TAXONOMY OF EXPLOSIVES

Explosives are classified as high-order explosives (HE) or low-order explosives (LE).[74]

High-order explosives produce a defining supersonic over-pressurisation shock wave. Examples of HE include TNT, C-4, Semtex, nitroglycerin, dynamite, and ammonium nitrate fuel oil (ANFO).

Low-order explosives create a subsonic explosion and that does not produce over-pressurisation. Examples of LE include pipe bombs, gunpowder, and most purely petroleum-based bombs such as *Molotov cocktails* or aircraft improvised as guided missiles. High-order and low-order explosives cause different injury patterns.

Explosive and incendiary (fire) bombs are further characterised based on their source.

Manufactured implies standard military-issued, mass-produced, and quality-tested weapons. Improvised describes weapons produced in small quantities, or the use of a device outside its intended purpose, such as converting a commercial aircraft into a guided missile.

Manufactured (military) explosive weapons are exclusively HE-based. Terrorists will use whatever is available-illegally obtained manufactured weapons or improvised explosive devices (IEDs) that may be composed of HE, LE, or both. Manufactured and improvised bombs cause markedly different injuries.

BLAST INJURIES

Mechanisms of Injuries

The four basic mechanisms of blast injury are termed as primary; secondary, tertiary, and quaternary (Table 20.7). An example that causes primary injury is blast wave, which refers to the intense over-pressurisation impulse created by a detonated HE. Blast injuries are characterised by anatomic and physiologic changes that occur when the direct or reflective over-pressurisation force impacts the body's surface. The HE blast wave (over-pressure component) should be distinguished from blast wind (forced superheated air flow). Blast wind may be encountered with both HE and LE.

Low-order explosives are classified differently because they lack the defining over-pressurisation wave of HEs. Low-order explosives cause injury from ballistics (fragmentation), blast wind (not blast wave), and thermal. There is some overlap between LE descriptive mechanisms and HE's secondary, tertiary, and quaternary mechanisms.

Up to 10 per cent of all blast survivors have significant eye injuries, generally due to perforations from high-velocity projectiles. They can occur with minimal initial discomfort, and present for care days, weeks, or months after the event. Symptoms include eye pain or irritation, foreign body sensation, altered vision, periorbital swelling, or contusions. Findings can include decreased visual acuity, hyphaema, globe perforation, subconjunctival haemorrhage, foreign body, or lid lacerations. Liberal referral for ophthalmologic screening is encouraged.

The extent and pattern of injuries produced by an explosion are a direct result of several factors such as:

- Amount and composition of the explosive material (e.g. the presence of shrapnel or loose material that can be propelled, radiological or biological contamination),
- Surrounding environment (e.g. the presence of intervening protective barriers),
- Distance between the victim and the blast,
- Delivery method if a bomb is involved,
- Other environmental hazards.

A bomb is a solid container filled with an explosive mixture (2-10 kg maximum as in terrorist bomb), sometimes also other missiles (nails, bolts, bits of metal) and a detonator or fuse device for the purpose of initiating the explosion. When a bomb explodes, a tremendous amount of energy is released in the shape of heat, kinetic energy and a very forceful air blast, which in turn can lead to injuries to the victims in the vicinity of explosion (Figs 20.16, and 20.17A to C). Furthermore the container itself will split and fragment into shrapnel, any inclusions in the bomb will do likewise and objects broken and fragmented by the blast will also act as secondary missiles, which will still have a very large momentum and cause further damage to persons and property.

No two events of blast are identical, and the spectrum and extent of injuries produced varies widely. Between 1991 and 2000, of the terrorist attacks worldwide, 885 were involving explosions.[72] The 2005 London subway bombings, the 1995 bombing of the Murrah Federal Building in Oklahoma City, and

Table 20.7: Mechanisms of blast injury

Categories	*Characteristics*	*Body parts*	*Affected types of injuries*
Primary	Unique to high-order explosives; results from the impact of the over pressurisation wave with body surfaces	Gas-filled structures are most susceptible: lungs, GI tract, middle ear	Blast lung (pulmonary barotrauma) Tympanic membrane rupture and middle-ear damage Abdominal hemorrhage and perforation Globe (eye) rupture Concussion (traumatic brain injury without physical signs of head injury)
Secondary	Results from flying debris and bomb fragments	Any body part	Penetrating ballistic (fragmentation) or blunt injuries Eye penetration(can be occult)
Tertiary	Results when bodies are thrown by blast wind	Any body part	Fracture and traumatic amputation Closed and open brain injury
Quaternary	All explosion-related injuries, illnesses, or diseases not due to primary, secondary, or tertiary mechanisms; includes exacerbation or complications of existing conditions	Any body part	Burns (flash, partial and full-thickness) Crush injuries Closed and open brain injury Asthma, COPD, or other breathing problems from dust, smoke, or toxic fumes Angina, hyperglycemia, hypertension

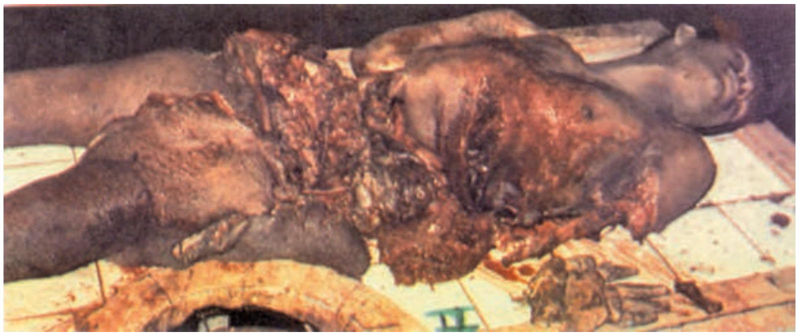


Fig. 20.16: Victim of blast injury death due to the blast of the bomb he was carrying in his hand bag (*Courtesy:* Dr Thyagarjan Formerly Professor and HOD Forensic Medicine, Govt Medical College, Coimbatore)

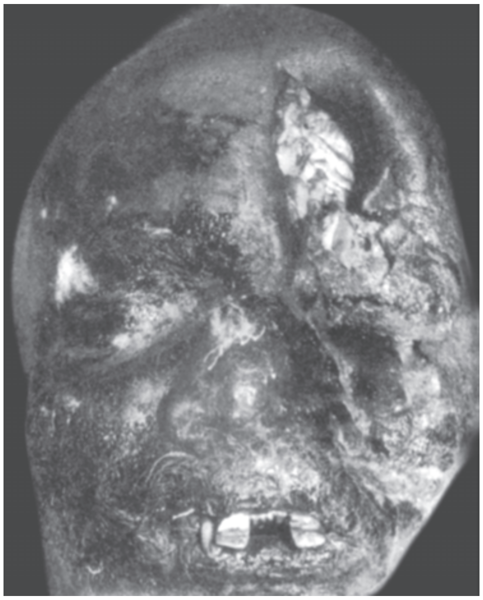


Fig. 20.17A: Victim of blast injury death. Blast injury of face

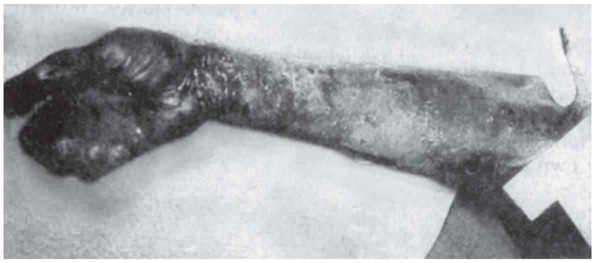


Fig. 20.17B: Victim of blast injury death. Blackening of palmar aspect of hand

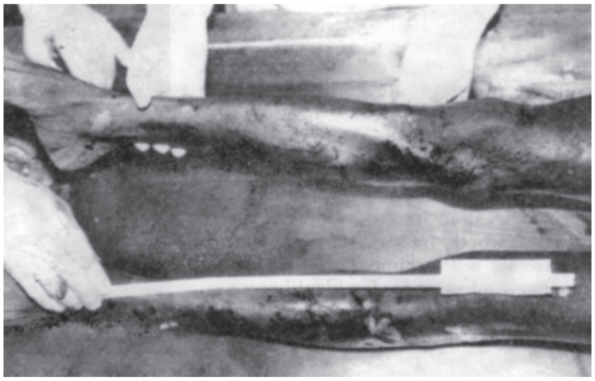


Fig. 20.17C: Victim of blast injury death. Legs with multiple lacerations

the catastrophic explosions of aircraft into 3 buildings on September 11, 2001 in New York City and Washington DC, all keeps us reminding the magnitude of the injuries and deaths resulted from blast mechanism.[73] Added to these deliberately-set explosions, the industrial accidents resulting in explosions such as in mining operations and fuel transportation and storage, situations worsen the outcome further.

In several parts of the world, military incendiary devices such as land mines and hand grenades contaminate the sites of abandoned battlefields. Such devices can cause significant numbers of civilian casualties. During wartime, injuries arising from such explosions usually outnumber those from gunshots victimising many innocent civilians.

Types of Blast Injuries

Blast injuries traditionally are divided into 4 categories: primary, secondary, tertiary, and miscellaneous injuries.[72] A patient may be injured by more than one of these mechanisms.

- A primary blast injury — is caused solely by the direct effect of blast overpressure on tissue. Air is easily compressible, unlike water. As a result, a primary blast injury almost always affects air-filled structures such as the lung, ear, and gastrointestinal (GI) tract.
- A secondary blast injury — is caused by flying objects that strike people.
- A tertiary blast injury — is a feature of high-energy explosions. This type of injury occurs when the victim is flown/blown through the air and striken other objects.
- Miscellaneous blast-related injuries — encompass all other injuries caused by explosions. For example, the collision of two jet airplanes into the World Trade Centre created a relatively low-order pressure wave, but the resulting fire and building collapse killed thousands.

OTHER INJURIES (TABLE 20.8)

Ear Injury

Primary blast injuries of the auditory system are easily overlooked but they can cause significant morbidity. Injury is dependent on the orientation of the ear to the blast. Tympanic membrane (TM) perforation is the most common injury to the middle ear.

Table 20.8: Overview of explosive-related injuries

Systems	*Injury or conditions*
Auditory	Tympanic membrane (TM) rupture, ossicular disruption, cochlear damage, foreign body
Eye, orbit, face	Perforated globe, foreign body, air embolism, fractures
Respiratory	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, A-V fistulas (source of air embolism), airway epithelial damage, aspiration pneumonitis, sepsis
Digestive	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Circulatory	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, peripheral vascular injury, air embolism-induced injury
CNS injury	Concussion, closed and open brain injury, stroke, spinal cord injury, air embolism-induced injury
Renal injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Extremity injury	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, cuts, lacerations, acute arterial occlusion, air embolism-induced injury

Signs of ear injury are usually present at time of initial evaluation and should be suspected for anyone presenting with hearing loss, tinnitus, otalgia, vertigo, bleeding from the external canal, TM rupture, or mucopurulent otorrhea. All patients exposed to blast should have an otologic assessment and audiometry.[75]

Abdominal Injury

Gas-containing sections of the gastrointestinal tract are most vulnerable to primary blast effect. This can cause immediate bowel perforation, hemorrhage (ranging from small petechiae to large haematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture. Blast abdominal injury should be suspected in anyone exposed to an explosion who has abdominal pain, nausea, vomiting, haematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolaemia, or any findings suggestive of an acute abdomen. Clinical findings may be absent until the onset of complications.

Brain Injury

Primary blast waves can cause concussions or mild traumatic brain injury (TBI) without a direct blow to the head. Consider the proximity of the victim to the blast, particularly when the patient complains of headache, fatigue, poor concentration, lethargy, depression, anxiety, insomnia, or other constitutional symptoms. The symptoms of concussion and post-traumatic stress disorder can be similar.

The signs and symptoms of a TBI can be subtle. Symptoms of a TBI may not appear until days or weeks following the injury or may even be missed when patients appear fine, even though they may act or feel differently.[78]

Diagnosing a TBI is challenging because symptoms are often common to other medical conditions and the severity of the symptoms can change over time. Any patient may have a TBI who has a history of head trauma or who is suffering from confusion, disorientation, amnesia of events around the time of injury, loss of consciousness of 30 minutes or less, neurologic or neuropsychological problems, or who has a Glasgow Coma Scale (GCS) score of 13 or higher. Taking a careful medical history can be key for detecting a TBI. Any unusual or unexplained signs or symptoms should be evaluated further.[78]

All blast injuries can be further of different types and they are: Burns injuries, disruptive injuries, air blast hurl injuries, flying missile injuries, injuries due to falling masonry, effects due to toxic gases in a bomb blast and Distance blast injuries.

Burns Injuries

Burn injuries are due to the temperature of the explosive gases (may exceed even 2000°C), which can cause flash burns (Fig. 20.18).

Disruptive Injuries

Victims who are very close to the exploding bomb or if they are in the process of carrying the bomb on their person or in a car, they may be shattered into several pieces and these pieces may be scattered over an area of 200 m. This is also true for so-called suicide bombers who have explosive on their persons.

Air Blast Hurl Injuries

Air blast can hurl the victims against walls or toss them through the air in various directions thereby causing them to sustain very severe blunt force injuries.

Flying Missile Injuries

Flying missile injuries occur due to numerous missiles within the bomb or pieces of fragments of objects nearby, e.g. gravel, glass, mound, brick, etc.

Injuries due to Falling Masonry

It is usual that due to a blast, building or heavy structures around the blast may also get wrecked and heavy objects such as beams, walls, roof, etc. may collapse and land on the victims and may individually contribute to several mechanical injuries.

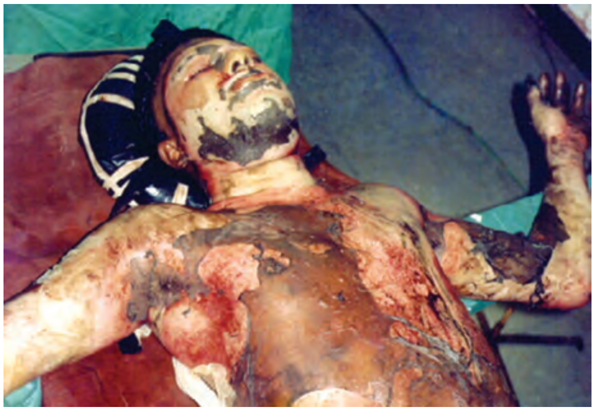


Fig. 20.18: Burns following bomb explosion (*Courtesy:* Dr G Goonetilleke, Consultant Surgeon, Sri Jayewardenepura General Hospital, Nugegoda, Sri Lanka)

Effects due to Toxic Gases in a Bomb Blast

Effects due to toxic gases in a bomb blast — toxic gases like CO, CO_2, etc. in a bomb blast can also kill the victim when inhaled.

Distance Blast Injuries

These occur at solid/ fluid or air/fluid interphones with disruptions at these points and are particularly prominent in the ears, in the lungs and in the intestines.[75]

Mortality/Morbidity

- Analysis of 29 large terrorist bombing events during 1966 and 2002 showed 8,364 casualties, including 903 immediate deaths and 7,461 immediately surviving injured. Immediate death/injury rates were higher for bombings involving structural collapse (25%) than for confined space (8%) and open air detonations (4%).[72,73]
- Unique patterns of injury are found in all bombing types. Injury is caused both by direct blast overpressure (primary blast injury) and by a variety of associated factors. Enclosed-space explosions, including those occurring in busses, and in-water explosions produce more primary blast injury. Explosions leading to structure collapse produce more orthopedic injuries. Land mine injuries are associated with a high risk of below — and above the knee amputations. Fireworks-related injuries prompt estimated 10,000-12,000 casualties in the United States annually, with 20-25 per cent involving either the eye or hand.[72]
- Tympanic membrane (TM) rupture indicates that a high-pressure wave (at least 40 kilopascal [kPa]/ (6 psi) was present and may correlate with more dangerous organ injury. Theoretically, pressure of > 100 kPa (15 psi) is threshold for lung injury, and tympanic membrane (TM) to rupture routinely; however, a recent Israeli case series of 640 civilian victims of terrorist bombings contradicts traditional beliefs about a clear correlation between the presence of TM injury and coincidental organ damage. Of the 137 patients initially diagnosed as of having isolated eardrum perforation who were well enough to be discharged, none later developed manifestations of pulmonary or intestinal blast injury. Furthermore, 18 patients with pulmonary blast injuries had no eardrum perforation.[72]

INJURIES DUE TO ANTIPERSONNEL LANDMINES

Landmines are defined as any “ammunition placed under/on or near the ground or other surface area and designed to be detonate or explode by the presence, proximity or contact of a person or vehicle.” Antipersonnel landmines are small in size and are specifically targeted for human beings on foot. They are difficult to locate or detect and are activated by direct pressure. These mines could be highly destructive and serve their purpose with telling effect. They are mainly used in guerilla war and in addition to causing physical harm. They also serve as a psychological deterrent to advancing combatants.[81]

Since 1970s the landmine, like the automatic riffle, has become a weapon of choice for many armies and resistance groups around the world. They are readily available from governments and also a vast global network of private arms suppliers. These mines could also be manufactured relatively easily and cheaply.[82]

The explosion of mines causes hideous mutilation, deaths and devastating injuries. The small antipersonnel mines disable those who traverse their paths. The injury potential of this weapon system, in comparison to that of riffle bullets and fragmentation \weapons is primarily focussed on the lower extremities with a fair proportion of those wounded having injuries remote from the legs. The high amputation rate of these injuries poses a psychological problem as well as orthopaedic and rehabilitative ones.

Unlike bombs or artillery shells which are designed to explode when they approach or hit their target, these landmines lie dormant until a person, a vehicle or animal triggers their firing mechanism. They are blind weapons that cannot distinguish between the footfall of combatant from that of a noncombatant. They recognise no cease-fire and could go on killing and injuring long after the hostilities have ended.[83]

This manmade epidemic has been largely unreported in the medical literature until relief workers drew attention to the thousands of limbless victims of antipersonnel mines in Cambodia.[81] It is estimated that there are more than 80 million landmines laid in 65 countries worldwide, some recently and others from conflicts of the past. Together they kill or maim about 150 people a week.[83-86] In recent past, landmines were injuring large numbers of people each year in Cambodia. Burma, Mozambique, Ethiopia, Somalia, Iraq, Nicaragua, Angola, Sri-Lanka and many other countries.[81-86]

As landmines could cause injuries even long after the conflicts are over, medical professionals who are likely to encounter these injuries should have a detailed knowledge and skill in treating such injuries.

Pattern of Injury

Victims of antipersonnel mines present recognizable patterns of injury. These injuries were categorised according to the patterns described by Coupland and Korver of the International Committee of the Red Cross.[87] Each pattern carries its own implications for the surgeon and the patients long-term disability.

Pattern 1

The victim triggeres buried mines by stepping on the device. They usually have a traumatic amputation of part of the lower limb, with less severe injuries elsewhere: mud, grass. Fragments of mine, boot and the remains of the foot were blown upwards into the leg, genitals, buttocks and the contralateral are causing secondary infection. These injuries are the most severe as there is close contact between the device and the foot, when it explodes.

Pattern 2

The pattern of wounding is more random and consists of multiple shrapnel wounds from the mines triggered near the victim.

Pattern 3

The mine explodes while being handled resulting in injury to the hands and face. Eye injury is sustained from fragments, mud or sand thrown up by the explosion; many victims lose one or both eyes.

Type of Injury to Foot

Types of injuries to foot are graded in to four grades and they are presented in the form of a line drawing (Fig. 20.19A) and discussed individually:

Grade 1 Injury

The injury occurs through the fore foot. There is no damage to the ankle and subtalar joints. The resulting disability is minimal. This type of injury is uncommon. Amputation can be carried

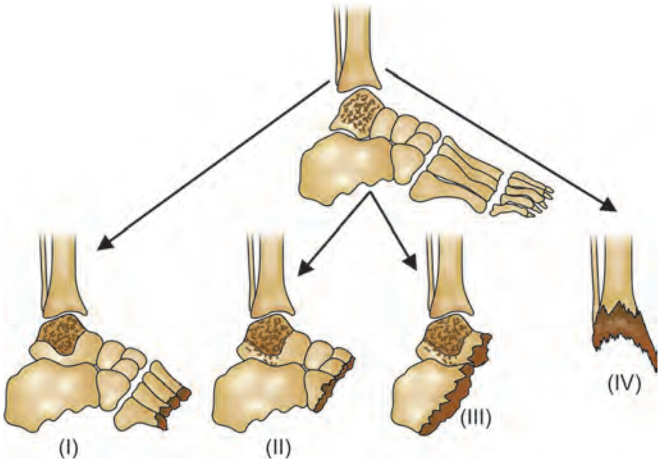


Fig. 20.19A: Classification of foot injuries (Grades I to IV)

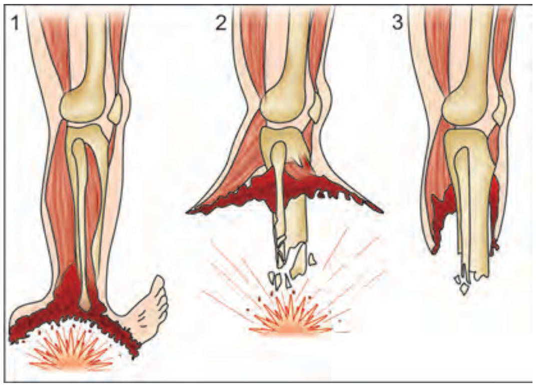


Fig. 20.19B: Diagram showing: (1) How an injury confined to foot, is associated with damage to proximal compartmental muscle. (2) Explosive injury with traumatic amputation of lower leg. (3) How the extent of proximal damage to leg be hidden when the skin is returned to position

out through the metatarsal bones giving the patient a useful stump.

Grade 2 Injury

The injury takes place through the distal row of tarsal bones and is accompanied by damage to the long flexor tendons on the dorsal aspect of the foot. The ankle and subtalar joints, calcaneum and the heel pad are intact. An amputation at this level will result in a viable stump, but on weight bearing the stump goes into plantar flexion as a result of which it becomes difficult to fit a prosthesis. These victims invariably end up with a below-knee amputation.

Grade 3 Injury

The injury occurs through the calcaneum with complete destruction of the heel pad. They require a below knee amputation.

Grade 4 Injury

This is the most common and most severe injury where the entire foot is blown off above the ankle joint. It is her unfortunate that this is the commonest injury seen. Treatment is a below knee amputation.

Pathological Aspects of Limb Injury

Severe limb injury or traumatic amputation produces different levels of tissue damage within the limb due to the variable resilience to injury of skin, fat and muscle. The most striking example is the extreme form of compartment syndrome produced within the leg by antipersonnel mines. This aspect of tissue damage at different levels in the leg caused by the blast of antipersonnel mines should be remembered by all surgeons treating such injuries and the correct level of surgical amputation should be selected to avoid complications (Fig. 20.19B).

The three objectives of primary amputation for mine injuries of the foot. In order of priority are:

- To excise dead and contaminated tissue and to remove accessible foreign bodies.
- To be able to perform delayed primary suture
- To leave a stump that is acceptable for fitting prosthesis.

The victims of antipersonnel mines are disabled for life. The majority are disabled as a result of traumatic amputation of the lower limb, while a smaller number suffer disability as a result of traumatic amputation of the hand, loss of vision or both.

Rehabilitation of victims of various mine explosions (Fig. 20.20) should begin as early as possible. This should include physical exercise to the uninvolved muscles and joints and more importantly psychological rehabilitation. Victims of mines suffer tremendous psychological trauma and may take years to regain confidence. Therefore, the aspect of psychological rehabilitation should not be neglected in the overall management of these victims of landmines.

Prevention of Antipersonnel Mine Injuries (Figs 20.21 and 20.22)

- *Public awareness:* The medical profession should document the mental and physical suffering experienced by victims of land mines. They should also ensure that the public is made aware of the suffering caused by this man made epidemic. The community living in regions of conflict should also be informed of dangerous areas that are probably laid with mines and best avoided.
- *Mine survey and eradication:* Casualties should be reduced by clearing and destroying the mines that have been already laid. However, complete mine clearing is an expensive, dangerous and perhaps an impossible task as observed in Falklands, Afghanistan and more recently in Kuwait.
- *Ban on the use of antipersonnel mines:* A campaign for the ban of these indiscriminate weapons has already been started by two medical organisations in the United States, namely, *Physicians for Human Rights and Human Rights Watch.*

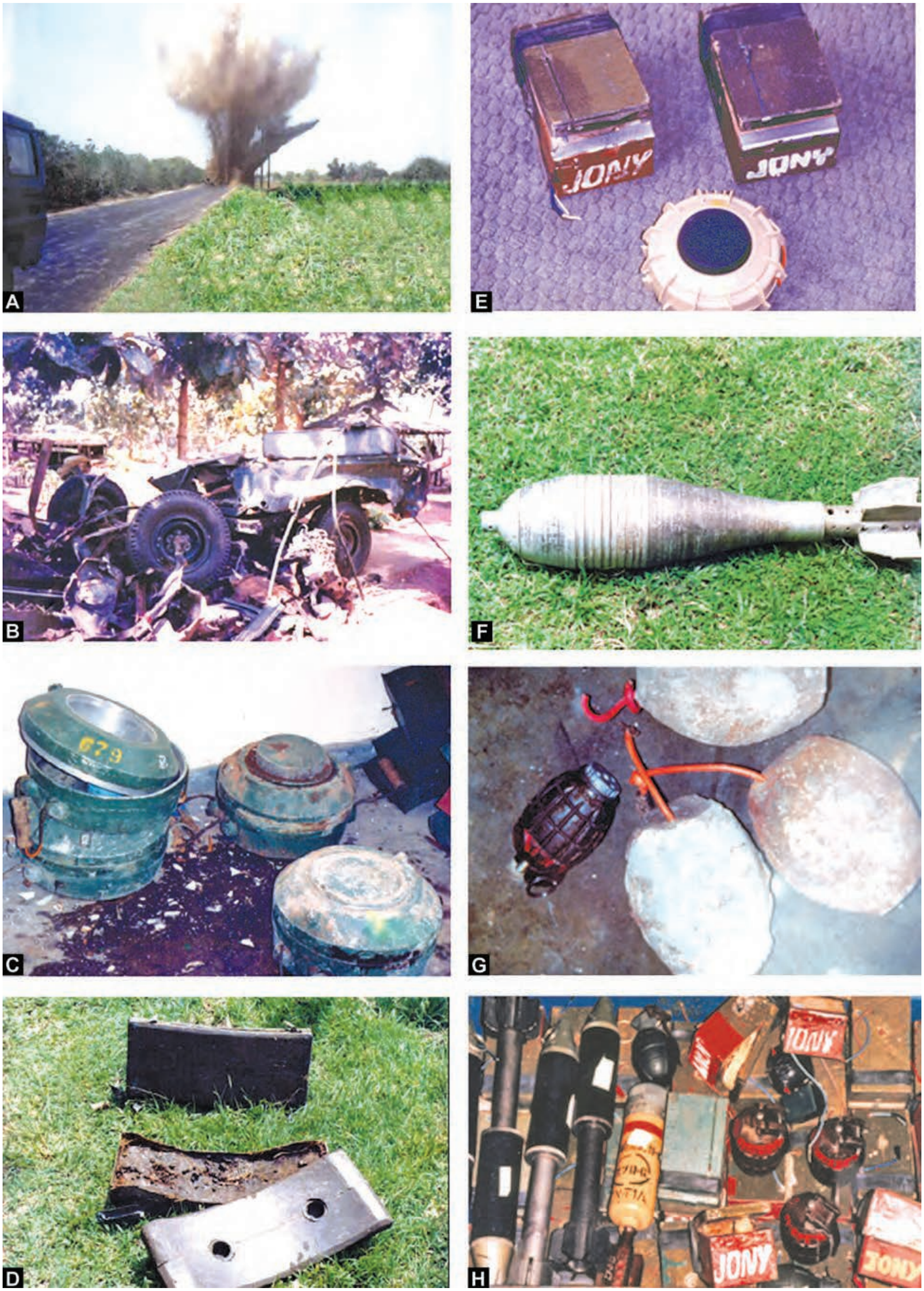
EMERGENCY MANAGEMENT OPTIONS

Follow your hospital and regional disaster plan. Expect an upside-down triage, with the most severely injured arrive after the less injured, who typically bypass EMS triage and go directly to the closest hospitals. Double the first hour's casualties for a rough prediction of the total first wave of casualties.

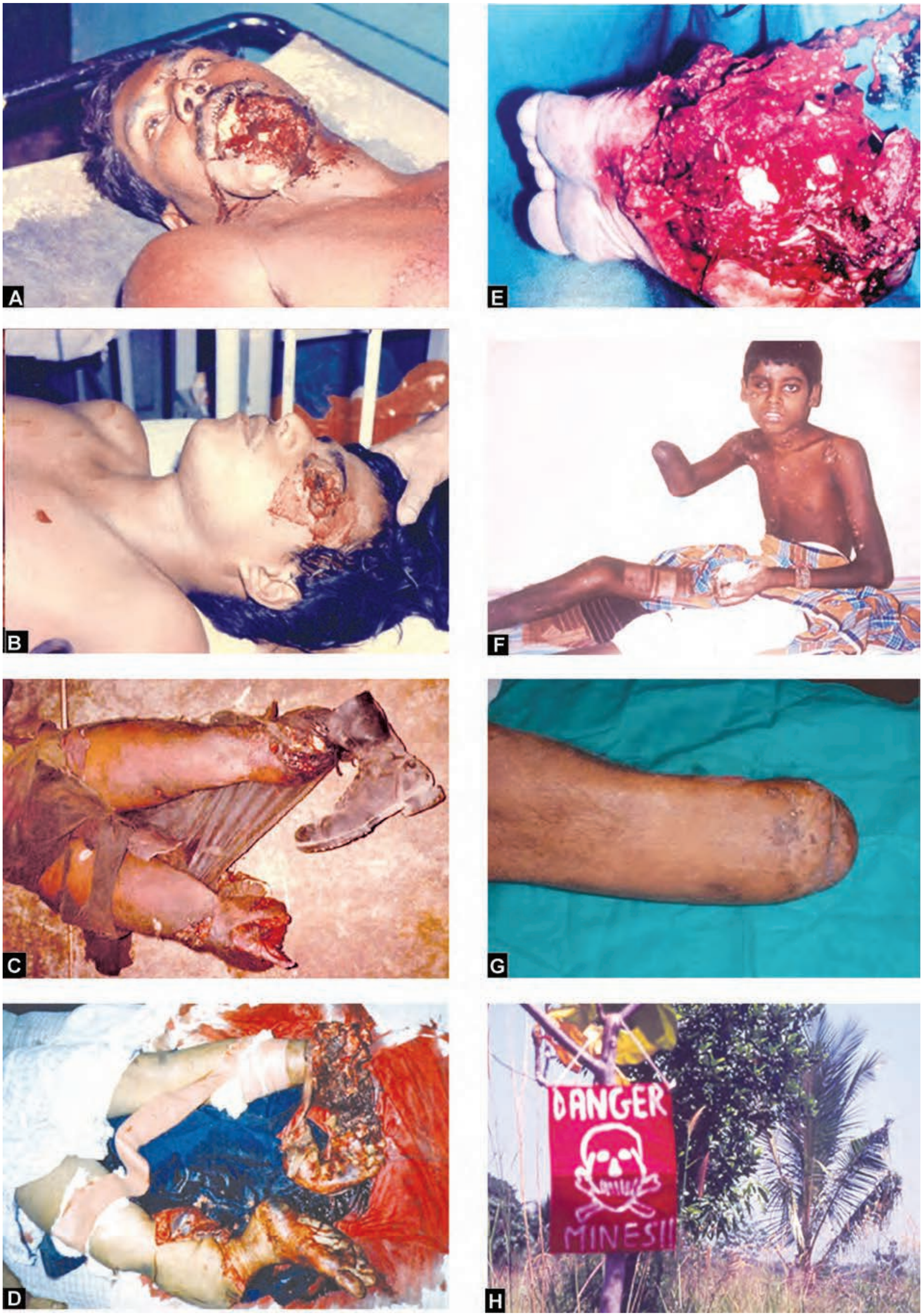
Mass Casualties Predictor[76-80]

To predict the total number of casualties your hospital can expect, double the number of casualties the hospital receives in the first hour.

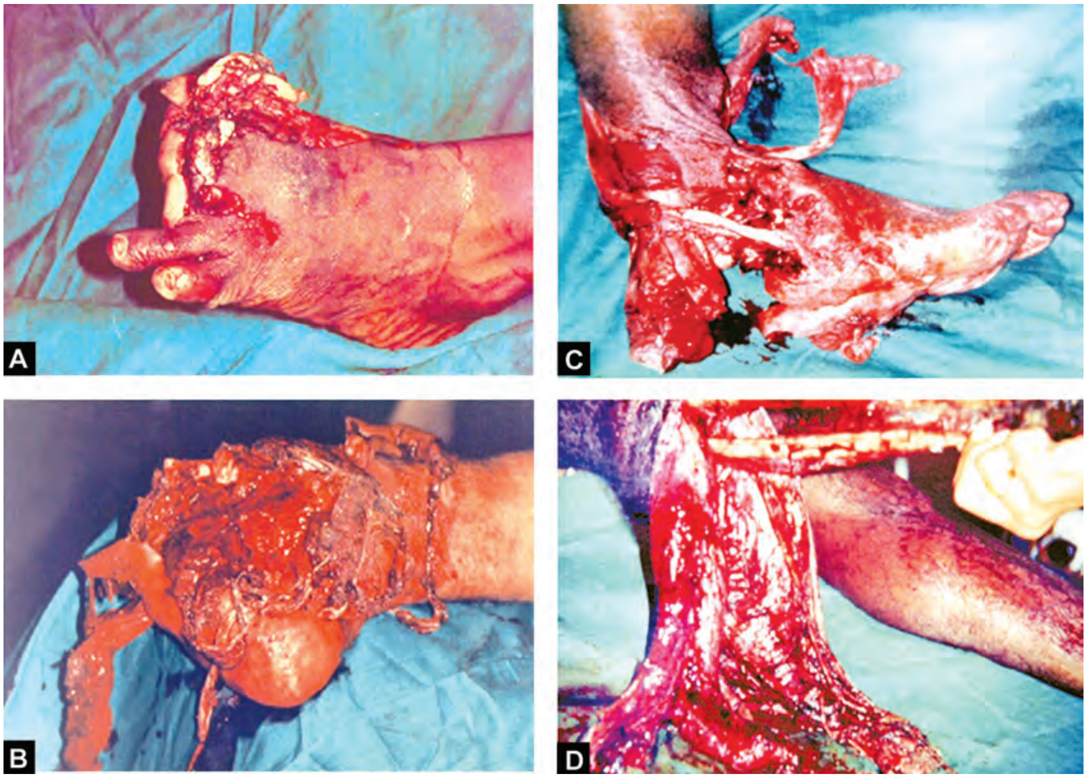
Total expected casualties = (No. of casualties arriving in 1-hr window) × 2.



Figs 20.20A to H: Different types of explosives: (A) Landmine explosion, (B) Jeep blown up by landmine, (C) Anti tank mines, (D) Claymore mines, (E) Antipersonnel mines, (F) Mortar shell, (G) Hand grenades, (H) Mortar shells (*Courtesy:* Dr G Gunetilleke, Consultant Surgeon, Sri Jayawardenepura General Hospital, Nugglgoda, Sri Lanka)



Figs 20.21A to H: Blast injuries: (A) Shrapnel injury to chin, (B) Shrapnel injury to eye, (C) Bilateral traumatic amputations, (D) Blast injury to both lower limbs, (E) Antipersonnel injury to foot, (F) Child injured by mines, (G) Below knee amputation stump, (H) Danger signs anticipating mines planted (*Courtesy:* Dr G Gunetilleke, Consultant Surgeon, Sri Jayawardenepura General Hospital, Nugglgoda, Sri Lanka)



Figs 20.22A to D: Types of antipersonnel injuries to foot by landmines: (A) Antipersonnel injury to foot – Grade I, (B) Antipersonnel injury to foot – Grade II, (C) Antipersonnel injury to foot – Grade III, (D) Antipersonnel injury to foot – Grade IV (*Courtesy:* Dr G Gunetilleke, Consultant Surgeon, Sri Jayawardenepura General Hospital, Nugglgoda, Sri Lanka)

Obtain and record details about the nature of the explosion, potential toxic exposures and environmental hazards, and casualty location from police, fire, EMS, ICS commander, regional EMA, health department, and reliable news sources. If structural collapse occurs, expect increased severity and delayed arrival of casualties.

When trying to determine how many casualties a hospital can expect after a mass-casualty event, it is important to remember that casualties present quickly and that approximately half of all casualties will arrive at the hospital within a one-hour window. This one-hour window begins when the first casualty arrives at the hospital.

The total expected number of casualties will be an estimate. There are many factors that may affect the accuracy of this prediction, including transportation difficulties and delays, security issues that may hinder access to victims, and multiple explosions or secondary effects of explosion (such as a building collapse).

PATTERNS OF HOSPITAL USE

In the confusion that often follows a mass-casualty event, managing a hospital can be challenging. Historically, mass-casualty events show patterns of hospital use. Public health professionals and hospital administrators can use this information to handle resource and staffing issues during a mass-casualty event.

Within ninety minutes following an event, 50 to 80 per cent of the acute casualties will likely arrive at the closest medical facilities. Other hospitals outside the area usually receive few or no casualties. The less-injured casualties often leave the scene under their own power and go to the nearest hospital. As a result:

- They are not triaged at the scene by emergency medical services (EMS).
- They may arrive to the hospital before the most injured.
- On average, it takes 3-6 hours for casualties to be treated in the emergency department (ED) before they are admitted to the hospital or released.[80]

MEDICAL MANAGEMENT OPTIONS

Blast injuries are not confined to the battlefield. They should be considered for any victim exposed to an explosive force. Clinical signs of blast-related abdominal injuries may be silent initially until signs of acute abdomen or sepsis come forward. Standard penetrating and blunt trauma to any body surface are the most common injuries seen among survivors.

Primary blast lung and blast abdomen are associated with a high mortality rate. Blast lung is the most common fatal injury among initial survivors. Blast lung presents soon after exposure. It can be confirmed by finding a butterfly pattern on chest X-ray. Prophylactic chest tubes (thoracostomy) are recommended when general anesthesia and/or air transport are likely.

Auditory system injuries and concussions are easily overlooked. The symptoms of mild traumatic brain injury and post-traumatic stress disorder can be identical.

Isolated tympanic membrane rupture is not a marker of morbidity; however, traumatic amputation of any limb is a marker for multi-system injuries.

Air embolism is common, and can present as stroke, myocardial infarction, acute abdomen, blindness, deafness, spinal cord injury, or claudication. Hyperbaric oxygen therapy may be effective in some cases.

Compartment syndrome, rhabdomyolysis, and acute renal failure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings. Consider the possibility of exposure to inhaled toxins and poisonings (e.g. carbon monoxide, CN, MetHgb) in both industrial and criminal explosions.

Wounds can be grossly contaminated. Consider delayed primary closure and assess tetanus status. Ensure close follow-up of wounds, head injuries, eye, ear, and stress-related complaints. Communications and instructions may need to be written because of tinnitus and sudden temporary or permanent deafness.

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Chapter 21

Effects of Cold and Heat

Thermal injury is defined as injury to the body resulting from localised or generalised exposure to extremes of temperature due to various etiological factors. It can be classified as:[1-10]

1. Hypothermia:
   - Trench foot
   - Frostbite
2. Hyperthermia:
   - Heat cramps
   - Heat prostration
   - Heat hyperpyrexia
3. Injury due to heated solid objects
4. Flame burns (dry heat)
5. Scalds (moist heat)
6. Chemical burns
7. Corrosive burns
8. Radiation injury
9. Electrical injury – Electrocution, lightening
10. Frictional heat – *Corn, shoe bite, brush burns, rope burns*.[9]

Normally body temperature is said to be 98.6°F (36°C) when measured orally. However, the body temperature may vary from individual to individual, depending on age, time of the day, physical exertion, and so on. Maintenance of normal body temperature is a delicate balance between heat load and heat loss. Heat load is the sum of heat generated by oxidation of metabolic products and heat acquired from the environment around[1,6-8] Heat is lost by three mechanisms:[6-8]

i. *Conduction,*
ii. *Radiation,* and
iii. *Evaporation.*

Heat loss by *evaporation* has *further two more mechanisms.*[1-3,5,6]

- *Insensible heat loss* – this is due to continued diffusion of water molecules through the skin and respiratory surfaces regardless of the body temperature.
- *Heat loss by sweating* – this is more important. In cold weather, the sweating is essentially zero, while in hot weather it is maximum.

HYPOTHERMIA

The term hypothermia is used when an individual's body temperature is below 95°F (35°C). This will occur when the loss of body heat exceeds heat production.[2-6,10-13] This has both clinical and forensic aspects, as even in temperate climates in winter, many people suffer and die of hypothermia.[14] Effect of hypothermia may commonly involve extremities and other exposed body parts, i.e. face. This condition is further subclassified as:

Trench Foot

This is due to exposure to cold (5-8°C) coupled with dampness and there will be no tissue freesing. There will be no permanent injury in trench foot.

Frostnip and Frostbite

This is due to exposure to cold below 2.5-0°C. Here there will be tissue freesing.[14] Initially there will be just numbness and tingling, and no actual tissue damage. This is just because the blood vessels supplying the affected tissue constrict. The skin turns white and waxy or gray in colour and mottled, but feels normal to touch. At this phase the cold injury is known as *frostnip*.[14] *Frostbite* follows then, when no treatment is given at the initial phase of *frostnip*. Ice crystals are then formed in the skin and deeper tissues, which can exert osmotic force, causing water to move from intercellular space. This results in oedema of tissues (filling up of intercellular spaces with fluid). At the same time there is cellular dehydration and hyperosmolarity. There is also denaturation of proteins and destruction of enzymes. The skin is numb and discoloured; it is purple in most severe cases. On touching the skin, it does not give normal '*bouncy*' feeling over skin, rather it feels *hard* like a rock, block of ice or perhaps like a piece of chicken removed from the freezer. Blisters appear which may become haemorrhagic.[14]

Incidence

The most common cause is *exposure* to low temperature.[2] The term *exposure* usually means a considerable element of hypothermia. *Frost bite* commonly occurs in soldiers at winter warfare hiding in the trenches or in a shipwreck in Antarctic waters, etc. Many deaths in mountaineers, hill workers, swimmers and other sportsmen are due to *exposure* to low temperature. Another group that may suffer *exposure* to cold, include *drunken people*, who may collapse or lie down to sleep after too much alcohol and become hypothermic, especially since alcohol causes vasodilatation of the skin, increasing heat loss. Accidental hypothermia occurs among individuals who are lost while hiking or skiing, individuals immersed in ice cold water,[2] etc., in places such as higher altitudes of *Himalayas, North Bihar, Uttar Pradesh, Kashmir,* etc. In marine disasters, hypothermia may be as common a cause of death as drowning or may cause terminal drowning in survivors who can swim or who are clinging to wreckage. Here death may occur within a couple of minutes from sheer heat loss.[14]

According to Bernard Knight, among the civilian life most of the deaths from hypothermia are seen among old people and in children.[1,14] In some old people, hypothermia may be linked with strange psychiatric condition, in which they hide themselves in some corners, in cupboards, or under piles of furniture or other house hold goods. Often they are found nude, making their death suspicious of criminal victimisation.[14] However, this so called *'hide-and-die syndrome'* is though well known, it is not clear whether victim first become hypothermic and then confused, or whether they are already confused, so take off their clothing or hide in a cold place, and thus become hypothermic.[14]

Factors Modifying Effects of Cold

- *Age* – adults are able to bear cold better than very young and old. Children have a high *body surface-to-weight ratio* and lose heat rapidly. In some cases of deliberate neglect or careless family circumstances, infants may be left in unheated rooms in winter and suffer hypothermia.[1,2,14]
- *Duration of exposure* – the longer the duration, the more severe are the effects due to evaporation of body heat.
- *Bodily condition* – fatigue, exhaustion, intoxi-cation and starvation hasten the effects of cold.
- *Thyroid deficiency* – degree of thyroid deficiency, even clinical *myxoedema*, which predisposes to low body temperature, may predispose to hypothermia.[2,14]
- *Drugs/ Medications* – taking of phenothiazine drugs also predisposes towards hypothermia.[14]

Pathophysiology of Cold

When a healthy person is exposed to extreme cold, following changes may occur and they are[10-14] (Fig. 21.1):

- Reduction of heart rate and respiration.
- Impairment of tissue respiration due to failure of dissociation of oxygen from hemoglobin, resulting in tissue anoxia.
- Lowering of *body metabolism*.
- Slowing of *body enzymatic process*.
- Fall of body temperature resulting in *cessation of vital functions*.
- *Vascular response* of the body for excessive cold includes-*Vasoconstriction of superficial blood vessels* resulting in numbness of skin which creates great discomfort resulting in removal of the garments and is known as paradoxical undressing.[6,9,15]

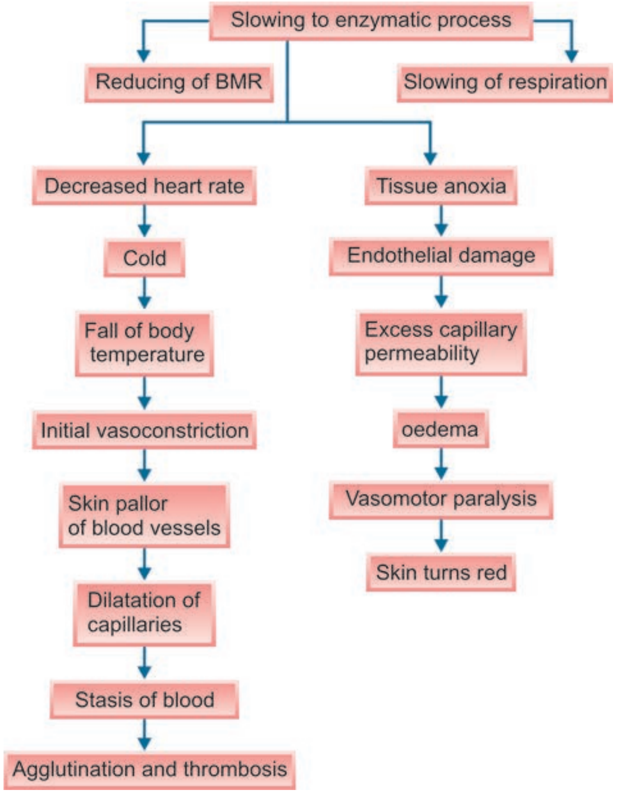


Fig. 21.1: Pathophysiology of hypothermia

Paradoxical Undressing

Victims of hypothermia often exhibit altered states of judgement, which is know in mountaneering circles as being *"cold stupid"*. One common but especially bizarre behaviour of hypothermia victims is a conudrum known as *'paradoxical undressing'*.

Pathophysiology

What exactly happens in paradoxical undressing is discussed below in detail.

To understand the mystery of why hypothermia victims exhibit *paradoxical undressing*, we need to know something regarding how the body attempts to protect itself when stressed by the cold. As the body becomes colder, it shunts blood away from the extremities and into the warm core in order to protect vital organs. This is why the feet and hands will often become cold first. Shunting of blood to the core and away from extremities is accomplished through *vasoconstriction* of peripheral circulation. This allows the outer portions of the body to become better at insulating the core, since it is losing less heat to the outside world.

What Causes Paradoxical Undressing?[15]

Vasoconstriction occurs when the smooth muscles within the vasculature contract. This effort requires a steady input of energy in the form of glucose from the body's energy stores. However, due to a lack of blood now travelling to these muscles, they eventually exhaust. As the muscles of the constricted blood vessels run out of energy, they fatigue, relax, and open up. This is known as vasodilatation. With vasodilatation of the blood vessels, an infusion of warm blood from the core of the body rushes into the peripheral extremities. This causes the hypothermia victim to feel overly warm and to start shedding layers of clothing, contrary to the reality that their body temperature is continuing to drop. The victim's warm blood rushing from their core, coupled with the removal of warm clothing, causes their body temperature to fall even faster. This serves to hasten death from hypothermia and results in another case of *paradoxical undressing*. Mountaineers with hypothermia have been known to push aside warm clothing and resist rescuers' efforts to warm them. It is interesting to note that there are no known hypothermia victims who have reached the stage of *paradoxical undressing* and survived without outside intervention.

- *Paralysis of vasomotor control* of the blood vessels results in thrombosis, capillary dilation and stasis of blood and tissue necrosis. This is called *frostbite.*
- Damage to capillary endothelium resulting in increase in capillary permeability, transudation and oedema.

Frost Bite

(Chilblain, Trench foot, Immersion foot)

Frost bite is a syndrome complex comprising of local tissue necrosis.[2-8,13]

Incidence

Frost bite commonly occurs in soldiers at winter warfare hiding in the trenches or in a shipwreck in antarctic waters, etc.

Causes

- Vasospasm
- Paralysis of vasomotor control of blood vessels.

Clinical Findings

- Skin will be icecold, showing erythematous patches on distal and exposed parts such as ear, nose, fingers and toes.
- Generalised muscular stiffness.
- Feeble pulse and low heart rate with low blood pressure.
- Depressed reflexes.
- Lethargy, feels heavy, drowsiness *(due to cerebral anemia)*.
- Stupor, delirium, coma and death gradually due to reduction in oxygen supply to tissue, due to its inability of dissociation from oxyhaemoglobin.

Treatment

- Gradual restoration of body warmth by putting the victim on warm bed, hot water fomentation, hot coffee or tea and stimulants like—digitalis, alcohol, strychnine, etc. orally.
- Warm saline may also be given intravenously.

Postmortem Findings

- Skin—pale with irregular dark red patches on exposed parts.
- Brain—congested with ventricles filled with serum.
- Heart—full of blood on both sides.
- Pancreas—microscopic examination may reveal fat necrosis.

Medicolegal Importance

- Suicide by exposing to cold is unlikely.
- Most of the deaths by exposing to cold are:
  - *Accidental:* As observed in alcoholics, who fall asleep even in snow, or a person lost in snow drifts, etc.
  - *Homicidal:* As observed in getting rid of unwanted newborn babies or elderly people, etc. by leaving them exposed to cold weather.
  - *'Paradoxical undressing'* and *'Hide-and-die syndrome'* (Refer above) – Both of these can create *suspicion of sexual offence* on the victim, especially if the victim is a female.

HYPERTHERMIA (HYPERPYREXIA)

Hyperthermia is an acute condition which occurs when the body produces or absorbs more heat than it can dissipate. It is usually due to excessive exposure to heat. The heat-regulating mechanisms of the body eventually become overwhelmed and unable to effectively deal with the heat, and body temperature climbs uncontrollably. This is a medical emergency that requires immediate medical attention.[16-21] In its advanced state hyperthermia is referred to as *heat stroke* or *sunstroke*, Heat stroke may come on suddenly, but usually follows a less-threatening condition commonly referred to as *heat exhaustion* or *heat prostration*.

Progression

Body temperatures above 40°C (104°F) are life-threatening. This compares to normal body temperature of 36-37°C (97-98°F). At 41°C (106°F), brain death begins, and at 45°C (113°F) death is nearly certain. Internal temperatures above 50°C (122°F) will cause rigidity in the muscles and certainly, immediate death.[17]

Causes

Trauma and death from hyperthermia or heat is due to the exposure to heat, derived from:[1-9]

- Natural source such as the heat derived from the sun
- Artificial source such as industrial furnaces, huge baking ovens, etc.
- Poorly ventilated or closed rooms or a factory wherein the temperature is high and air is moist.

Three conditions may result due to high environmental temperature:

- *Heat cramps*—no rise in body temperature
- *Heat prostration*—subnormal body temperature
- *Heat hyperpyrexia*—rectal temperature above 41 degrees.

Predisposing Factors

Following factors are considered predisposing to the effects of hyperthermia:

- Malnourishment
- Overexertion
- Fatigue
- Chronic alcoholism, hunger, lack of sleep, etc.
- Mental depression.

Pathophysiology of Hyperthermia

Effects of hyperthermia may vary from heat cramps, simple exhaustion or transient fainting to profound comatose condition with respiratory and cardiac failure resulting in death of the victim. The various pathological events are presented in the Figure 21.2A[6] and the signs and symptoms, treatment are discussed in detail.[6,16,17] One of the body's most important methods of temperature regulation is through perspiration. This process draws heat from inside, allowing it to be carried off by radiation or convection. Evaporation of the sweat causes further cooling, since this endothermic process draws yet more heat from the body. Initially the victim will likely be sweating profusely. This results in body becoming sufficiently dehydrated. With this, production of sweat and avenue of heat reduction through sweating, is arrested. Thus, when the body is no longer capable of sweating, core temperature begins to rise swiftly.

Signs and Symptoms

Heat prostration/heat exhaustion is characterised by mental confusion, muscle cramps, and often nausea or vomiting. With continued exposure to ambient heat, temperature may rise into the 39 to 40°C range (103 to 104°F), and lead to full-blown heat stroke.

Victims may become confused, may become hostile, often experience headache, and may seem intoxicated. Blood pressure may drop significantly from dehydration, leading to a possible fainting or dizziness, especially if the victim stands suddenly. Heart rate and respiration rate will increase (tachycardia and tachypnoea) as blood pressure drops and the heart attempts to supply enough oxygen to the body. The skin will become red as blood vessels dilate in an attempt to increase heat dissipation. The decrease in blood pressure will cause blood vessels to contract as heat stroke progresses, resulting in a pale or bluish skin colour. Complaints of feeling hot may be followed by chills and trembling, as is the case in fever. Some victims, especially young children, may suffer convulsions. Acute dehydration such as that accompanying heat stroke can produce

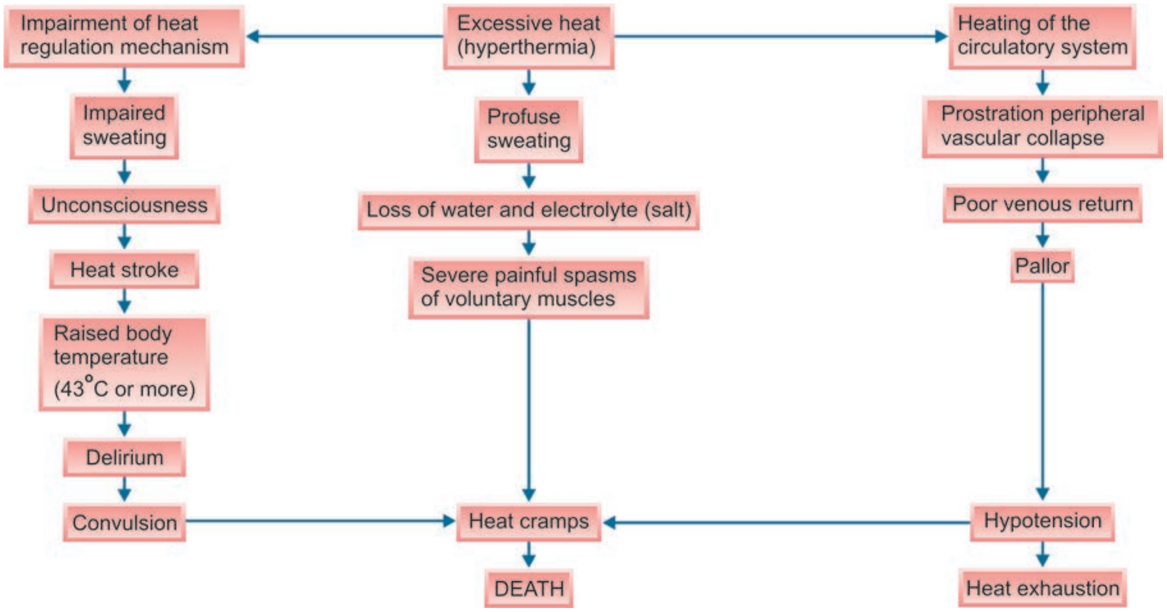

Impairment of heat regulation mechanism
Excessive heat (hyperthermia)
Heating of the circulatory system
Impaired sweating
Profuse sweating
Prostration peripheral vascular collapse
Unconsciousness
Loss of water and electrolyte (salt)
Poor venous return
Heat stroke
Severe painful spasms of voluntary muscles
Pallor
Raised body temperature (43°C or more)
Delirium
Convulsion
Heat cramps
Hypotension
DEATH
Heat exhaustion

Fig. 21.2A: Pathology of hyperthermia

nausea and vomiting; temporary blindness may also be observed. Eventually, as body organs begin to fail, unconsciousness and coma will result. Given below is the summary of signs and symptoms:

Clinical Manifestations

Exposure to high temperature produces several effects, which can be studied under following three headings, namely *heat cramps, heat exhaustion* and *heat stroke:*[6-8, 16-17]

Heat Cramps

The victim here complains of severe painful spasms in the voluntary muscles of the body due to excessive perspiration and loss of body electrolytes *(following strenuous work in a hot atmosphere).* The mortality rate in this is almost nil or negligible.

Heat Exhaustion (Heat Collapse, Heat Syncope, Heat Prostration)

Heat exhaustion is chiefly because of the effect of the heat on the circulatory system and characterised by[6]:

- Prostration
- Peripheral vascular collapse
- Pallor *(due to poor venous return)*
- Hypotension *(due to poor venous return)*
- Flushing of the face, throbbing temples, scanty perspiration
- Collapse

The patient usually recovers and mortality is very rare.

Heat Stroke (Heat Hyperpyrexia, Sunstroke, Systemic Hyperthermia, Thermic Fever)

Heat stroke is due to the impairment of heat regulation mechanism in the body, especially resulting in death of the victim due to the paralysis of medullary centres. The syndrome complex may present in two forms:

- An acute form with sudden onset without any prodromal symptoms.
- A gradual onset form with definite prodromal symptoms.

Prodromal symptoms These include headache, nausea, vomiting, dizziness, weakness in legs, excessive desire to micturate, etc.

Manifestations of the syndrome Various manifestations of the syndrome are:

- Sudden unconsciousness and falling or dropping down of the victim to the ground *(called heat stroke).*
- Findings in the face—it will be flushed; pupils dilated initially and become constricted (pinpoint pupil) in later stage, conjunctiva is congested.
- Findings in the skin—it will be hot and dry, with no sweating.
- Cardiovascular system—pulse will be full, bounding and rapid.
- Respiratory system—breathing will be stertorous and rapid.
- Body temperature—43°C or more (heat hyperpyrexia)
- Central nervous system—the victim will be presenting with delirium and convulsions.

The death is due to paralysis of heat regulatory centre in medulla *(hypothalamus).*

Treatment

First Aid

Heat stroke is a medical emergency requiring hospitalisation, and the local emergency system should be activated as soon as possible. The body temperature must be lowered immediately. Both passive and active cooling is helpful.

Passive Cooling

The victim should be moved to a cool area (indoors, or at least in the shade) and clothing removed to promote heat loss (passive cooling).

Active Cooling

- The person is bathed in cool water, a hyperthermia vest can be applied, or the person may be wrapped in a cool wet towel.
- Cold compresses to the torso, head, neck, and groin will help cool the victim. A fan may be used to aid in the evaporation of water (evaporative method).
- Use of ice and very cold water may lead to hypothermia; hence they should be used only when there are means to monitor the victim's temperature continuously.

- Immersing a victim into a bathtub of cold water (immersion method) is a recognised method of cooling. This method requires the effort of 4-5 persons and the victim should be monitored carefully during the treatment process. This should be avoided in an unconscious victim; if there is no alternative, the victim's head must be held above water.

Hydration

- Hydration is of paramount importance in cooling the victim. This is achieved by drinking water (Oral rehydration). Commercial isotonic drinks may be used as a substitute. Alcohol and caffeine should be avoided due to their diuretic properties. Some authorities oppose the administration of any fluids, except by emergency personnel. Intravenous hydration (via a drip) is necessary if the victim is confused, unconscious, or unable to tolerate oral fluids.
- The victim's condition should be reassessed and stabilised by trained medical personnel.
- The victim's heart rate and breathing should be monitored, and CPR may be necessary if the victim goes into cardiac arrest.
- The victim should be placed into the recovery position to ensure that their airway remain open.

Prevention

- Avoid overheating and dehydration.
- Light, loose-fitting clothing will allow perspiration to evaporate.
- Wide-brimmed hats in bright colour keep the sun from warming the head and neck; vents on a hat will allow perspiration to cool the head.
- Avoid strenuous exercise during daylight hours in hot weather.
- Be aware of humidity in presence of direct sunlight which cause the heat index to be 10°C (18°F) hotter than the atmospheric temperature shown in thermometer.[16,17]
- Persons in hot weather need to drink plenty of liquids to replace fluids lost by sweating. Thirst is not a reliable sign indicating that a person needs fluids. A better indicator is the colour of urine. A dark yellow colour indicates dehydration. While alcohol, tea, and coffee are all diuretics, they will replace more water than they remove (with the exception of concentrated alcohol). However, pure water is still preferred.

Susceptible Populations

While anyone can be affected by hyperthermia, some populations are especially susceptible to heat illness and injury. Heat illness most seriously affects the poor, urban-dwellers, young children, those with chronic physical and mental illnesses, substance abusers, the elderly, and people who engage in excessive physical activity under harsh conditions.[16]

Medicolegal Importance

- Hyperthermia can be created *artificially* by drugs or *medical devices*. This is known as *thermotherapy* or therapy by inducing hyperthermia. Hyperthermia can be intentionally produced for medical purposes.
- It may be used as a cancer treatment to kill or weaken tumor cells, with negligible effect on healthy cells.
- Malignant hyperthermia is a rare complication of some types of general anaesthesia.
- Death due to hyperthermia is usually accidental, as observed in dry humid places. It may rarely be suicidal or homicidal.
- Unfavourable working conditions at industrial unit leading to hyperthermia in an employee can attract provisions of *Workmen's Compensation Act.*
- Murder by administration of mixture of belladonna or atropine and possibly cocaine simulating heat apoplexy is also reported.
- In cases of sunstroke patient may pass into stage of suspended animation and hence resuscitative measures should be offered before pronouncement of death.

Table 21.1 summarizes the salient points on mechanisms, clinical features, treatment complications and medicolegal aspects of hyperthermia[17-22]

Postmortem Findings

No findings can be considered as characteristic. However, following are observed:

- Postmortem caloricity, i.e. the body temperature is high even after the death. The normal postmortem cooling of the dead body is not observed.
- *Eyes* – pitting and sinking of eyeballs is usually seen, which could mimic the evolution of eyeballs.
- Rigor mortis sets in early and passes off early.
- Postmortem stains are marked on account of a greater fluidity of blood.
- Putrefaction is rapid.
- Degeneration of cerebral cortex, cerebellum and basal ganglia are common.
- Congestion of the entire viscera is common.

Note – Blood may be preserved for detection of alcohol as over indulgence in alcohol often precipitates fatal attacks.

SCALDS[1-9]

Scalds are trauma resulting from the application of moist heat commonly involving only superficial layers of the skin.

Causes of Moist Heat

Moist heat is generated in the following forms:

- Hot water or oil or any liquid at or near boiling point
- Superheated industrial steam.

Clinical Features

Usually the scalded area presents a swollen, vesicated and bleached appearance. Since the clothing worn cools faster, the scalding effect is usually less prominent in clothed areas. Likewise, the temperature of the moist heat cools gradually as it disperses all around from the point of contact producing maximum damage at point of commencement.

However, clinically scalding is classified into three degrees:

i. *Erythema,*
ii. *Vesication,* and
iii. *Necrosis of dermis.*

Erythema: This is the reddening of the skin which appears at once as the moist heat is applied (Fig. 21.2B).

Vesication: Also called blister formation is chiefly due to increased capillary permeability, and this needs a few minutes to develop. Usually blisters are surrounded by a bright red zone of inflammation and present as a swelling. On removing a blister, it open leaves a pink coloured raw area.

Table 21.1: Summary of mechanisms, clinical features, management and medicolegal Importance and complications of hyperthermia

Hyperthermic disorders	*Mechanisms*	*Clinical features*	*Management*	*Medicolegal importance and complications*
HEAT STROKE	Failure of thermoregulatory systems resulting in severe hyperthermia and multisystem damage. Usually accompanied by electrolyte imbalance and cardiac arrhythmias. There are often seizures, neurological damage, DIC, rhabdomyolysis, renal and hepatic failure	• Often rapid in onset • May be preceded by headache, nausea, weakness, myalgia • High fever 105-107°F (40.6-46.7°C) • Dilated pupils • Hot dry skin • Lack of sweating (classic) • Confusion, ataxia and/or loss of consciousness • Fast thready pulse • Hypotension • Heart failure	• Support airway, breathing, and circulation • Shift to cooler place and hospitalize • Provide aggressive cooling • I/V fluid and electrolyte therapy • Support vital functions	• Highly lethal • 20 per cent or more have residual neurological damage • Patients with exertional heatstroke may continue to sweat
HEAT EXHAUSTION	Water and/or salt depletion from excessive sweating that is inadequately replaced Hypovolaemia causes weakness and collapse	• Insidious or sudden onset • Weakness, light-headedness, syncope, sweating, nausea • Low-grade fever • Pale, cool, clammy skin • Sweating • Thready pulse • Low BP • There may be ataxia and confusion	• Patient should lie down in a cool area • Fluid and salt should be replenished. Slightly salty or sports drinks can be given in sips over 2-4 hours. IV's needed if very ill or unable to take oral fluids	• May be similar in presentation to insulin shock, alcohol/drug abuse/withdrawal, or hypovolemia from occult blood loss • Usually benign, but may progress to heat stroke
HEAT CRAMPS	Heat cramps are deep and painful spasms in the most actively used muscles and are a direct result of salt depletion	• The affected muscles harden and become tender. Spasms of the upper or lower extremities can be debilitating, and abdominal muscle spasms may mimic an acute abdomen	• Eating salted foods and drinking enough fluid in the heat can usually prevent heat cramps	• Patients should move to a cool area and replenish salt and fluids.
SUNBURN	Sunburn is not considered a heat disorder but occurs with overexposure to ultraviolet (UV) sunrays	• The skin becomes tender and reddened several hours after exposure and may blister and later peel	• Limiting skin exposure to direct sunlight, (midday) and the application of a sunscreen are the best ways to prevent sunburn • Aspirin, and cool moist compresses, relieve discomfort • Corticosteroids may be needed in severe sunburns	• Para-aminobenzoic acid (PABA) and/or benzophenone with sun protection factor (SPF) should be applied prior to sun exposure • Opaque zinc oxide and titanium oxide creams can block sunrays.

Necrosis of dermis: This results when deeper layers of skin are involved.

On healing, scar is much thinner and it produces less contraction and disfigurement.

Medicolegal Importance

- Usually scalds are accidental due to splashing or pouring of fluids while cooking or bathing, etc.
- The accident is common in children or in elderly.
- Boiling water may be thrown with malicious intent.
- Deliberate scalding by hot water is common in child abuse.
- However, suicide and homicide by scalding is extremely rare.
- Scalding could be either antemortem or postmortem. Table 21.2 gives the differences between them.

At times scalding and dry heat burns may have to be differentiated from each other. Table 21.3 gives these differences.

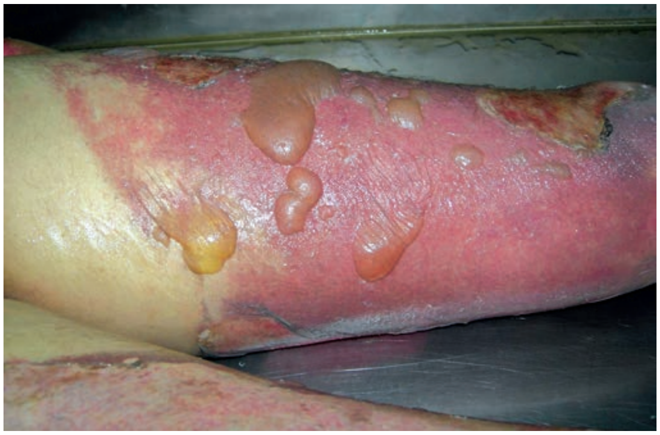


Fig. 21.2B: Scalds of the left thigh

Table 21.2: Differences between antemortem and postmortem scald

Characteristics	*Antemortem scalds*	*Postmortem scalds*
Line of redness	+ve	–ve
Vesicle	+ve	–ve
Content of vesicles	Albuminous	Gas/Air
Infection	+ve	–ve

DEATH DUE TO FIRE *(DEATH DUE TO BURNS)*

In India there are several thousands of deaths occurring due to fire or burns.[1-9] Unfortunately a vast majority of these cases occur in the home and are due to smoking, defective electrical wiring, defective kerosene stove bursts, attempted suicides by *self-immolation*, homicidal burns of young women by the husband or in-laws *(Dowry deaths/bride burning[23,24])*, etc.

Definition

Deaths due to fire or burns usually result from the application of dry heat to the body.[6]

Incidence of Burns

Incidence of burns could be due to building catching fire, clothes worn catching fire, inflammable liquid fire explosions, industrial furnace burns, etc.

Classification of Burns

Three different types of classification are accepted[1-9] (Table 21.4) and they are:

- Dupuytren's classification
- Heba's classification
- Modern classification.

Effects of Burns

- Usually all the two stages of *Modern classification* (Table 21.4) may be seen together.
- *Scarring* is usually more with burns due to dry heat. It would be present in cases involving dermis. Burns involving only epidermis will heal without scar formation.
- *Keloid* formation is more common with corrosive burns.
- *Curling's ulcer*—is a rare sequel of severe burns, seen in the duodenum. It is due to tissue *hypoxia* and capillary endothelial damage (Named after Dr TB Curling who reported it in 1842).

Factors Modifying Effect of Burns[6-7]

- Intensity of heat applied—higher the intensity the more severe will be the effects
- Duration of exposure—more the duration, the more severe will be the effects
- Depth of burn—The depth of burn injuries is particularly important especially if the burn is causing decreased oxygen supply to the end digits of the body or difficulties with chest expansion and breathing. Depending on skin and its morphology (Fig. 21.3) burns could be of three types: *superficial burns, partial thickness burns (mid-dermal and deep-dermal) and full-thickness burns.*

Superficial Burns

These burns are usually red, moist and very painful. The outermost layer of the skin is involved (the epidermis) and there may be blisters present. Healing generally occurs within 7-10 days with minimal or no scarring.

Partial Thickness Burns

- *Mid-dermal:* The outermost layer of skin is lost, as well as parts of the dermis (the next layer of skin). The burn is pink in colour, with small white patches. The skin still blanches on pressure and is painful. Healing occurs in 7-14 days depending on the degree of skin destruction. Some mild pigmentation or scarring may result.

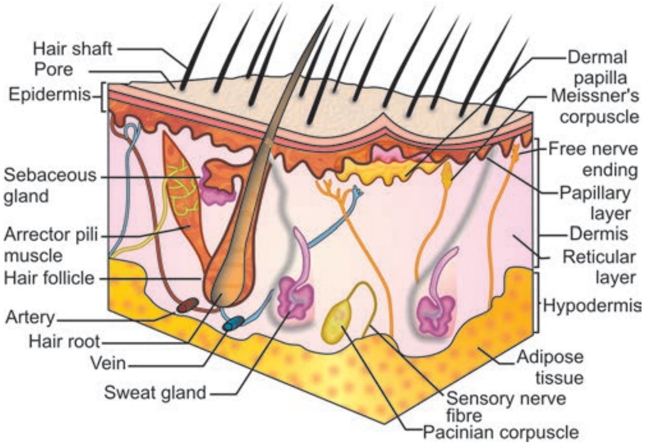


Fig. 21.3: Skin and its morphology presenting the structures in its depth

Table 21.3: Differences between scalds and burns[1-9]

Characteristics	*Scalds*	*Burns*
Skin	Sodden and bleached	Dry and shrivelled
Vesicles	Seen all over scalded area	Only seen at burnt area
Singeing of hair	Absent	Present
Charring	Absent	Present
Soot particles in upper respiratory tract	Absent	Present
Scar	Thin	Thick

Table 21.4: Differences in classification of burns

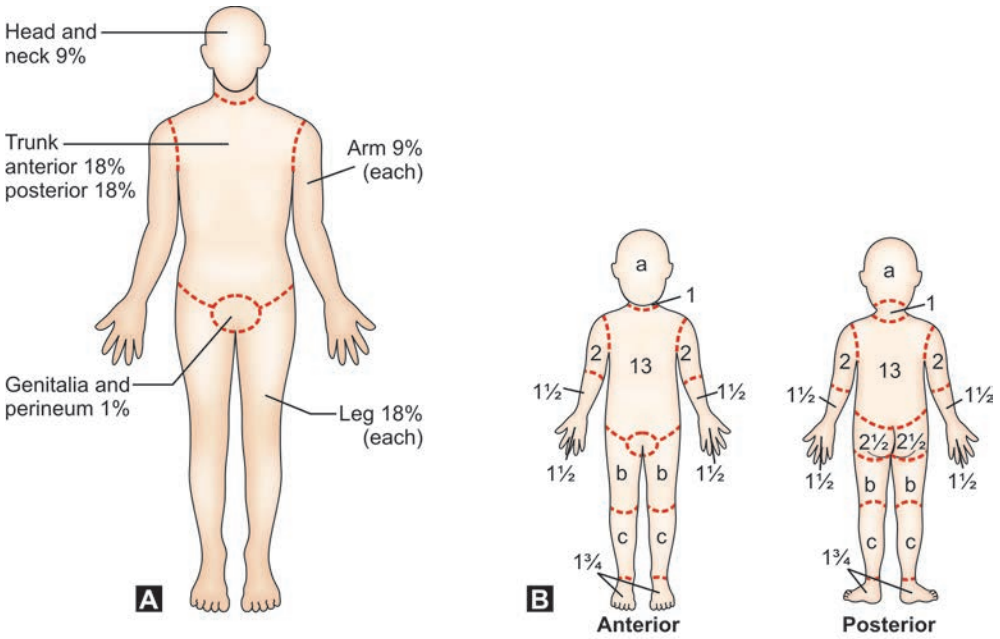
Degree of damage	*Dupuytren's*	*Heba's*	*Modern*
Superficial redness	1st degree	1st degree	Superficial
Vesication	2nd degree	1st degree	Superficial
Destruction of superficial skin	3rd degree	2nd degree	Superficial
Destruction of whole skin	4th degree	2nd degree	Deep
Destruction of muscles	5th degree	3rd degree	Deep
Complete charring	6th degree	3rd degree	Deep

- *Deep-dermal burns:* Here there is deeper dermal destruction. The burn appears white and does not blanched on pressure. The skin is less sensitive and takes a longer period of time of heal, with scarring.

Full Thickness Burns

Full thickness burns extend deep down into the dermis. The burn is leathery, ranges in colour from white/grey/black and is non-painful. There is loss of sensation and it does not blanch on pressure. Healing occurs from around the edges of the surrounding skin but the process is slow, with scarring and contracture.

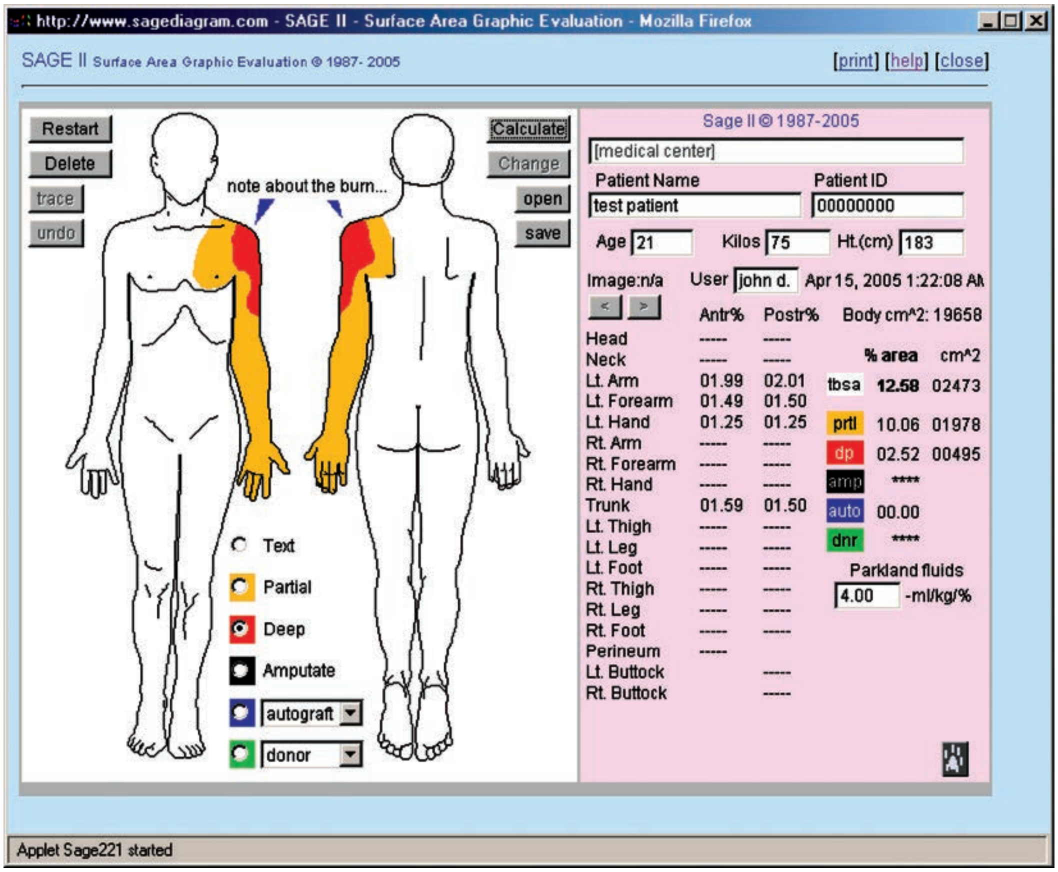
- Extent of body surface area (BSA)–if the surface area is great, fatality is usual. Thus, for estimating the prognosis and deciding the line of treatment, usually the clinicians adopt a rule called *rule of nines,* which helps in estimating fluid loss, shock, etc. Under this, the body is divided into different areas, each representing 9 per cent. When surface area involved is more than 20 per cent the fluid loss is marked, resulting in shock and usually, involvement of 30 to 50 per cent is fatal. Figures 21.4A and B, and Table 21.5 present the idea of estimating the percentage of body surface area involved in burns. *Lund and Browder chart* for children is useful to calculate body surface area in which age of victim is an additional factor incorporated. *'Rule of nines'* is used to calculate the body surface area burnt in an adult. This does not apply to infants whose body proportions are different from adults. Recently, computer-based softwares have been introduced with colour coded calculation and instant resuscitation guide (Fig. 21.4C).[26]



Relative percentage of body surface area (% BSA) affected by growth

Body parts	Ages				
	0 year	1 year	5 years	10 years	15 years
a = 1/2 of head	9½	8½	6½	5½	4½
b = 1/2 of 1 thigh	2¾	3¼	4	4¼	4½
c = 1/2 of 1 lower leg	2½	2½	2¾	3	3¼

Figs 21.4A and B: (A) Rule of nines (for adults) and (B) Lund-Browder chart (for children) for estimating extent of burns (Relative percentage of body surface area–% BSA) (*Courtesy:* Artz CP, JA Moncrief: The Treatment of Burns (2nd edn). Philadelphia, WB Saunders Company, 1969)[25]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[print] [help] [close]
Restart
Delete
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note about the burn...
Calculate
Change
open
save
Text
Partial
Deep
Amputate
autograft
donor
Applet Sage221 started

Sage II © 1987-2005

[medical center]

Patient Name: test patient
Patient ID: 00000000
Age: 21
Kilos: 75
Ht.(cm): 183
Image:n/a
User: john d.
Apr 15, 2005 1:22:08 AM

Body cm^2: 19658

	Antr%	Postr%
Head	-----	-----
Neck	-----	-----
Lt. Arm	01.99	02.01
Lt. Forearm	01.49	01.50
Lt. Hand	01.25	01.25
Rt. Arm	-----	-----
Rt. Forearm	-----	-----
Rt. Hand	-----	-----
Trunk	01.59	01.50
Lt. Thigh	-----	-----
Lt. Leg	-----	-----
Lt. Foot	-----	-----
Rt. Thigh	-----	-----
Rt. Leg	-----	-----
Rt. Foot	-----	-----
Perineum	-----	
Lt. Buttock		-----
Rt. Buttock		-----

	% area	cm^2
tbsa	**12.58**	02473
prtl	10.06	01978
dp	02.52	00495
amp	****	
auto	00.00	
dnr	****	

Parkland fluids: 4.00 -ml/kg/%

Fig. 21.4C: Sample surface area graphic evaluation (SAGE) software method (*Courtesy:* Estimating burn area ICU room pearls, Archive of www.icuroom.net Issue)[26]

Table 21.5: Percentage of body surface area involved in burns *(Rule of nine)*

Anatomic areas	*Percentage of body surface*
Head and neck	09
Right upper limb (Arm)	09
Left upper limb (Arm)	09
Right lower limb (leg) (Front and back)	(9+9)
Left lower limb (leg) (Front and back)	(9+9)
Anterior trunk (Thorox and abdomen)	(9+9)
Posterior trunk (Thorox and abdomen)	(9+9)
External genitalia/perineum	01

- Site of the body—burns of the trunk, lower abdomen, genitalia are fatal
- Age of the patient—aged people are more prone to fatality than children and adults
- Sex of the patient—sensitive nervous women are more susceptible to fatality than strong women and men.

Causes of Death in Burns

Death usually occurs either before 48 hours or after 48 hours, hence, the causes can be classified accordingly into two groups.[6-7]

Death Occurring within Few Hours

Victim may die due to shock, coma and asphyxia. Each of these is discussed individually below:

Shock

- Primary *(neurogenic)* due to—fear, severe pain, injury to vital organs leading to death within 24 to 48 hours
- Secondary *(vascular)* due to loss of serum from burnt area—developing depletion of blood volume and hypovolaemic shock, leading to death within 24 to 48 hours.

Coma

Coma due to congestion of brain and serious effusion into ventricles.

Asphyxia

Suffocation due to the inhalation of smoke or gasps of combustion. Asphyxia may also be caused by pressure on the chest due to *falling roof, beams, walls,* etc. when a house is on fire.

Death Occurring within Few Days

Victim may die due to inflammation of internal organs, gangrene, exhaustion, septic absorption, toxaemia, hepatorenal syndrome, etc. Each of these is discussed individually:

Inflammation of Internal Organs

These are inflammation leading to meningitis, peritonitis, pneumonia, bronchitis, pleurisy, enteritis, and *Curling's ulcer* in the duodenum.

Gangrene

Complications connected with the ulcers produced by burn such as gangrene, erysipelas, tetanus, profuse haemorrhage on separation of slough, etc.

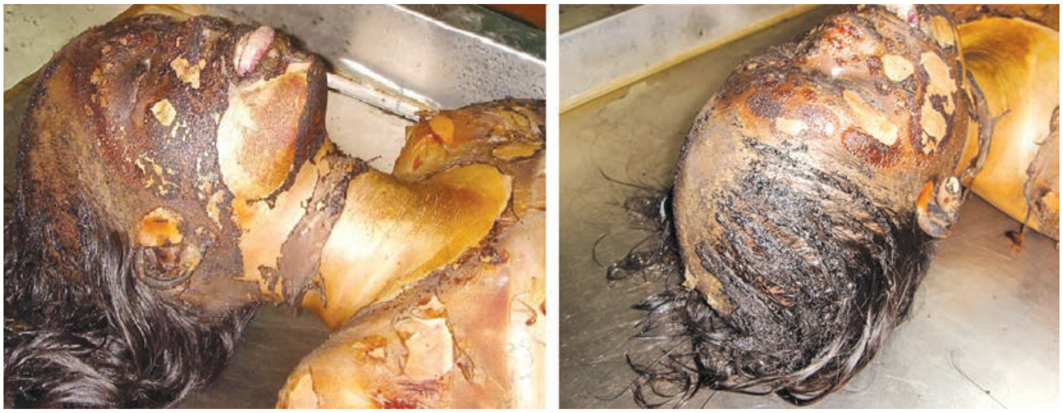

Fig. 21.5: A female victim of self-immolation by pouring kerosene on the body and lighting with matches. Swollen face, lips, singed scalp hairs, burnt eyebrows and eyelids, disfigured completely

Exhaustion

Exhaustion due to severe pain and dehydration from loss of fluid.

Septic Absorption

Septic absorption from excessive suppuration. Suppurative case death may occur by 5 to 6 weeks or even after a longer time. Pseudomonas is most common organism responsible for infection and sepsis in turn.

Toxaemia

Toxaemia occurs due to absorption of histamine formed as a result of combustion of tissue.

Hepatorenal Syndrome

In every case of burns of any severity, absorption of altered protein occurs and this in turn leads to cellular damage to liver and kidneys (acute tubular necrosis). Heart may also undergo similar damage.

Postmortem Appearances of Burns[1-9]

In any case of burns postmortem signs of burns will be present even if burns are postmortem in nature, but signs of antemortem burns suggest death due to burns.

External

Clothing It should be removed carefully and examined for the presence of kerosene, petrol and other such inflammable and combustible substances.

Any other articles Such as keys, metallic rings, ornaments, etc., worn on the body should be removed and preserved. It may be useful in establishing identity.

Face Usually distorted, swollen with tongue protruded out (Fig. 21.5).

Skin Findings observed vary according to the nature of the substance used to produce burns:

- Radiant heat-whitish (Fig. 21.6)
- Highly heated solid objects when:
  - *Applied momentarily:* Blister and reddening corresponding to the shape and size of the material used.
  - *Prolonged application*: Roasting and charring.
- *Explosions in coal mines or by gun-powder*—blackening and tattooing of the parts.
- *Kerosene oil burns:* characteristic odor and sooty blackening of the parts (See Figs 21.5 and 21.6). However, all antemortem burns skin will show line of redness (hyperemia) which is a sign of vital reaction (Fig. 21.7).
- Degloving / destocking may be seen due to cuticular peeling (Fig. 21.8).

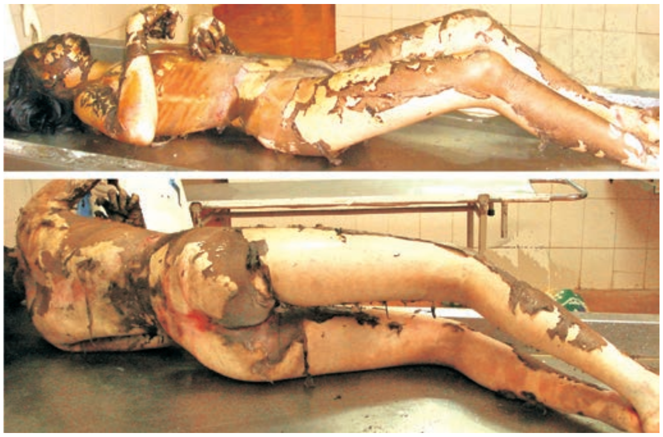

Fig. 21.6: Entire body of the same victim (as in Fig. 21.5) in *pugilistic attitude* with 70% of skin burns

Hair They undergo a peculiar effect of heat called *singeing*. The singed hair looks curly and is highly fragile. Cut section shows presence of plenty of vacuoles within microscopically.

Pugilistic attitude (Boxer's attitude, Fencing attitude)—It is a condition wherein the body assumes a rigid position with the limbs flexed and resembles a boxer in defending position[3-9] (Fig. 21.9).

- *Appearance:* All the four limbs are flexed with closed fist, body is bent forward and skin is tense, leathery, hard and frequently shows splitting
- *Causes:* Under the effect of heat, the muscle proteins coagulate causing them to become contracted
- *Medicolegal importance:* It could be due to antemortem or postmortem burns, especially if the body is burnt, charred and black. The pugilistic attitude can be mistaken for a pre-death attempt to shield oneself from an attacker.[23]

Cracks and fissures resembling incised wound may be seen in line with blood vessels exposed through them.

Charring of the body depends on degree of postmortem burns or burning of the body after death.

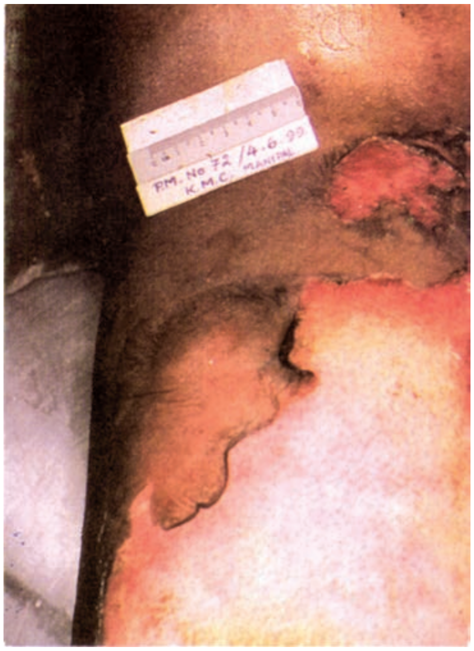

Fig. 21.7: Line of redness and vital signs of the burns wounds at it margins over the front of the left thigh, suggestive of antemortem burns note—vesication on the inner aspect of the thigh

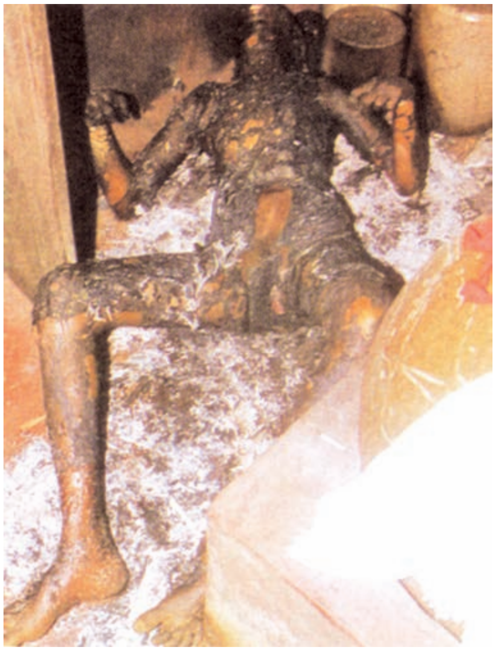

Fig. 21.9: Pugilistic attitude in a burns victim

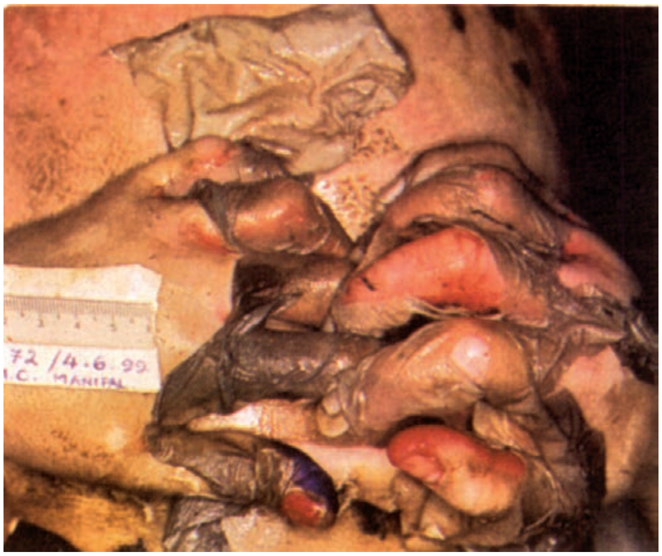

Fig. 21.8: Degloving of cuticle of both hands in same victim as in Figure 21.5

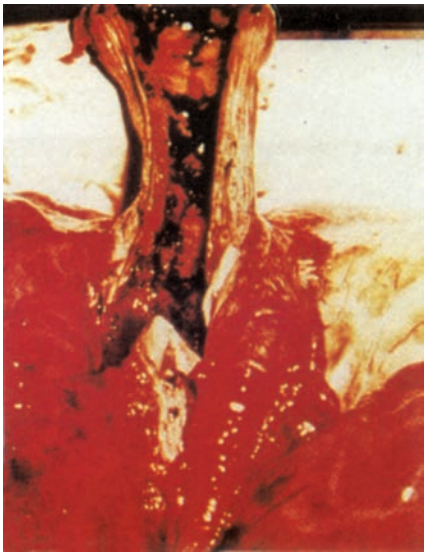

Fig. 21.10: Soot particles in the upper respiratory tract

Internal

- *Skull bones*—may be fractured and burst open due to intense heat, along the skull sutures
- *Brain and meninges*
  - Congested
  - Blood is usually extravasated imparting a brick red colour on upper surface of dura mater (heat haematoma)
  - Brain is sometimes shrunken.
- *Larynx, trachea and bronchial tubes*— contain carbon and soot particles (Fig. 21.10) and mucosa is congested with frothy mucous secretions. This is suggestive of antemortem burns due to inhalation of gases resulting in suffocation and asphyxia
- *Pleura*—congested and inflamed with serous effusion
- *Lungs*—congested and oedematous
- *Heart*—chamber full of blood, cherry red in colour due to inhalation of carbon monoxide
- *Stomach and intestines*—stomach may contain carbon impregnated mucous membrane. It may be red. There may be inflammation and ulceration of Peyer's patches and solitary glands of intestine. Ulceration may be sometimes found in duodenum known as *Curling's ulcer.* This is due to the liberation of some irritating substances in liver which cause thrombosis of small vessels only when victim survives for 7-10 days.[3-9]
- *Spleen*—enlarged and softened
- *Liver*—cloudy swellings and necrosis of the cells if death is delayed
- *Kidneys*—show signs of nephritis. Straight tubules are filled with debris of blood cells giving reddish brown marking.

Table 21.6: Differences between antemortem and postmortem burns

Characteristics	*Antemortem burns*	*Postmortem burns*
Line of redness+vital sign	Present (See Fig. 21.7)	Absent
Vesication	Present and true, contains serous fluid	Present but are false, contains PM gases
Reparative process	Present	Absent
Internal organs	Congested	Usually roasted+emits peculiar odour
Blood	Cherry red due to CO	Not so
Curling's ulcer	Present	Absent
Inflamed Payer's patches	Present	Absent
Carbon/soot particles in trachea, bronchus	Present	Absent

Medicolegal Importance

1. *Identification of the deceased*—Though identification of the deceased is difficult when the body is completely burnt, following may be helpful:
   - Metallic objects on the body like rings, bangles, keys, etc.
   - Sex of the deceased: Prostate and nulli-parous uterus will not get burnt even at very high temperature and could help in sex identity.
   - Age of the deceased: Usually established by the teeth and ossification of the bones.
2. *Whether the burns are antemortem or postmortem?* Table 21.6 enumerates these differences.
3. *Whether the burns are the cause of death or not?* Following two factors confirm burns as cause of death:
   - Presence of carbonaceous or soot particles in the respiratory tract mixed with mucoid secretions.
   - Cherry red discolouration of blood due to carboxyhaemoglobin.
4. *Whether the burns are suicidal, accidental or homicidal?*
   - *Suicidal burns* are common among Indian women. They pour kerosene oil and set fire to themselves. Some women stuff clothes inside the mouth also to prevent shouting and being heard by others.
   - *Accidental burns* are common among children and elderly people. Accidental kerosene stove bursting is also reported often.
   - *Homicidal burns* are quite common in India. The pernicious customs of dowry among certain Hindu castes, sometimes leads to young maidens, being murdered by pouring kerosene and set on fire by husband or in-laws (later claimed to be accidental burns death). This has lead to the concept of dowry deaths or bride burning which has enforced a rule by the Home Ministry of India to involve a panel of two doctors in conducting the postmortem examination of married woman dying of burns or any other reasons within 7 years of marriage or if her age is less than 30 years at the time of death in suspicious circumstances (IPC, Section 304B).[24,25]
5. *Self-inflicted burns for false accusation*—these burns are usually seen on accessible parts of the body.[6]
6. *Spontaneous combustion and preternatural combustion*—occasionally cases are reported of burns occurring due to the natural gases evolved in the intestine, *(inflammable gases such as hydrogen sulfide, methane, etc.)* When these gases are passed out per anally in a living person if they get ignited/come across a flame may lead to burns. Recently all these cases have been turned down considering them as *myth*.[6]
7. Dead body of victim may be burnt after death to conceal homicide. Head injury and fatal neck compression are commonly reported methods of homicide.

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Chapter

22

Electrocution, Lightning and Radiation

Electricity, lightning and radiation are also considered as physical agents that can result in to both nonfatal injuries and death. Recently these are included under *environmental emergencies*[1] and are dealt independently for better understanding of trauma produced.

ELECTROCUTION

Electrical injuries are relatively common, complex and potentially devastating form of trauma, both in industrial and domestic circumstances. The manifestations and severity of electrical trauma encompass a wide spectrum, ranging from a transient unpleasant sensation due to brief contact with low-intensity household current to instantaneous death and massive injury from high-voltage electrocution/lightning injury. Unlike thermal burns, electrical injuries commonly involve multiple body systems with the potential to pose difficult challenges regarding accurate assessment and proper management.[1-2]

Thus injury due to electricity may include burns to the skin and deeper tissues, cardiac rhythm disturbances and other associated injuries due to falls and other trauma. The amperage, voltage, type of current (AC vs DC) duration of contact, tissue resistance and current pathway through the body will determine the type and extent of injury. Higher voltage, greater current, longer contact and flow through the heart are associated with worse injury and worse outcome. In general, lightning exposure/contact may result in the most severe form of electrical injury.[2]

The passage of substantial electrical current through the tissues can cause skin lesions, organ damage and death. This injury is commonly considered as electrocution injuries. Fatalities are usually accidental, both in domestic and industrial environment.[2-13]

Electricity energy may be generated spontaneously in nature by lightning or artificially in the form of electric current. Electric current generated artificially are of two types, *direct* and *alternating*.[4-6,8-13]

Direct current (DC) Wherein the current flowing continuously in one direction is less dangerous (200-250 milliamp intensity of direct current is lethal). A high amperage DC (above 4 A) may even cause an arrhythmic heart to revert to sinus rhythm, as in medical defibrillation using defibrillator.

Alternating current (AC) Wherein the current shows rapid alteration in direction of flow. Alternating current is more dangerous than direct current (70-80 milliamp intensity of alternating current is lethal). This is because of the "*hold on*" effect it imparts making the muscles undergo a tetanoid spasm which prevents the victim from releasing the live conductor. It is also much more likely to cause ventricular fibrillations and arrest.

In fatal electrocution often three major events may occur (Fig. 22.1), which are a threat to life and are as follows:

- The most common is the passage of current across the heart, usually when a hand is brought into contact with live conductor, and the body is opposite to the hand. It has been claimed that the most dangerous is contact with the right hand and exit through feet, as this causes the current to pass obliquely along the axis of heart. The fatal process is cardiac arrhythmia, usually a ventricular fibrillation ending in asystole
- Less often, the passage of a current across the chest and abdomen may lead to respiratory paralysis from spasm of intercostal muscles and diaphragm

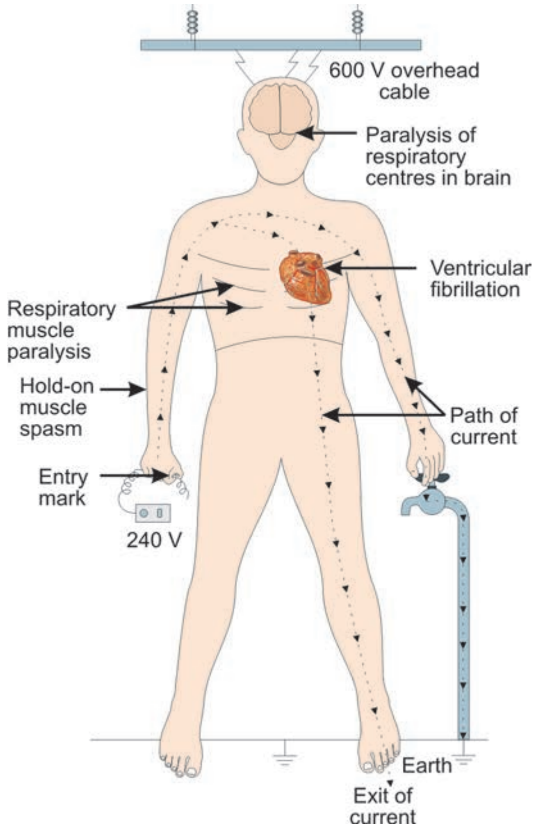


Fig. 22.1: Pathways of current in electrocution

- Rarely, current passes through the head and neck, usually in circumstances when the head of a worker on overhead power lines comes into contact with conductor. In such instances, there may be a direct effect on the brain stem so that cardiac or respiratory centres are paralysed.

It is commonly said that tolerance can be gained to electric shock and the professional electricians often work with live conductors with impunity. It seems more likely that expectation of a shock decreases sensitivity, but only for brief contacts, less than would be required for physiological or structural damage.

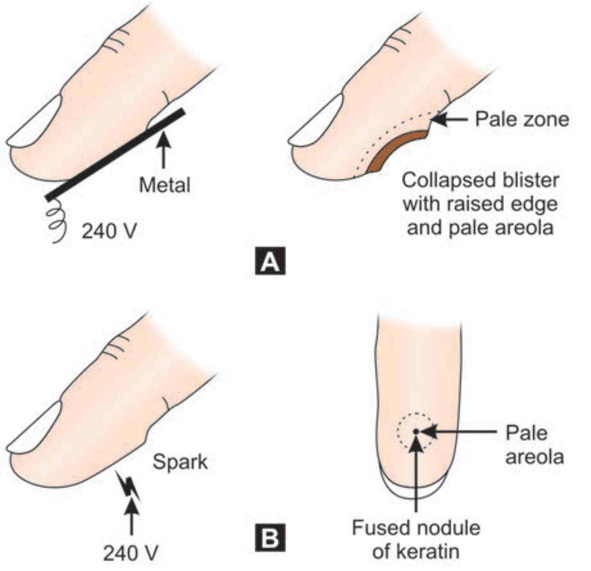
Effect due to Passage of Electricity

Effects of electric current in the human body are of two types:[3,5,6,8-13,16]

i. Local effects and
ii. Constitutional effects.

Local Effects

- *Burns and blisters*: Characteristically these are seen in the skin and referred to as cutaneous electrical mark. A *Joule burn is* the more popular terminology designated to this, while it is also known as *electrical burns* or *electrical marks* (Figs 22.2A and B). *Joule burn* is an endogenous thermal burn, i.e. it is due to the heat generated within the body during the flow of electric current.[16] It differs from exogenous burn, where the burn is caused by *sparking*; wherein the source of heat is outside the body.[6,8,16] If the skin is touching the conductor wire/faulty electrical appliance is moist, it may not show any electrical burns/marks/joule burns; while thick and dry skin will show a well circumscribed electrical burns/marks.[16,17] The lesion is seen as puckering of skin around the edges of the electrical burns without any red line surrounding the burns or reddening of the base at the *point of entry* of electric current (hands, fingers) (Fig. 22.2C) and *point of exit* (opposite hand or feet) (Fig. 22.2D). As with many other injuries shape of the object causing electrocution may some times be noticed, constituting *patterned electric mark*.[8] This finding may be helpful in reconstruction of the injury events and giving final opinion on cause of death in an unwitnessed electrocution deaths/ in situation of *obscure/ negative autopsy cases*.
- *Contusions and lacerations*: The wound may also be lacerated, and punctured with contusions at its margins. The point of exit of current or the '*earth*' takes place through the bare sole of the foot. Sole of the foot may turn hard and thick and even be ruptured giving a *deep laceration like appearance*.[11] Singeing of hair and burning of clothes may also be noticed at the location.
- *Metallisation of electrical marks*: When current passes from a metal conductor into the body, a form of electrolysis occurs so that metallic ions are embedded in the skin and even the subcutaneous tissues. This can occur with both AC and DC because of the combination of metallic ions with tissue anions to form metallic salts. These are though invisible to naked eye, may be detected through chemical, histochemical and spectrographic techniques. They persist for few weeks when alive and resist a moderate amount of post mortem change when dead.[8,20] Electron microscopy has recently visualised these metallisation as tiny globules of molten metal on the skin at and near electric marks.[8,21]
- *Micropathological skin changes at electrocution site:* Basically it is an *electrical burn*. These local lesions are usually found



Figs 22.2A and B: *Firm contact:* (A) Electrical marks, (B) Spark burn across air gap

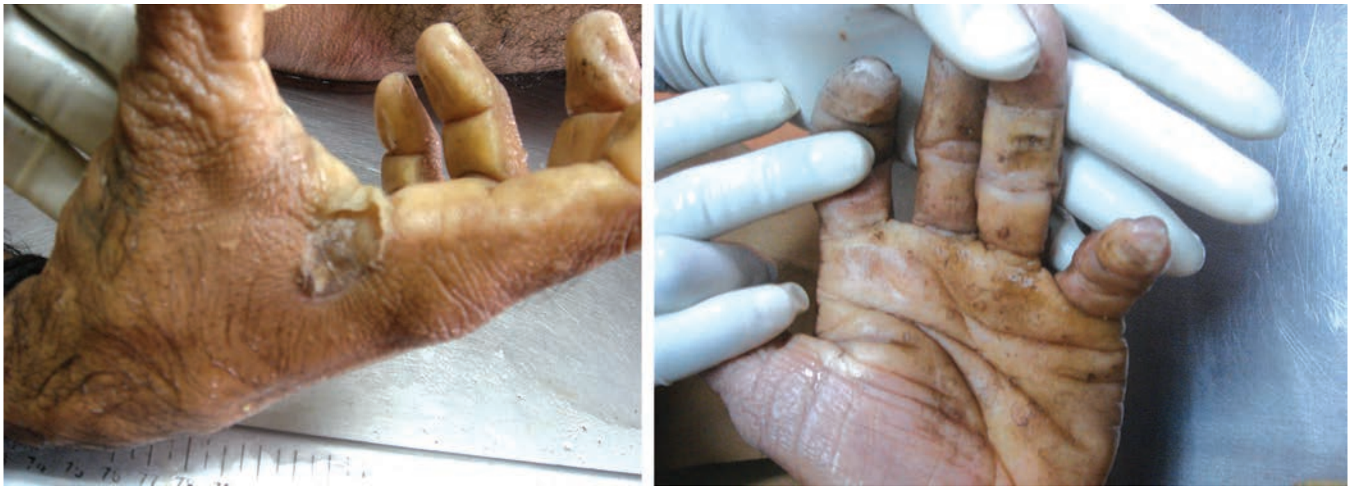


Fig. 22.2C: *Joul burn/electrical burns mark/electrical mark: Note:* A well circumscribed mark with puckering of skin around but no red line around burns mark/at its base-point of entry, suggestive of point of entry (*Courtesy:* Dr B Suresh Kumar Shetty, Assoc. Prof. of Forensic Medicine, KMC, Mangalore)

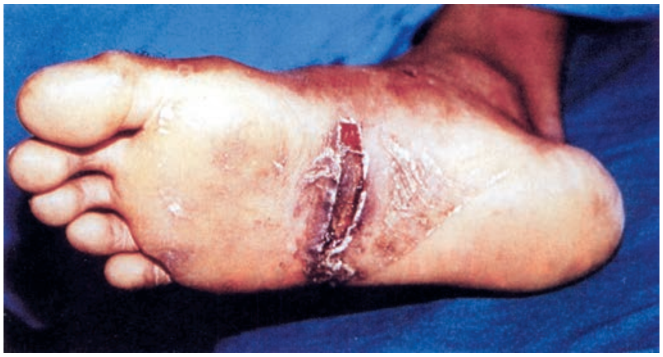


Fig. 22.2D: Sole of the foot showing lacerated wound due to exit of current

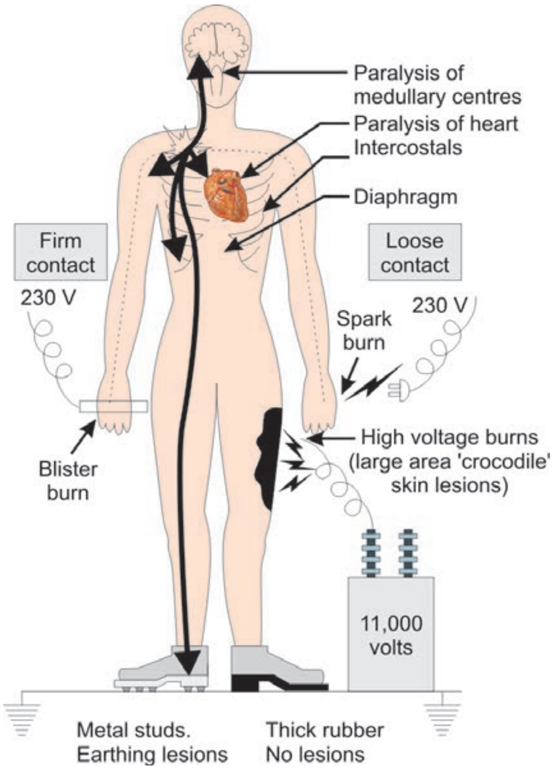
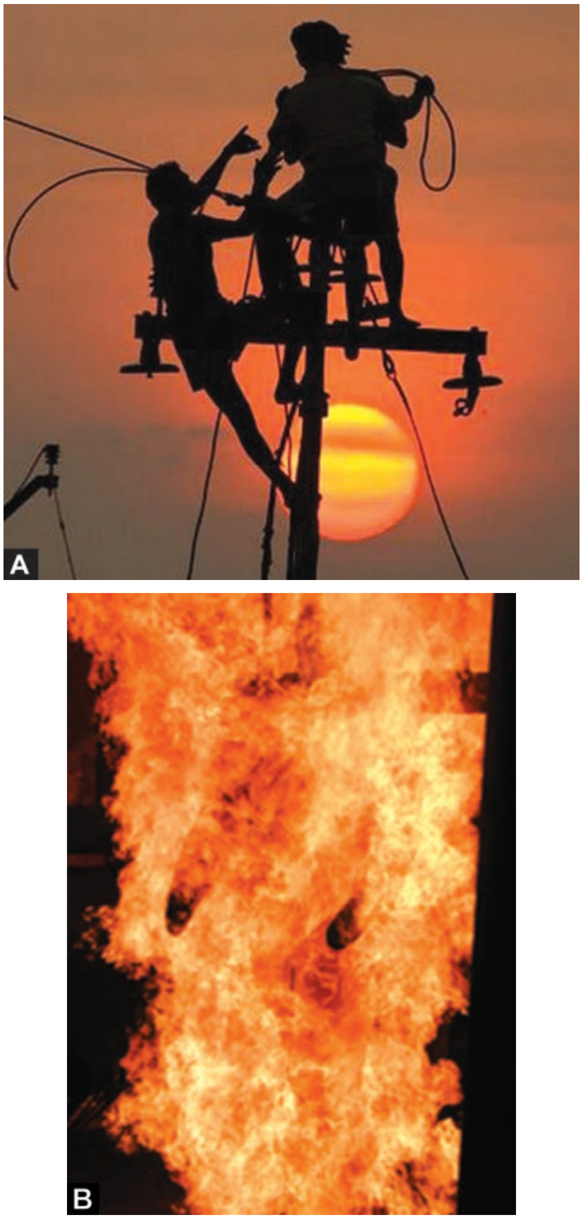

Paralysis of medullary centres
Paralysis of heart
Intercostals
Diaphragm
Firm contact
230 V
Loose contact
230 V
Spark burn
Blister burn
High voltage burns (large area 'crocodile' skin lesions)
11,000 volts
Metal studs. Earthing lesions
Thick rubber No lesions

Fig. 22.3: Diagrammatic representation or features of electrocution



Figs 22.4A and B: (A) Working on power line on an electrical pole; (B) Electrical high voltage flash fire

in the hands or fingers at the points of entry and exit of electric current, which are more severe and are observed mostly over feet or opposite hands (Fig. 22.3). Following changes are usually observed at these sites microscopically.[6,8,17,19-21]

— *Compression of the horny layer*—into homogenous plaque and ironing out of the underlying papillary processes.

— *Fissures and hollows* may appear between the stratum corneum and germinativum.

— *Basal cells changes*—are the surest sign of electrocution and the coalescence of basal cells into a star-shaped or rod-like structure in each group of the rete malphigii occurs.

— *Charring and vacuolation*—in the deeper cells of epidermis and dermis, formed by gas spaces from heated tissue fluids splitting the cells apart.

— *Metallisation in the skin* – with occasional deposition of fine metallic particles of the conductor substance may also be seen in a few cells.[8,20,21]

— *Epidermal nuclei* are pyknotic, elongated and aligned in a parallel or palisading fashion, often referred as "*nuclear streaming*".

- *Flash or spark burns*—High tension currents can jump several millimeters across air and cause lesions. It is estimated that in dry air an electric current of 100 kV can jump up to 35 mm. Such high tension currents can produce extremely high temperatures (up to 4000°C), just like the spark plug of petrol engine. This intense heat, which may result from flash of electricity, produces burns (Figs 22.4A and B), which resemble thermal burns over large area, and can cause the *keratin* of the skin to melt over multiple small areas. This molten keratin over these area fuses into multiple hard brownish nodules on cooling and resembles skin of the crocodile. Thus, the terminology '*crocodile skin*' (Fig. 22.4C) lesion was coined.[3-13,16] Sometimes, multiple lesions (Figs 22.4D and E) are found in the region of excess flexion of a limb where the current has passed across the joints instead of passing around it.

Constitutional Effects

Victim may suffer from the constitutional effects which comprise of the following:[22-27]

- *Momentary shock* with complete recovery if the current is small.
- Get *stunned* and/may go into *suspended animation* like state and/suffer from hemiplegia/paraplegia/loss of sight/loss of hearing/loss of speech, etc if alive.
- Immediate death when the current flown in is lethal. However, the alternating current with moist clothes, bad health, state of anxiety, etc. may aggravate these effects.

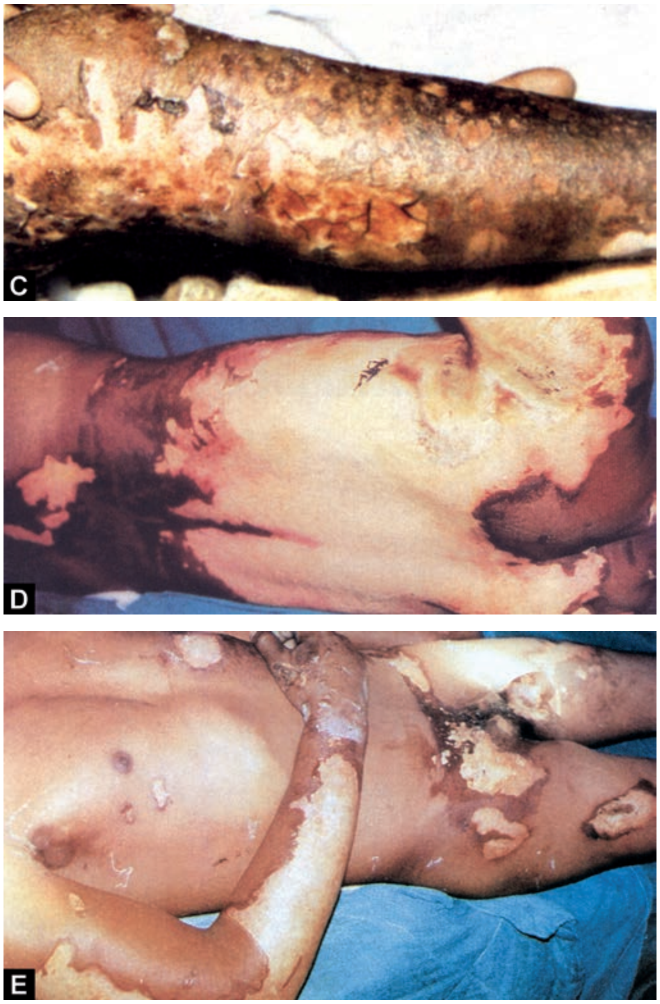
Causes of Death

As already stated most of the deaths in electrocution are due to *ventricular fibrillation* (in low voltage current) ending in *cardiac arrest*. This is caused by passage of current through myocardium, especially in the superficial epicardial layers and possibly across the endocardium. The current has a profound effect directly upon the myocardial syncytium, the possible dislocation of the pace making nodes and conducting system being ill-understood. A recent study of cardiac pathology among the victims of death due to electrocution, reported that the frequency of MFB (myofibre break-up) is maximal histopathologically in cases of electrocution deaths (90%) and the finding of MFB is considered as an *ante-mortem change* and declared as a distinct finding in all cases electrocution.[18] The term myofibre break-up (MFB) includes the following histological patterns[19] (Figs 22.5A to C).

a. Bundles of distended myocardial cells alternating with hyper-contracted cells. In the latter group of cells, there is also widening or rupture (segmentation) of the intercalated discs. Myocardial nuclei in the hyper-contracted cells have a "square" aspect rather than the ovoid morphology seen in the distended myocytes.
b. Hyper-contracted myocytes alternated with hyperdistended cells that are often divided by a widened disk.
c. Non-eosinophilic bands of hyper-contracted sarcomeres alternating with stretched, often apparently separated sarcomeres.

This observation is extremely helpful in establishing cause of death in obscure/negative autopsy cases wherein opinion as to cause of death is difficult for want of gross findings at autopsy examination in spite of positive history of electrocution. Among the other causes of death in electrocution inhibition of respiratory centre (in high voltage), which is though second common cause, is far less common. Here when the current passes through the thoracic cage, it causes the intercostal muscles and diaphragm to go into spasm, or become paralysed. In either case however, respiratory movements are inhibited and a congestive-hypoxic death results. The brain stem is rarely affected in electrocution, when the current enters through the head. Either cardiac arrest or respiratory paralysis can then supervene.

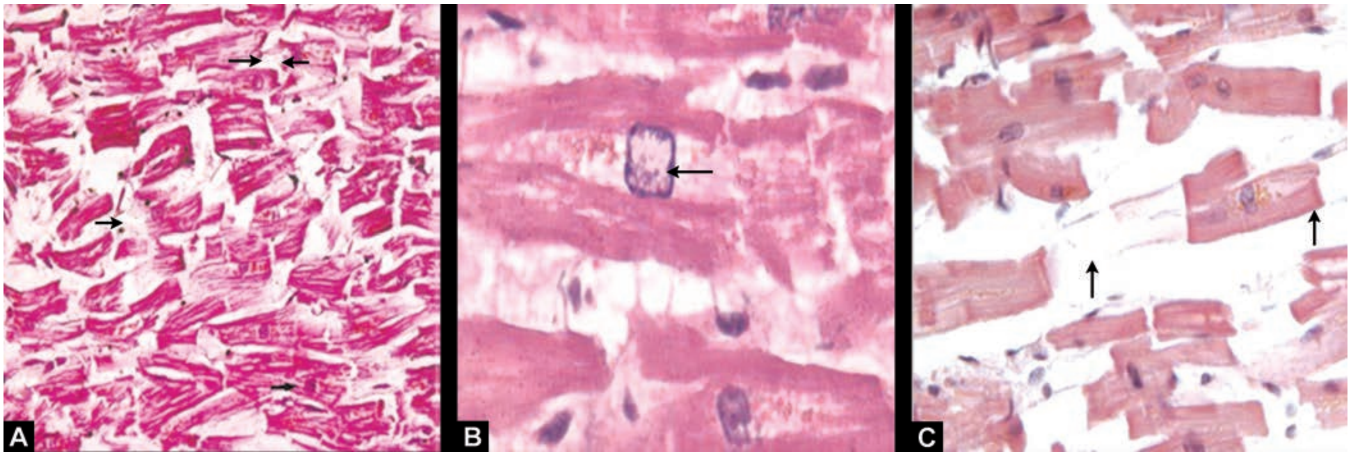
Finally, it may also be remembered here that non-electrical causes such as fall/being thrown from height resulting in associated injuries, more often result in fatality in a victim of electrocution.



Figs 22.4C to E: *High voltage burns:* (C) the leg, involves large area and called crocodile skin lesion (*Courtesy:* Dr IG Ghosh, Former Prof. and HOD, Forensic Medicine, IG Medical College, Simla), (D) on the trunk, (E) over the right forearm, thighs, etc. (*Courtesy:* Dr Mitha Prasanna, Former Prof and HOD, Burns and Plastic Surgery Unit, Kasturba Hospital, Manipal)

Medicolegal Aspects

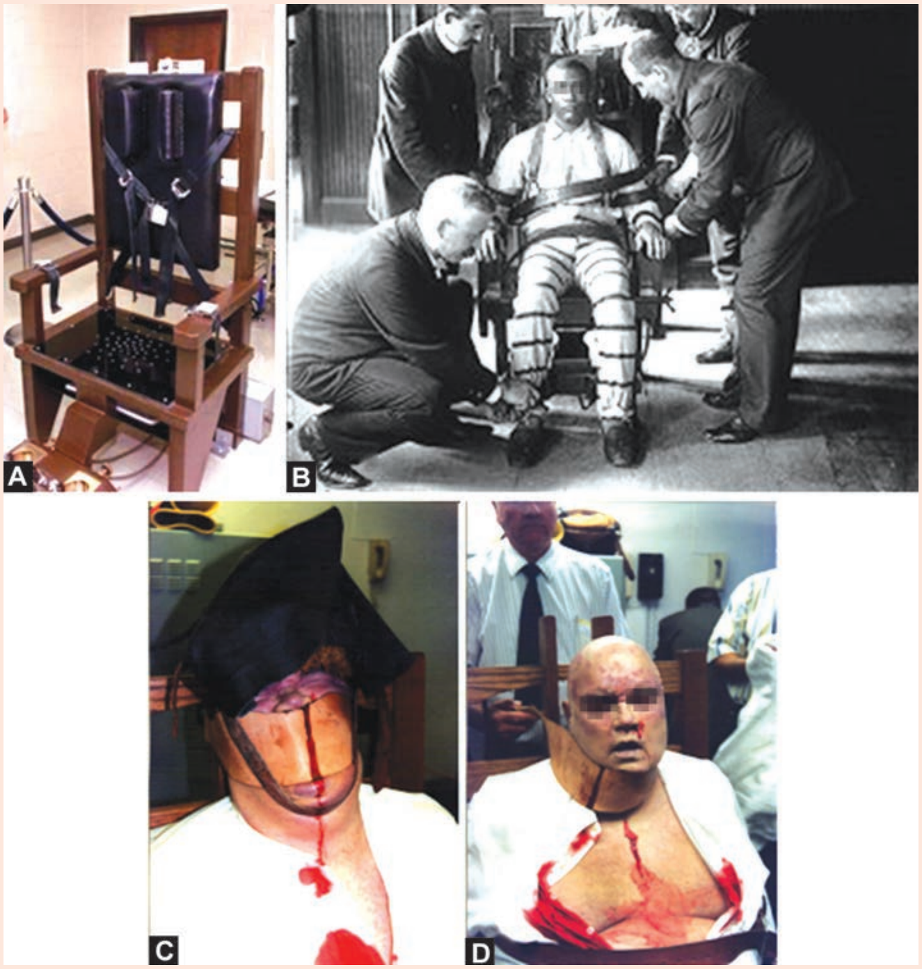
- Death by electric current is usually *accidental* but cases of *suicide* and *homicide* have also been recorded.[13-17] Bathroom is a common site for electrical tragedies. Accidents, suicides and even homicides occur there because of its vulnerability to electric shock.[8] Suicides from electricity have increased recently especially in Germany.[14] Though homicide is rare, it is recorded in United States.[15] Again in United States, electricity has become a mode of judicial execution.[15,16]
- Judicial electrocution – death penalty is carried out in the electric chair, in some of the states, namely: Alabama, Florida, Georgia, Kentucky, Nebraska and Tennessee, in the USA (Figs 22.6A to D). The condemned person is strapped to a wooden chair, and one electrode is put on the shaven scalp (in the form of a helmet) and the other on the right lower leg and current is passed with initial burst of 2000-2400 volts, for a short time (seven seconds) of one minute through the body, which makes the person lose consciousness immediately. It is the second surge, of lower intensity of 500-600 volts for a longer duration



Figs 22.5A to C: *Histopathological pictures presenting MFB:* (A) Bundles of hyper-contracted myocytes (arrows) alternated with bundles of hyper-distended myocardial cells (trichrome stain 100); (B) Square nucleus expression of contraction (arrow) (H&E 630); (C) Separation of sarcomeres (arrows) in myofibres connected with contracted ones (H&E × 250)[18,19]

Execution by Electrocution in Electric Chair

Electrocution was first introduced in New York in 1888 as a more *humane* method of execution then hanging. But there have been horrific instances of inmates catching on fire, multiple jolts being needed to kill, and bones being broken by convulsing limbs, etc. In USA the *electric chair* is an option for those who committed crimes before 1999, when *lethal injection* became the state's primary method of execution. Since the US Supreme Court reinstated the death penalty by electrocution in 1976, there have been several inmates executed in the electric chair, most recent being the condemmed murderer in Virginia in July 2006.



Figs 22.6A to D: *Excution by electric chair:* (A) Tennessee's electric chair; (B) The condemned is first strapped to the wooden electric chair. Note one of the electrode fixed to the right lower leg; (C) Showing another criminal executed by electrocution: Note other electrodes fixed to the shaven scalp in the from of a helmet and the electric current is passed; (D) After the excution (*Source:* http://www.smh.com.au, Sunday Morning Herald, Tennessee, September 12, 2007-5:19PM, Retrieved: 16.5.09)

(17 seconds to one minute) which actually kills the victim. The process is repeated. After five minutes, a physician checks the heart beat. If the heart is still beating, the voltage is delivered again. The first person to be electrocuted was *William Kemmler*, in the New York's Auburn Prison, on August 6th, 1890. Underneath the electrodes the skin temperature may rise as high as 60°C. The temperature of brain in such cases also rises to similar levels. Histopathologically rupture of neuroaxons and blood vessels can be demonstrated in the brain. Right leg goes into cadaveric spasm immediately. Often ejaculation of semen may take place at death.

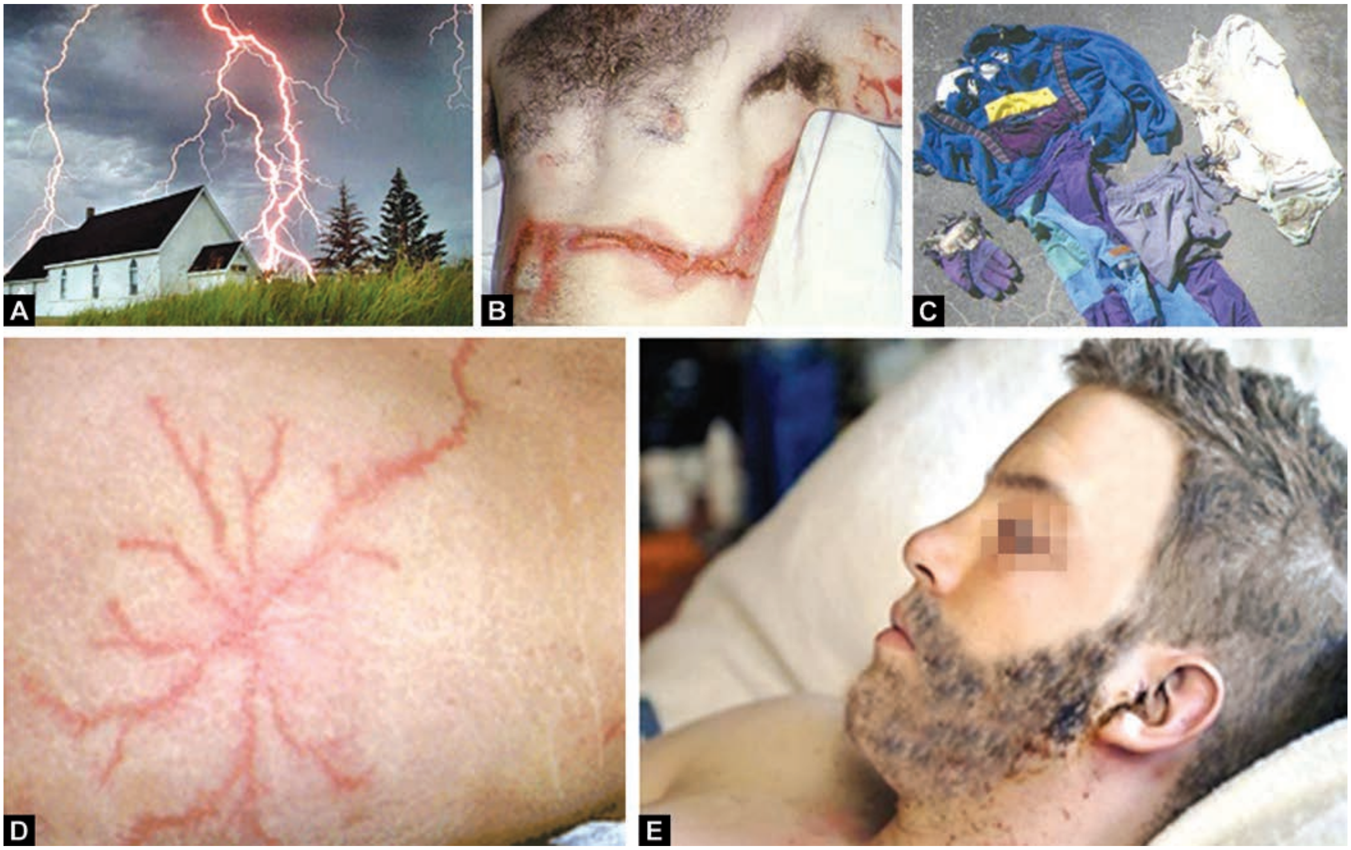
- In cases of electrocution with wet body surface—no positive findings may be present and autopsy in such cases may be an obscure one (*refer above cause of death*).

LIGHTNING

Lightning differs only in degrees from ordinary electric currents. A lightning bolt is produced when the charged undersurface of a thundercloud discharges its electrical charge to the ground. Since the under surface of the cloud is usually negatively charged, virtually all discharges are negative. Approximately 5 per cent of the lightning flashes, however, are positive charges. These are most frequent in mountain regions.[1-12] A flash of lightning (Fig. 22.7A) from a thundercloud to the earth can pass *direct current* of enormous potential (1000 million Volts or more). Along the track of the current much energy is liberated most of which is converted into heat.[12,13,16]

Four electrical mechanisms of lightning injury (Figs 22.7B to D) have been described: *direct strike, contact, side flash, and ground current.*[22] *Mechanical injury* may occur if the person falls or is thrown by muscle contraction.[22] Added to these four, is the *fifth mechanism* of injury by upward *streamer* such as a flag, banner, bunting, etc. as the cause.[22,23,29-31] These mechanisms are discussed briefly:

- *Direct strike*—The terminology is self explanatory and here the lightning bolt hits the victim directly.
- *Contact/Conduction through another object*—The terminology means the victim making contact with another object which is hit by lightning and thus getting the injuries rather indirectly.[22]
- *A side flash*[12,22]—In a side-flash strike, the bolt of lighting hits an object, such as a tree, and then jumps from the object, striking the individual nearby. In a direct strike or a side-flash strike where the individual is relatively close to the object from which the bolt jumps, the current can spread over the surface of the body, enter it, or follow from both routes. In the majority of cases seen by the autopsy surgeon, the current has both spread over the surface of the body as well as entered. In such cases it is quite common to find the clothing torn, shoes burst, hairs seared (singed), burns on the skin due to metal zipper and other metal objects heated by the lightning, and burns of entrance and exit of



Figs 22.7A to E: *Lightning and Lightning injuries:* (A) Lightning hits beyond a church building, making a severe electrical storm made its way across southern Manitoba, July 1995; (*Source:* Marc Gallant, www.cbc.ca/.../story/2008/06/20/f-lightning.htm1) (B) Lightning injuries lesions (burns sustained over the trunk at points of lightning entry) (*Source:* http://pagesperso-orange.fr/dmtmcham/jpeg/trajet.jpg); (C) Torn cloths of lightning victim; (D) Fernlike patterns are classic called as — Arborecent marks/Lichtenberg bodies/Filigree burns etc. (E) Lightning injury over left ear and left side of the face struck by lightning while listening to i-pod music. (*Source:* http://www.labnol.org/assets/images/Photograph When Lightning Strikes PeopleList _FEE5/ipodburnears.jpg)

current.[12,16,17] Rupture of tympanic membrane is not uncommon.[12] A lightning victim found on the road with torn clothing, burst shoes, etc and other injuries have many a times been misinterpreted as hit and run victim.[8-12]

- *Ground current*—Once the electrical energy that is generated with lightning has flown into the body of a victim, who has been struck by the lightning, it will move towards the ground/ earth and this results in injuries.
- *Fifth mechanism*—This *fifth mechanism* of lightning injury may aid in the investigation of deaths and injuries when previously described mechanisms of lightning injury cannot be implicated. Thunderclouds can drag charge inside them as well as underneath them. Cloud-to-ground lightning approaches the earth in *jagged branched steps* about 30-90 meters in length.[32] When the tip of any branch gets within a few hundred meters of the ground, the electric field becomes very large, inducing charges to begin at the ground and surge through any *object projecting above the ground*, including *people*.[32,33] An example for this would be a lighting bolt hitting a tallest crane/ which in turn will let the electricity flow down its metal structure and strike the worker touching it. The injury produced on the victim will be same as if the crane had hit a high-power electrical line, resulting in burns at the entrance and exit points, which are often multiple and severe.[12]

However, it may be noted here that, not all *upward streamers* get connected with *downward leaders* to complete lightning channels. Uman MA, suggests that individuals can be injured by a *weak upward streamers*[34] also, which, may not be connected with *downward leaders*.[30] Darveniza M, adds the facts that these non-discharge currents could vary from 10 to 400 A, "*certainly enough to impair body sensors and functions*," but that "*the gross physical effects of such currents are likely to be small, because of their relatively small magnitude and because of their short duration*".[23]

Lightning While in an Automobile/Using Telephone/ I-pod Music System

If one is inside a metal vehicle, such as car, bus, truck or even a train, when struck by lightning, the probability of injury is extremely small.[12,22-24] A report on death or injury of an individual while using a telephone and the line, hit by lightning is though quite unusual, cannot be ruled out.[25] Another surprising report is on lightning injuries in the ear and the face while listening music in an i-pod (Fig. 22.7E)

Cause of Death in Lightning

Immediate death from lightning is usually caused by high-voltage direct current.[3-12, 22-24] Death in lightning is due to syncope/ cardiopulmonary arrest/electrothermal trauma/paralysis of nervous system. Death may also be delayed in lightning victims and is usually attributed to the complications of burns. If the electrocution is secondary to a close proximity point of impaction, survival of the victim is possible. However, it is a well established fact is that the most of the victims of lightning episode survive.[12]

Postmortem Features of Lightning Death

Includes findings on the *body* and findings on *clothing* worn. Body findings are described under external and internal findings.[24]

External

Ecchymosed burns of all degrees (usually caused by fused metallic substances).

- *An arborescent marking is* a fern-like injury, is also known as *filigree burns* or *lightning prints*, etc. on the skin are pathognomonic of lightning injury. It is a patterned area of transient erythema which appears within an hour of accident and gradually fades within 24 hours. It is reported to be caused by positive discharges over the skin.[24]
- It is said to occur when a person is stuck by a *negative lightning bolt* is then hit by a secondary positive flashover from a nearby grounded object. A second possibility is that it represents an entrance point in an individual struck by a positively charged lightning bolt.[8,12, 24] A third precedent explanation of the good past is that it is due to deposition of copper on the dermis and also said to be due to staining of tissues by haemoglobin from lysed red cells along the path of electric current.[4-13] These are not lesions due to burns (see Figs 22.5A to C). All these explanations, neither of which is exclusive of other, would explain the relative rarity of arborescent lesion in individuals struck by lightning.

Internal

- Congested membranes—often with laceration
- Intracranial and intracerebral effusion of blood
- Patchy hemorrhage on pleura and lung surface
- At times, severe disruption may cause widespread petechiae.

Findings on the Clothing

- Burnt clothes at the site of entry and exit of lightning. Clothing are usually torn, shoes burst.
- Fused metallic articles in the vicinity.

Medicolegal Aspects

Death by lightning is always *accidental*.[24] There is always a thunderstorm in the locality. There may be presence of fused metallic substances in the vicinity, absence of any wound in the body. Evidence of damage caused by lightning in the vicinity, may substantiate the circumstantial evidence in the diagnosis of accidental death by lightning.

RADIATION

Exposure to radiation can occur through two mechanisms:[3,28]

The First Mechanism is from a *strong radioactive source* such as *uranium*;

The Second Mechanism is *contamination by dust, debris and fluid containing radioactive material*.

Factors that Determine Severity of Exposure

- Duration of time exposure,
- Distance from radioactive source, and
- Shielding from radioactive exposure.

Types of Radiation Exposure

The three types of radiation exposure include *alpha, beta* and *gamma*. The most severe exposure is *gamma* (X-ray radiation).

Effects of Radiation

In general, radiation exposure does not present with any immediate side effects unless exposure is severe. Most commonly, serious medical problems occur years after the exposure. Acute symptoms include nausea, vomiting and malaise. Severe exposure may present with burns, severe illness and death (*beta* or *gamma*).

In the modern world people are exposed to radiation from various sources which can be classified as:

a. Ionising radiation
b. Non-ionising radiation, i.e. U-V rays, visible light, infrared rays, microwaves.

IONISING RADIATION (IR)

IR can produce radiation injury by *tearing* the atoms and molecules of a substance and there by damage the body. Thus, when it passes through a living cell, it can damage the cell by tearing apart the chemical make up of the cell. It gets injured badly, loses its ability to function and ultimately killed. Usually cells in tissues which are growing rapidly are highly sensitive to radiation. For example, bone marrow cells in the centre part of a bone are the fastest-growing structures in human body and thus they gets destroyed first, when exposed to ionising radiation. IR can come in the from of electomagnetic waves. IR is usually given off by the sun, X-ray machines and radioactive elements.

Sources of IR Injurious to Human Health

Humans are exposed to ionising radiation (IR) from a variety of sources. These sources fall into four general categories:

- Natural
- Intentional
- Accidental and
- Therapeutic.

Natural

Exposure to natural sources of IR account for a very fraction of radiation injuries. Natural sources include *sunlight* and *cosmic radiation*. Sunlight includes not only visible light, which has relatively few health effects, and radiation of higher frequency, such as ultraviolet radiation. Just stepping outdoors exposes a person to IR in sunlight. Cosmic rays are similar to sunlight in that they are always present around us. They are not visible, but they do contain *ionising radiation*.

Intentional Exposure

Intentional exposure to IR is uncommon, unusual and very rare. It occurs when nuclear weapons (hydrogen and atomic bombs) are used as weapons of war. This has occurred only twice in history, when the United States dropped atomic bombs on Hiroshima and Nagasaki, Japan, at the end of World War II. Many thousands of people were killed or injured by these attacks. They are the only people ever to have been injured by intentional exposure to IR.

Accidental Exposure

Accidental exposure occurs when a person is exposed to IR by mistake. For example:

- *Research laboratory spillage:* Radioactive elements are sometimes spilled in a research laboratory. Workers in the lab may be exposed to the IR from those elements.
- *Nuclear reactor accidents:* 1945 and 1987, there were 285 nuclear reactor accidents worldwide. More than fifteen hundred people were injured and sixty-four were killed in the se accidents. e.g. Chernobyl Nuclear Reactor accident victims are even today suffering from the after effect.

Therapeutic Exposure to IR

Occurs during various medical procedures. Radioactive elements and ionising radiation have many valuable applications in diagnosing and treating disorders. But those treatments can have harmful as well as beneficial effects on patients. The rate of radiation injuries due to this cause probably cannot be measured. Many people who may have been injured by a radiation treatment probably died of the condition for which they were being treated.

Medical—Diagnostic and therapeutic doses of radioactive (tracer) elements are given to the patients. Sometimes these are applied by external beam using radioactive cobalt (supervoltage therapy) for the treatment of cancers. Radiation in therapeutic doses is by and large harmless, but skin reactions at the site of supervoltage application are common. This at first presents with depletion and erythema of skin but later may produce blistering and discolouration of skin.

Industrial—In various industries especially in watch, drug and chemical analysis radioactive substances are used.

War—Nuclear weapons used by superpowers usually produce mechanical trauma, burns and radiation sickness due to ionising radiation

Action—The ions produced alter the chemical structure of various enzyme systems. Foetus and child are more susceptible. Hematological changes and disability are more likely with dose above 50 to 100 rads. Hemopo, etic cells, Payer's patches of small intestine, germinal epithelium of testis and cornea are highly sensitive to it as compared to musculoskeletal tissues.[35-40]

Medicolegal Aspect

- The doctor who is in charge of the patient's treatment has to be careful regarding application of *supervoltage therapy* as patient can sue him or her if the patient can prove negligence on the part of doctor.
- Autopsy in cases with amount of radioactivity more than 5 millicuries need extraprecautions like-use of rubber gloves, plastic aprons, spectacles and plastic shoe covers.

Burns due to Ultraviolet Rays

- These produce erythema and eczematous reaction.

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Chapter

23

Trauma in its Medicolegal View Points

Medicolegal aspects of injuries are often not taken heed by treating doctor, which may obviously lead to unnecessary legal litigation. The various ingredients of this major subdivision which the doctor must better be aware of are:

- IPC *(Indian Penal Code)* sections relevant to injuries
- Examination of the injured
- Complications of injuries
- Injuries of medicolegal importance

More emphasis is given to the former two subdivisions in the enumerated list as the latter two are already discussed in depth under relevant chapters (*see Chapters 16 to 19)*.

IPC SECTIONS RELEVANT TO TRAUMA

Injury (*Section 44, IPC*)

Section 44 defines injury.

Definition

As per this section, injury is defined as any harm whatsoever illegally caused to any person in body, mind, reputation or property.

Explanation

Thus, in the legal sense it is clear that injury can be caused by without touching the body. Causing mental agony, damaging the reputation of the person by making false allegation (*defamation* case), or causing damage/loss of property belonging to another person, etc. are also considered as injuries in law.

Hurt (*Section 319, IPC*)

Section 319 defines hurt.

Definition

Hurt is defined as causing bodily pain, disease or infirmity to a person.

Examples

Pulling hairs of another person to cause pain, transmitting syphilis to the sex partner, or mixing some deleterious substance with food, leading to infirmity (ill health) to the person consuming it, etc. are all examples of hurt.

Grievous Hurt (*Section 320, IPC*)

Section 320 defines grievous hurt. Grievous hurt is more serious kind of hurt and is a specific hurt, inflicted voluntarily to another person and comprise of any of the eight kinds (*clauses*) enumerated below.

Definition

Section 320 designates following list of eight grievous hurt:

- Clause 1 — Emasculation.
- Clause 2 — Permanent privation of sight of either eye.
- Clause 3 — Permanent privation of hearing of either ear.
- Clause 4 — Privation of any member or joint.
- Clause 5 — Destruction or permanent impairment of powers of any member or joint.
- Clause 6 — Permanent disfigurement of head or face.
- Clause 7 — Fracture or dislocation of bone or tooth.
- Clause 8 — Any hurt which endangers life or which causes the sufferer to be, during the period of 20 days, in severe bodily pain or unable to follow his or her ordinary pursuits.

Examples and Explanations

Emasculation—This means depriving a male, of masculine vigour. Accordingly castration, cutting away of penis, etc. constitute ideal examples.

Permanent privation of sight of either eye or hearing of either ear – To be considered as grievous hurt, the loss or privation of sight or hearing has to be permanent. Thus, injury which causes loss of vision due to fisting of the eye resulting in oedema, redness it cannot constitute the offence of grievous hurt, as the loss of vision with this injury is only of temporary nature. On the contrary, a forcible slap on the left side of face near the ear leading to permanent loss of hearing constitutes an ideal example for the offence of grievous hurt.

(Note: Permanent does not mean that, it should be incurable. For example, when the loss of sight is due to corneal opacity due to some injury over corneas, it is curable by corneoplasty. But, since corneal opacity due to scarring resulting from an injury is permanent by itself, it will be considered grievous hurt and chance of cure by corneoplasty does not minimize its gravity).

Privation of any member or joint – Privation of joint means cutting away of one limb or joint, which needs no explanation.

Destruction or permanent impairment of powers of any member or joint – This is self explanatory. However, any injury leading to impairment of powers of any joint or member form an ideal example to constitute this offence.

Permanent disfigurement of head or face – Accordingly cutting the nose, ears or a deep wound on the cheek leading to an ugly scar, etc. which brings about permanent disfigurement changes constitutes some of the examples. This means, minor injuries on the face do not come under this section.

Note: However, when we consider disfigurement factor, grievousness may not be same in all persons. An irregular small, permanent scar on the face of a young unmarried girl or a stage or cinema actress may be considered as grievous hurt, because this may affect her life and career as well as livelihood, most adversely. But, such a scar in an old woman may not be considered for the purpose of this offence to have disfigured her face as her face may be already having multiple creases and other scars due to aging.

Fracture or dislocation of bone or tooth: This is considered as grievous hurt (irrespective of size or extent), because it can cause great pain and suffering to the injured. Bone need not cut through and through or crack need not extend to the whole thickness of bone. Partial cut of the bone or fracture of outer table alone as with the fracture skull, come under this clause.

Note: However, dislocation of bone may not be a feature to persist for long but dislocation of tooth may retain the feature for considerably long period or may even be a permanent feature when the dislocated tooth falls off.

Eighth clause: Under this clause *hurt which endangers life,* meaning injury which may or may not be likely to cause death in ordinary course of nature, irrespective of whether treatment is given or not. As regards severe bodily pain, it is correct that, one or two bruises or abrasions may not be considered as grievous hurt. However, multiple bruises and abrasions involving excessive body surface may amount to grievous hurt, in a way causing severe bodily pain or even endangering the life.

Thus, only if the injury causes danger to life of the patient, it becomes grievous. The phrase "*unable to follow his ordinary pursuit*" for 20 days means the person is unable to go to the toilet by himself, taking bath himself, or taking food himself, for 20 days. Ordinary pursuits also mean those activities by which a person earns his livelihood (e.g. a taxi driver cannot earn his livelihood if another person has caused fracture of his upper limb intentionally). Thus to say one is suffering from grievous hurt, mere hospital stay for 20 days is not enough. It must be proved that during the stay, he was either in severe bodily pain or unable to follow his ordinary pursuits.

Punishment for Hurt and Grievous Hurt

Sections 323, 324, 325 and 326 IPC, describe the punishments for hurt and grievous hurt, are given below.

Section 323, IPC

Punishment for voluntarily causing hurt – imprisonment up to one year or with fine up to Rs. 1000 or both.

Section 324, IPC

Punishment for voluntarily causing hurt by dangerous weapons or means–imprisonment up to three years or fine or with both.

Section 325, IPC

Punishment for voluntarily causing grievous hurt –imprisonment up to seven years and fine.

Section 326, IPC

Punishment for voluntarily causing grievous hurt by dangerous weapons or means—life imprisonment or imprisonment upto for ten years and fine.

These two sections create the need for understanding the two terms–*dangerous weapon* and *means causing* hurt or grievous hurt.

Dangerous Weapon

Instruments used for shooting, stabbing or cutting or any other instrument which is used as a weapon of offense is likely to cause death, constitute dangerous weapons.

Means Causing Hurt or Grievous Hurt

Fire, heated substances, a poison or corrosives, or explosives, or any substance deleterious to the body to inhale, swallow, or received into the blood or by means of animal constitute means causing hurt or grievous hurt.

HOMICIDE

Homicide means causing the death of one person, by the act of another. Homicide is punishable under certain circumstances (*culpable homicide*) and not punishable under other circumstances (*excusable or justifiable*). Thus homicide can be *lawful* and *unlawful* (Fig. 23.1). Each of these is presented below with relevant IPC sections.

LAWFUL HOMICIDE

These homicides which are not punishable and also known as *simple homicide* are enumerated as follows:

- Homicide done by a person of unsound mind (*Section 84, IPC*)
- Homicide by a child below the age of 7 years (*Section 82, IPC*)
- Homicide due to an accident/misfortune (e.g.: firing into a bush thinking that there is a rabbit and accidentally shooting a human being instead) (*Section 80, IPC*)
- Homicide during private defence of body or property, e.g.: personal defence in order to prevent death or rape (*Section 100, IPC*), or private defence of property (*Section 103, IPC*) e.g. inn robbery.
- Homicide done as per the order of the court (*Judicial hanging*) (*Section 78, IPC*)

UNLAWFUL HOMICIDE

Culpable Homicide (Section 299, IPC)

This is also known as *culpable homicide not amounting to murder (Manslaughter in UK).*

Definition

Culpable homicide is defined as causing of death by doing an act:

- With an intention of causing death or
- With the intention of causing such bodily injury as is likely to cause death or
- With the knowledge that he is likely to cause death by such act.

Examples

- Immersing the head of a child under water column proves the intention to cause death.
- Firing a revolver aimed at the head of another person also proves the intention.
- Causing multiple injuries resulting in rib fractures, compound fracture of femur, rupture of one kidney, etc. shows that the intention to cause bodily injuries are likely to cause death.
- Over a trivial quarrel in dim light, A threw a knife at B. It pierced the chest and caused death of B. There was no attempt to cause any more injury. Here A had the knowledge that he is likely to cause death by his Act.

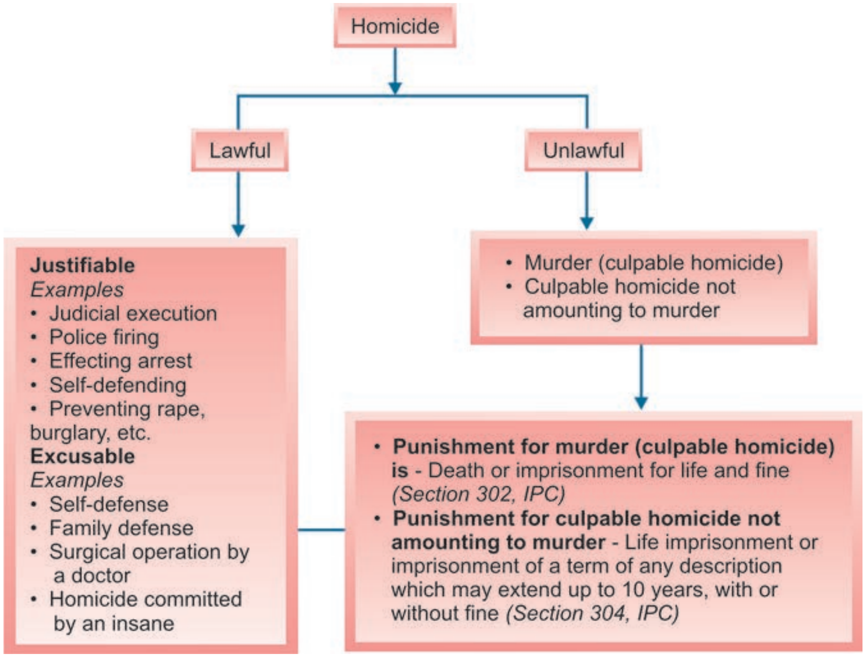
Homicide
Lawful
Unlawful
Justifiable
Examples
• Judicial execution
• Police firing
• Effecting arrest
• Self-defending
• Preventing rape, burglary, etc.
Excusable
Examples
• Self-defense
• Family defense
• Surgical operation by a doctor
• Homicide committed by an insane
• Murder (culpable homicide)
• Culpable homicide not amounting to murder
• Punishment for murder (culpable homicide) is - Death or imprisonment for life and fine (Section 302, IPC)
• Punishment for culpable homicide not amounting to murder - Life imprisonment or imprisonment of a term of any description which may extend up to 10 years, with or without fine (Section 304, IPC)

Fig. 23.1: Murder: Different types and punishments

Explanation: This Section says that if death is caused by bodily injury, the person causing it shall be deemed to have caused death even if death could have been prevented by restoring to proper skillful treatment (Proper remedy and treatment may not be within the reach of the wounded man. Even if proper treatment is present, he may refuse to get it. In such cases if the wounded man dies, the person who caused the injury is deemed to have caused the death).

Punishment for Culpable Homicide Not Amounting to Murder (Section 304, IPC)

- Life imprisonment or imprisonment up to 10 years and fine if the act by which death is caused is done with the intention of causing death or such bodily injury as is likely to cause death.
- Imprisonment up to 10 years or fine, or both if the Act is done with the knowledge that it is likely to cause death.

Murder (Culpable Homicide Amounting to Murder)

(Section 300, IPC) Causing death by an Act done:

- With the intention of *causing death*/kill.
  Example – Stabbing on the heart with a dagger.
- With the intention of *causing such bodily injury as the offender knows to be likely to cause death*. Here the assailant knows about the state of ill health of the victim and that the injury caused is likely to cause death in such a state of health, even though such an injury would not ordinarily have caused death of a healthy person.
  Example – A hits over the area of spleen on the abdomen of B knowing fully well that it is enlarged and thus, rupture it.
- With the intention of *causing such bodily injury, as is sufficient to cause death in the ordinary course of nature.*

  Here, as a result of the intentional act causing injury, the probability of death is very high. (If the probability of death is lesser, then, it is an injury which is likely to cause death – refer culpable homicide not amounting to murder). Thus a stab injury into a vital organ like heart or a major blood vessel is sufficient to cause death in the ordinary course.

  The *intention to cause injury, which is sufficient in the ordinary course of nature to cause death*, is absolutely necessary for making the offence of murder. If during a struggle, the accused merely swing his knife towards the leg of the victim and by a misfortune a blood vessel of the leg was cut leading to death of the victim, the offence cannot be called as murder, as there was no intention to cause death or to cause an injury, which is sufficient in the ordinary course of nature to cause death. (At the most, we can say that the accused had an intention to cause merely an injury on the leg). It is true that the injury caused was sufficient to cause death in the ordinary course as a major blood vessel was cut; however, the accused did not have the intention to cause it. Hence, his crime will fall under *culpable homicide not amounting to murder*.
- With knowledge that the act is *so imminently dangerous that it must in all probability cause death or such bodily injury as is likely to cause death*.

  Here the accused is presumed to have known that his act is imminently dangerous.

  Example – Inflicting serious injury on the neck with an axe or chopper, firing a gun into a crowd, etc.

Exceptions: Five exceptions are given in Section 300 IPC, whereby, culpable homicide will not become murder, and they are:

i. Causing death by grave and sudden provocation:
   Example – husband finds his wife in bed with her paramour unexpectedly. He kills the man then and there. The offence is only *culpable homicide* and not murder.
ii. Causing death by exceeding the right of private defence:
   Example – A slapped B on his cheek. B stabbed A on the heart, and caused his death. Here B had exceeded his right of private defence of body. So he is guilty of only culpable homicide.

iii. Public servant exceeds the powers given by law and causes death.
   Example – Police constable accompanying the convicted person (on the way to court) kills him by firing revolver when the convict tried to escape from custody.
iv. Death caused without premeditation in sudden flight, in the heat of passion.
   Example – Room mates (friends) get involved in a sudden flight on some trivial matter and one gets killed. If there was sufficient time for passion to subside and reason to interpose, the killing will become murder. So suddenness of the flight is important.
v. A person aged above eighteen years suffers death or takes the risk of death with his own consent.
   Example – A doctor gives some poison to a cancer patient, who, due to the pain and suffering, pleads for it and thus death ensues (mercy killing).

Differences between Grievous Hurt, Culpable Homicide and Murder

The line between grievous hurt and culpable homicide not amounting to murder is very thin. In the former, the injury should endanger life and in the latter, the injury must be of the nature which is likely to cause death. Thus, if the culpable homicide is genus, murder is a species. All murders are necessarily culpable homicides but not the vice versa. The Penal Code recognises three degrees of culpable homicide in order to fix punishment proportionate to the gravity of offence.[1-15]

1. *Culpable homicide of first degree* – the *gravest form* of culpable homicide also called as 'murder' (Section 300, IPC).
2. *Culpable homicide of the second degree* – punishable under the first part of Section 304, IPC.
3. *Culpable homicide of the third degree* – *lowest form* of culpable homicide punishable under the second part of Section 304, IPC.

Mental Elements in Culpable Homicide

The offence of culpable homicide supposes an '*intention*' or '*knowledge*' of likelihood of causing death. In the absence of such intention or knowledge, the offence committed may become grievous hurt or hurt, even if death is caused. Thus, if death is caused by an injury, which the offender did not know would endanger life or would be likely to cause death, it is treated as only grievous hurt or simple hurt. But, if the act was deliberate, and was not the result of an accident, rashness or negligence, then it becomes culpable homicide.

Case law: In the course of an altercation on a dark night, the accused 'A' aimed a blow at 'B' with a stick. To ward off the blow, the wife of 'B' who had a child in her arm, intervened between 'A' and 'B'. The blow missed its aim and fell on head of the child, who died due to head injury. In this case, the accused was held guilty of simple hurt only. (He had the intention to cause hurt only on 'B'. it was not a violent blow intended to cause death (culpable homicide) or endanger life (grievous hurt).

Thus, the Court approaches the problem in *three stages*:

1. Was death caused by the act of accused another person? (i.e. is it a *homicide*?)
2. Was it *culpable homicide?* (i.e. it is not coming under any of the exceptions mentioned under *Lawful homicide*)
3. a. If the act does not come under any one of the *four clauses* under Section *300, IPC*, it will only be *culpable homicide not amounting to murder*.
   b. If the act comes under any of the *four clauses* of *Section 300, IPC*, but, at the same time, it comes under any of the *five exceptions* (*refer above*) given under *Section 300, IPC*, then again, it is *culpable homicide not amounting to murder*.
   c. If the act comes under any one of the four clauses in *Section 300, IPC,* and does not fall under any of the five exceptions mentioned in *Section 300, IPC*, it becomes *murder*.

Injury likely to Cause Death and Injury Sufficient to Cause Death in the Ordinary Course of Nature: *(Refer above).*

Punishment for Murder (Section 302, IPC) – death or imprisonment for life and fine.

Rash or Negligent Homicide (*Section 304 A*)

If death of a person is caused by any *rash or negligent act*, *not amounting to culpable homicide*, the punishment is imprisonment up to 2 years, or fine or both.

Rash and Negligent Act

Here, the person doing the act is conscious that a dangerous consequence may follow; however he hopes that it may not result in that particular case e.g.: Penicillin injection given without a test dose. But since the doctor had no intention to cause death, it is not considered as culpable homicide. It may be noted that he would be charged for criminal negligence under this section.

Section 336, IPC

Deals with rash and negligent act endangering human life or personal safety of others (up to 3 months imprisonment or fine up to Rs. 250 or both). Here punishment is given even though no harm is actually caused. Rash act means, something more than mere inadvertence or want of ordinary care. It implies an indifference to obvious consequences. For example, a doctor may give penicillin injection without doing a test dose, knowing fully well the consequences of penicillin reaction, but neglecting to do test. It is typical example of a rash act. If the patient suffers a reaction because of it, Section 335, IPC, is applicable. If the patient dies due to it, Section 304A IPC comes in play.

Section 337, IPC

Rash and negligent act leading to hurt caused to another person (up to 6 months imprisonment or fine up to Rs. 500 or both).

Section 338, IPC

Rash and negligent act leading to grievous hurt caused to another person (up to 2 years imprisonment or fine up to Rs. 1000 or both).

Abetment of Suicide (*Sections 305, 306, IPC*)

It is also considered as unlawful homicide, since the accused is abetting or aiding the victim in committing suicide. It may be noted that if the person who wants to die asks another person to kill him, then it becomes culpable homicide only (i.e. by consent).

- *Punishment for abetment of suicide (Section 306, IPC):* the person abetting the suicide of another person shall be punished with imprisonment up to 10 years and shall also be liable to fine.
- *Attempt to commit suicide (Section 309, IPC):* If any act towards the commission of suicide is done, the punishment is imprisonment up to one year, or fine, or both.

Dowry Death (*Section 304 B, IPC*)

When death of woman is caused within seven years of her marriage and it is shown that soon before her death she was subjected to cruelty or harassment by her husband or any relatives of the husband for, or in connection with any demand for dowry such husband/ relative shall be deemed to have caused her death. The punishment for such dowry related death is imprisonment for not less than seven years, but may be extended to life imprisonment.

EXAMINATION OF AN INJURED[1-5]

Any case of injury is a potential medicolegal issue. When a case of injury is referred, normally the doctor will concentrate on providing all the best services as to save the life of the victim. However, an equally important entity that has to be taken into consideration at this juncture is the medicolegal management of the case. Usually this remains unattended and ignored unintentionally by the doctor mainly due to lack of knowledge about the same. A schematic representation on managing trauma case is shown in Figure 23.2.

MEDICAL MANAGEMENT

Medical management is mainly the treating clinician's concern and includes the following four steps:

- Save life
- Proper diagnosis (use all available laboratory diagnostic aid)
- Proper treatment
- Hospitalisation if necessary.

Medicolegal Management

Medicolegal management includes *specific* and *general* measures.

I. Specific Measures

- Consent for the examination of the injured
- Informing the police *(Appendix-1)*
- Confidentiality of medical examination findings of the patient.
- If the patient is conscious, ask him as to how the injury was caused. Record it in patient's own words. If the patient dies later, it will be accepted as dying declaration by the court. If the injured is unconscious, ask the friends/ relatives accompanying him. In such cases, it must be mentioned in the wound certificate (i.e. as informed by the relatives/ friends).

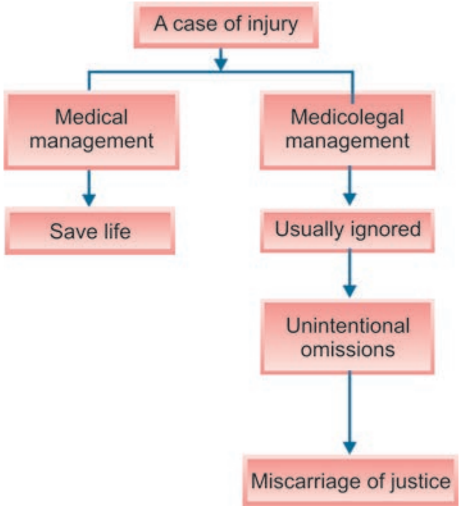

Fig. 23.2: Managing a trauma case

II. General Measures

1. Prepare, preserve and maintain the following documentary requirements.
   - Accident register/wound register *(Appendix-2)*
   - *Case sheet*, date, time of examination and observations noted
   - *Special reports* or other laboratory test reports
   - *Hospital discharge certificate (Appendix-3)*
   - *Dying declaration* – whenever death is likely to occur.
   - *Death certificate* – is not issued unless postmortem examination is completed.
2. In doubtful circumstances
   - Consult a professional colleague available nearby
   - Refer standard textbook
   - Refer the case to another hospital with better facilities *(Appendix-4)*
   - Perform the clinical and other essential medical investigation procedure as available to confirm the diagnosis, e.g. radiographic examination in suspected case of fractures.

COMPLICATIONS OF INJURIES

Injuries of Medicolegal Importance

This includes certain specific injuries[2,3,4,7-15] *(refer Chapters 16 to 19 for details)*, and are as follows:

1. *Hesitation cuts*
2. *Defense wounds/cuts*
3. *Self-inflicted wounds (fabricated wounds)*. These are wounds inflicted by oneself on his/her own body for false accusation purposes, e.g. *artificial bruises*.
4. *Crush syndrome* It constitutes multiple injuries, as seen with road traffic accidents and such other cases, wherein death may result due to renal failure.
5. *Concealed punctured wound*
6. *Head injuries*—following head injuries are important medicolegally:
   1. Coup and contrecoup injuries
   2. Cerebral concussion
   3. Cerebral compression
   4. Lucid interval.
7. *Homicide and suicide*
8. *Injuries and volitional acts*
9. *Fatal wound*—if an injury is present on a cadaver, then the question arises as to whether the injury could have been *fatal in the normal course*, if death is a result of this injury or could he have died of some other cause. If the victim dies after the injury and has no other cause detected to account for death, then we can conclude it as *fatal wound*. Injury to vital organs can also be opined as fatal wounds. When many injuries are present, opinion can be given as death is due to *cumulative effect of all injuries, e.g. multiple contusions*. When complication of wound results death is confirmed by meticulous autopsy and opinion should be given as *"deceased died because of the complications of the wounds sustained."*
10. *Necessarily fatal Injuries (imminently fatal Injuries)*—Injuries that end in death as a direct result of the injury irrespective of any medical aid constitutes necessarily fatal injury. Some examples – crush injury to head, decapitation, and separation of body into two or more fragments (railway accidents).

11. *Injuries likely to cause death* and *Injuries sufficient in the ordinary course of nature to cause death*—The former is given in the definition of culpable homicide (*Section 299 IPC*) and the latter in the definition of murder (*Section 300 IPC*). The distinction between the two is fine but real. In fact it is the degree of probability of death, which makes the distinction. '*Likely to cause death*' means that it is not just a possibility, but it is probable. 'I*njury sufficient to cause death in ordinary course*' denotes that death will be the most probable result of injury in ordinary course. In *'Likely to cause death'*, death is not a surprise. Some examples are:
    - A blow from the front by a stick on the head causing scalp contusion and concussion;
    - Multiple contusions over the body.
12. In *Injury sufficient to cause death in ordinary course* – survival of the victim is surprise. Some of the commonly encountered injuries which can be quoted as examples are:
    - A stab wound or rupture of heart; Injury to large blood vessels;
    - Stab on the chest/abdomen;
    - Blow on the head with an iron rod causing comminuted fracture of skull, intracranial hemorrhages and laceration of brain.
    - Incised wound of the neck – as such is not an injury sufficient to cause death. Unless the major neck vessels and trachea are cut, this is only an injury which endangers life, i.e. grievous hurt.
    - Squeesing of the testicles–as such is not an injury sufficient to cause death. Unless the major vessels are cut, it is only an injury which endangers life, i.e. grievous hurt;
    - Burns > 1/3rd of the body surface;
    - Administration of large dose of poison.

APPENDICES

Appendix 1: Police Intimations

Given below is the standard format of Police intimation letters giving information about a Medico-legal Case (e.g. Road Traffic Accident Case, Unnatural Death Case, Brought Dead Case, Dead on Arrival to Hospital Case, etc.) to the Police. It is the duty of the medical officer (DMO) to inform about all such cases to police as any other citizen, which, if not done amounts to suppression of evidence and is punishable. Details of death may be informed to the nearest police station by phone, followed by a written intimation in specific format given below.

Medicolegal Case

Police intimation letter on a medicolegal case is drafted as shown below:

Ref. No: ....

Place: ....
Date: ....
Time: ....

From:
Name of MO
Designation
Name of Institution/Hospital
Address

To:
The Sub-Inspector of Police
Name of Police Station
Address

Sir,

I write to inform you that a patient by name .... aged about .... inhabitant of (Address) .... has been brought into the casualty/outpatient department (OPD) of .... Hospital, at .... AM/PM On .... is alleged to have been .... at .... AM/PM at (place) ....

He/she is being treated as outpatient/inpatient in Ward No. .... Please do the needful.

Yours faithfully
Signature
Name ....
Reg. No. ....
Designation ....
Address ....
Official Seal ....

Appendix 2: Injury/Wound Report

The doctor who attends the injured patient usually has to record the details of the injuries found, in the *Accident-Register-cum-Injury or Wound Report/Certificate*. The duty doctor should keep this register under safe custody. The forms in the register are arranged in duplicates making a carbon copy. The original of the *Injury or Wound Report/Certificate*, is to be detached and issued to the Police Officer. The carbon copy will remain in the register and serve as a permanent record for the Medical Officer. The standard formats of the accident register and injury/wound certificate are given below:

Accident Register

1. Serial No. .... Date and hour of examination: ....
2. Name: .... Age: .... Sex: ....
3. Address: ....
4. Marks of identification
   a. ....
   b. ....
5. Brought by:
6. History and alleged cause of injury.
7. Details of injuries/clinical features.
8. No. of additional sheets, if any
9. Is dying declaration required?
10. If yes, whether Police/Magistrate is informed?
11. Investigation result, if any
12. Date of admission as inpatient and IP No.
13. Date of discharge
14. Condition on discharge
15. Opinion as to cause of injury

Name of Institution	Signature of DMO*
Station	Name of DMO
	Registration No:
Date	Designation
	Address:
	Official Seal

Issued to .... of Police Station, as per his requisition No. .... Dated ....

---

* Duty Medical Officer (DMO)

Injury Report

Also known as Injury Certificate/Wound Certificate

Ref. No.: .... **Place**: .... Date: .... Time: ....

Name: .... Age: .... Sex: ....

Address: ....

....

Brought and Identified by the Police:

Constable No. .... Name: .... of Police Station

....

Informed Consent:

Question asked

....

Reply given ....

Signature/Left hand thumb impression of consenting person:

Marks of Identification: 1. ....

2. ....

Brief history: ....

General Examination:

Details about the injury/wounds						*Other aspects*	
S.No.	*Type*	*Size—Dimensions*	*Location*	*Simple or grievous*	*Kind of causative weapon*	*Time since injury*	*Other remarks*
1.							
2.							
3.							
4.							
5.							

Name & Signature of DMO:

Designation:

Reg. No.:

Official Seal:

Appendix 3: Discharge Certificate

Issuing a Discharge Certificate of an injury case registered as medicolegal is very important. It may be drafted as below and sent to the concerned Police authority:

Hospital Discharge Certificate

Ref. No. ....

From
Name of MO ....

Designation ....

Name of Institution ....

Station ....

To
The SI of Police ....

Address of Police Station ....
....

Sir,

In continuation of wound certificate No. .... dated .... I have informed you that Sri/Smt/Kum ....
aged .... was admitted on .... in our hospital, IP No...
He/she is discharged/cured/relieved on..................... . Given below are further comments about the case:

a. X-ray and other special investigations..

b. The following surgeons and specialists were concerned in the treatment of the case ....

....

c. Other relevant information ....

Place...

Date..

Name & Signature..

Registration No...

Designation & address..

Official Seal:

Appendix 4: Referring an Injury Case to Second Hospital

For referring injury cases it is better to observe following formalities:

1. All medicolegal *(injury)* cases brought to a hospital should be examined by the Medical Officer first and treatment given.
2. However, if the patient is to be shifted to another hospital due to want of adequate facilities for treatment in the first hospital, necessary first aid should be given by the Medical Officer who examined the patient first.
3. A copy of the accident register/wound certificate should invariably accompany the patient referred to the next institution.
4. If the injured was admitted and treated in the first hospital and later referred to a second hospital for advance treatment, the Medical Officer of the first hospital should issue the wound certificate to the Police Officer so that it can be produced before the Medical Officer in the second hospital who continues the treatment of the injured.
5. Discharge certificate is issued by the attending doctor of the referral hospital *(As in Appendix-3)*.

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Chapter 24

Domestic Violence—Medical and Legal Aspects

INTRODUCTION

Synonyms for domestic violence include *partner violence, relationship violence*, and *intimate partner abuse, spouse abuse, domestic abuse,* and *wife abuse, wife beating, and battering*. Domestic violence is also described as a "*pattern of interaction*" in which one intimate partner is forced to change his or her behavior in response to the abuse or threats of the other partner. Domestic violence is considered to have occurred when one intimate partner uses physical violence, coercion, threats, intimidation, isolation, and/or emotional, sexual, and economic abuses over the other intimate partner to maintain power and control. Domestic violence refers to the victimisation of a person with whom the abuser has or has had an intimate, romantic, or spousal relationship. Domestic violence encompasses violence against both men and women and includes violence in gay and lesbian relationships.[1,2]

DEFINITION

The domestic violence in India is defined by the *Protection of Women from Domestic Violence Act 2005*. Accordingly, the term domestic violence includes elaborately all forms of actual abuse or threat of abuse of physical, sexual, verbal, emotional and economic nature that can harm, cause injury to, endanger the health, safety, life, limb or wellbeing, either mental or physical of the aggrieved person. The definition is wide enough to cover child sexual abuse, harassment caused to a woman or her relatives by unlawful dowry demands, and marital rape.

INCIDENCE

Domestic violence affects people from all races, religions, age groups, sexual orientations, and socioeconomic levels. Victims/persons of domestic violence are mostly women and they usually belonging to one of the following three categories:

1. Single and legally divorced, recently widowed, recently separated, recently sought an order of protection, younger than 28 years of age, addicted to alcohol or other drugs, pregnant, having excessively jealous or possessive partners.
2. Witnessed or experienced physical or sexual abuse as children.
3. Have partners who have witnessed or experienced physical or sexual abuse as children.

MEDICAL ASPECTS OF DOMESTIC VIOLENCE

Domestic violence consists of a pattern of coercive behaviors used by a competent adult or adolescent to establish and maintain power and control over another competent adult or adolescent. These behaviors, which can occur alone or in combination, sporadically or continually, include three types:[3]

- *Physical violence,*
- *Psychological abuse, and*
- *Nonconsensual sexual behavior.*

Each one is discussed individually. However, it may be noted here that, each incident builds upon previous episodes, thus setting the stage for future violence.

Physical Violence

Among the variety of physical violence observed, pushing, shoving, slapping, punching, choking, kicking, holding, binding, assault with weapons are frequent. Usually two forms of physical violence have been noticed at home and they are: *occasional outbursts of bidirectional violence* (i.e., mutual combat) and *frank terrorism.*[4] According to United States of America Preventive Services Task Force survey among the frank terrorist type *patriarchal* (male dominating) form of domestic violence, is more prevalent.[5]

Psychological Abuse

This includes threats of physical harm to the patient or others, intimidation, coercion, degradation and humiliation, false accusations, and ridicule. Annoyance may occur during a relationship, or during and after a relationship has ended. Of women who are stalked by an intimate partner, 81 per cent are also physically assaulted.[6] A new development is psychological abuse (generally threats) expressed through the Internet, so-called *cyberstalking*. Usually the Cyberstalkers target their victims through chat rooms, message boards, discussion forums, and e-mail. Cyberstalking takes many forms such as: threatening or obscene e-mail; spamming (in which a stalker sends a victim a multitude of junk e-mail); live chat harassment or flaming (online verbal abuse); leaving improper messages on message boards or in guest books; sending electronic viruses; sending unsolicited e-mail; tracing another person's computer and Internet activity, and electronic identity theft.[7,8] Recent federal law has addressed cyberstalking as well. The *Violence Against Women Act* passed in 2000, made cyberstalking a part of the federal interstate stalking statute in USA.[9]

Sexual Abuse

This may include nonconsensual or painful sexual acts (often unprotected against pregnancy or disease). Sexual abuse under domestic violence is said to have occurred when any one of the following several forms between an intimate partners has taken place:[10]

- Minimised the importance of your feelings about sex
- Criticised you sexually
- Insisted on unwanted or uncomfortable touching
- Withheld sex and affection
- Forced sex after physical abuse or when you were sick
- Raped you
- Been jealously angry, assuming you would have sex with anyone
- Insisted that you dress in a more sexual way than you wanted.

EFFECTS OF DOMESTIC VIOLENCE ON CHILDREN AND TEENAGERS

When describing the effects of domestic violence on children, it is important to note that domestic violence and child abuse are often present in the same families. In homes where domestic violence occurs, children are physically abused and neglected at a rate 15 times higher than the national average. Several studies have shown that in 60 to 75 per cent of families in which a woman is battered, children are also battered. In addition; children living in households where domestic violence is occurring are at a higher risk for sexual abuse.[11]

Many children in families where domestic violence has occurred appeared to be "*parentified*." They are forced to grow up faster than their peers, often taking on the responsibility of cooking, cleaning and caring for younger children. Many of these children were not allowed to have a real childhood. They do not trust their fathers because of his role as an abuser and they may have been worried about what to expect when coming home. They learned at a young age to be prepared for anything. Children may also be isolated. Typical activities such as having friends over to their house may be impossible due to the chaotic atmosphere. Kids aren't going to have their friends over home when mom has a black eye. However, school performance is not always obviously affected. Children may respond by being overachievers. Children in domestic violence tend to be either extremely *introverted* or extremely *extroverted*. Psychosomatic problems (aches and pains for no apparent reason) are common; these children's eating and sleeping patterns tend to be disrupted. Children who witness domestic violence Domestic violence can wipe out a child's confidence and leave them shocked. Infants and toddlers who witness violence show excessive irritability, immature behavior, sleep disturbances, emotional distress, fears of being alone, and regression in toileting and language, and may develop behavior problems, including aggression and violent outbursts.[12-14]

Teenagers living with domestic violence face the unique problem of trying to fit in with their peers while keeping their home life a secret. Teens in shelters often face the problem of having to move and begin school in a new place, having to make new friends while feeling the shame of living in a shelter. Needless to say, their family relationships can be strained to the breaking point. The result can be teens who never learn to form trusting, lasting relationships, or teens who end up in violent relationships themselves, ending up in violent relationships as adults either as victims or abusers.[11-14]

DOMESTIC VIOLENCE AND LAW—INDIAN SCENARIO

India is a developing nation and though there are cases of domestic violence, it is very rarely reported as for a wife husband is everything after marriage and she will some how cope up with her husband of whatever nature he is and lives with him without reporting the incidence if violence at home to any one. In certain instances the elders in the family when note these being a joint family culture try their level best in counseling such episodes without reporting to authorities. Even the family physician/general practitioners whenever suspect such instances hesitate to report for the fear that the problem may become worse to the victim when reported to authority.

India has adopted the *Convention on the Elimination of All Forms of Discrimination against Women* and the *Universal Declaration of Human Rights*, both of which ensure that women are given equal rights as men and are not subjected to any kind of discrimination. The Constitution of India also guarantees substantive justice to women. Article 15 of the Constitution provides for prohibition of discrimination against the citizens on grounds of religion, race, caste, sex or place of birth or their subjection to any disability, liability or restriction on such grounds. Article 15 (3) gives power to the legislature to make special provision for women and children. In exercise of this power, *the Protection of Women from Domestic Violence Act* was passed in 2005.

THE PROTECTION OF WOMEN FROM DOMESTIC VIOLENCE ACT, 2005

Main Features of the Act

The kinds of abuse covered under the Act are:

1. *Physical Abuse*
    - An act or conduct causing bodily pain, harm, or danger to life, limb, or health;
    - An act that impairs the health or development of the aggrieved person;
    - An act that amounts to assault, criminal intimidation and criminal force.
2. *Sexual Abuse:* Any conduct of a sexual nature that abuses, humiliates, degrades, or violates the dignity of a woman.
3. *Verbal and Emotional Abuse*
    - Any insult, ridicule, humiliation, name-calling;
    - Insults or ridicule for not having a child or a male child;
    - Repeated threats to cause physical pain to any person in whom the aggrieved person is interested.
4. *Economic Abuse*
    - Depriving the aggrieved person of economic or financial resources to which she is entitled under any law or custom or which she acquires out of necessity such as household necessities, stridhan, her jointly or separately owned property, maintenance, and rental payments;
    - Disposing of household assets or alienation of movable or immovable assets;
    - Restricting continued access to resources or facilities in which she has an interest or entitlement by virtue of the domestic relationship including access to the shared household.
5. *Domestic Relationship:* A domestic relationship as under the Act includes live-in relationships and other relationships arising out of membership in a family.
6. *Beneficiaries under the Act:*
    - *Women:* The Act covers women who have been living with the Respondent in a shared household and are related to him by blood, marriage, or adoption and

includes women living as sexual partners in a relationship that is in the nature of marriage. Women in fraudulent or bigamous marriages or in marriages deemed invalid in law are also protected.

- *Children:* The Act also covers children who are below the age of 18 years and includes adopted, step or foster children who are the subjects of physical, mental, or economical torture. Any person can file a complaint on behalf of a child.
- *Respondent:* The Act defines the Respondent as any adult male person who is or has been in a domestic relationship with the aggrieved person and includes relatives of the husband or male partner.

Shared Household: A shared household is a household where the aggrieved person lives or has lived in a domestic relationship either singly or along with the Respondent. Such a household should be owned or tenanted, either jointly by both of them or by either of them, where either of them or both of them jointly or singly have any right, title, interest or equity in it. It also includes a household that may belong to the joint family of which the respondent is a member, irrespective of whether the respondent or person aggrieved has any right, title or interest in the shared household.

RIGHTS GRANTED TO WOMEN

Right to reside in a shared household: The Act secures a woman's right to reside in the matrimonial or shared household even if she has no title or rights in the household. A part of the house can be allotted to her for her personal use. A court can pass a residence order to secure her right of residence in the household.

The Supreme Court has ruled in a recent judgment that a wife's claim for alternative accommodation lie only against her husband and not against her in-laws and that her right to 'shared household' would not extend to the self-acquired property of her in-laws.

Right to obtain assistance and protection: A woman who is victimised by acts of domestic violence will have the right to obtain the services and assistance of Police Officers, Protection Officers, Service Providers, Shelter Homes and medical establishments as well as the right to simultaneously file her own complaint under Section 498 A of the Indian Penal Code for matrimonial cruelty.

Right to issuance of orders: She can get the following orders issued in her favour through the courts once the offence of domestic violence is prima facie established:

- *Protection Orders:* The court can pass a protection order to prevent the accused from aiding or committing an act of domestic violence, entering the workplace, school or other places frequented by the aggrieved person, establishing any kind of communication with her, alienating any assets used by both parties, causing violence to her relatives or doing any other act specified in the Protection order.
- *Residence Orders:* This order ensures that the aggrieved person is not dispossessed, her possessions not disturbed, the shared household is not alienated or disposed off, she is provided an alternative accommodation by the respondent if she so requires, the respondent is removed from the shared household and he and his relatives are barred from entering the area allotted to her. However, an order to remove oneself from the shared household cannot be passed against any woman.
- *Monetary Relief:* The respondent can be made accountable for all expenses incurred and losses suffered by the aggrieved person and her child due to the infliction of domestic violence. Such relief may include loss of earnings, medical expenses, loss or damage to property, and payments towards maintenance of the aggrieved person and her children.
- *Custody Orders:* This order grants temporary custody of any child or children to the aggrieved person or any person making an application on her behalf. It may make arrangements for visit of such child or children by the respondent or may disallow such visit if it is harmful to the interests of the child or children.
- *Compensation Orders:* The respondent may be directed to pay compensation and damages for injuries caused to the aggrieved person as a result of the acts of domestic violence by the respondent. Such injuries may also include mental torture and emotional distressed caused to her.
- *Interim and Ex parte Orders:* Such orders may be passed if it is deemed just and proper upon commission of an act of domestic violence or likelihood of such commission by the respondent. Such orders are passed on the basis of an affidavit of the aggrieved person against the respondent.

Right to obtain relief granted by other suits and legal proceedings: The aggrieved person will be entitled to obtain relief granted by other suits and legal proceedings initiated before a civil court, family court or a criminal court.

LIABILITIES AND RESTRICTIONS IMPOSED UPON THE RESPONDENT

1. He can be subjected to certain restrictions as contained in the Protection and Residence order issued against him.
2. The respondent can be made accountable for providing monetary relief to the aggrieved person and her children and pay compensation damages as directed in the compensation order.
3. He has to follow the arrangements made by the court regarding the custody of the child or children of the aggrieved person as specified in the Custody order.

The Act does not permit any female relative of the husband or male partner to file a complaint against the wife or female partner.

AUTHORITIES RESPONSIBLE AND THEIR FUNCTIONS

The Act provides for appointment of *Protection Officers* and *Service Providers* by the State Governments to assist the aggrieved person with respect to medical examination, legal aid, safe shelter and other assistance for accessing her rights.

Protection Officers: These are officers who are under the jurisdiction and control of the court and have specific duties in situations of domestic violence. They provide assistance to the court in preparing the petition filed in the magistrates office, also called a Domestic Incident Report. It is their duty to provide necessary information to the aggrieved person on Service Providers and to ensure compliance with the orders for monetary relief.

Service Providers: These refer to organisations and institutions working for women's rights, which are recognised under the Companies Act or the Societies Registration Act. They must be registered with the state government to record the Domestic Incident Report and to help the aggrieved person in medical examination. It is their duty to approach and advise the aggrieved person of her rights under the law and assist her in initiating the required legal proceedings or taking appropriate protective measures to remedy the situation. The law protects them for all actions done in good faith and no legal proceedings can be initiated against them for the proper exercise of their powers under the Act.

Court of First Class Judicial Magistrate or Metropolitan Magistrate: This shall be the competent court to deal with cases of domestic violence and within the local limits of this court, either of the parties must reside or carry on business or employment, or the cause of action must have arisen. The Magistrate is allowed to hold proceedings in camera if either party to the proceedings so desires.

General Duties of Police Officers, Service Providers and Magistrate: Upon receiving a complaint or report of domestic violence or being present at the place of such an incident, they are under a duty to inform the aggrieved person of:

- Her right to apply for obtaining a relief or the various orders granted under the Act;
- The availability of services of Service Providers and Protection Officers;
- Her right to obtain free legal services; and
- Her right to file a complaint under Section 498 A of the Indian Penal Code.

Counsellors: The Magistrate may appoint any member of a Service Provider who possesses the prescribed qualifications and experience in counseling, for assisting the parties during the proceedings.

Welfare experts: The Magistrate can appoint them for assisting him in discharging his functions.

In-charge of Shelter Homes: The person in charge of a shelter home shall provide shelter to the aggrieved person in the shelter home upon request made by the aggrieved person, a Protection Officer or a Service Provider on her behalf.

In-charge of Medical Facilities: The person in charge of a medical facility shall provide medical aid to the aggrieved person upon request made by the aggrieved person, a Protection Officer or a Service Provider on her behalf.

Central and State Governments: Such governments are under a duty to ensure wide publicity of the provisions of this Act through all forms of public media at regular intervals, to provide awareness and training to all officers of the government, and to coordinate the services provided by all Ministries and various Departments.

PROCEDURE OF FILING COMPLAINT AND THE COURTS DUTY

- The aggrieved person or any other witness of the offence on her behalf can approach a Police Officer, Protection Officer, and Service Provider or can directly file a complaint with a Magistrate for obtaining orders or reliefs under the Act. The informant who in good faith provides information relating to the offence to the relevant authorities will not have any civil or criminal liability.
- The court is required to take cognizance of the complaint by instituting a hearing within three days of the complaint being filed in the court.
- The Magistrate shall give a notice of the date of hearing to the Protection Officer to be served on the Respondent and such other persons as directed by the Magistrate, within a maximum period of 2 days or such further reasonable time as allowed by the Magistrate.
- The court is required to dispose of the case within 60 days of the first hearing.
- The court, to establish the offence by the Respondent can use the sole testimony of the aggrieved person.
- Upon finding the complaint genuine, the court can pass a Protection Order, which shall remain in force till the aggrieved person applies for discharge. If upon receipt of an application from the aggrieved person, the Magistrate is satisfied that the circumstances so require, he may alter, modify or revoke an order after recording the reasons in writing.
- A complaint can also be filed under Section 498 A of the Indian Penal Code, which defines the offence of matrimonial cruelty and prescribes the punishment for the husband of a woman or his relative who subjects her to cruelty.

PENALTY/PUNISHMENT

- *For Respondent:* The breach of Protection Order or interim protection order by the Respondent is a *cognizable* and *non-bailable offence*. It is punishable with imprisonment for a term, which may extend to one year or with fine up to twenty thousand rupees or with both. He can also be tried for offences under the *Indian Penal Code* and the *Dowry Prohibition Act*.
- *For Protection Officer:* If he fails or does not discharge his duties as directed by the Magistrate without any sufficient cause, he will be liable for having committed an offence under the Act with similar punishment. However, he cannot be penalised without the prior sanction of the state government. Moreover, the law protects him for all actions taken by him in good faith.

APPEAL

An appeal can be made to the Court of Session against any order passed by the Magistrate within 30 days from the date of the order being served on either of the parties.

THE PROTECTION OF WOMEN FROM DOMESTIC VIOLENCE RULES, 2005

The Act empowers the Central Government to make rules for carrying out the provisions of the Act. In exercise of this power the Central Government has issued the Protection of Women from Domestic Violence Rules 2005 relating to the following matters:

- The qualifications and experience to be possessed by a Protection Officer and the terms and conditions of his service;
- The form and manner in which a domestic incident report may be made;

- The form and the manner in which an application for Protection Order may be made to the Magistrate;
- The form in which an application for legal aid and services shall be made;
- The other duties to be performed by the Protection Officer;
- The rules regulating registration of Service Providers;
- The means of serving notices;
- The rules regarding counseling and procedure to be followed by a Counsellor;
- The rules regarding shelter and medical assistance to the aggrieved person;
- The rules regarding breach of Protection Orders.

DOMESTIC VIOLENCE AND LAW—GLOBAL SCENARIO

Despite its widespread occurrence, most domestic violence is largely unrecognised or ignored by professionals, including physicians, family therapists, psychotherapists, and law enforcement officials. Importantly, health care professionals can play a crucial role in the diagnosis, treatment, and referral of victims, helping to break the often intergenerational cycle of domestic violence. Physicians can screen, assess, and intervene efficiently and effectively by eliciting a history of violence, asking specific questions when battering is suspected, documenting the physical findings that often accompany domestic violence, assessing the victim's immediate and future safety, and communicating to the victim all realistic options. Globally, a few countries such as in USA have enacted law against this crime.

Law specifically requires medical staff to report suspected domestic violence. However, many experts suggest that it is *"absolutely contraindicated"* to report on cases of domestic violence to any agency or authority without the *victim's direct request* and *consent*. These experts believe that mandatory reporting of domestic violence often *increases* the survivor's sense of powerlessness and may increase the risk of further harm, including the *risk of homicide*.[15]

Medicolegal Aspects: In all US jurisdictions the victim of domestic violence can obtain by statute a *Civil Protection Order* (*CPO*).[16] In most of the countries an abused adult can file on his or her own behalf. An adult also can file on behalf of a child or decision-incapable adult. A few states in USA allow minors also to petition for protection on their own behalf.

Persons most likely to experience domestic violence include:

- Women who are single or who have recently separated or divorced
- Women who have recently sough an order of protection
- Women who are younger than 28 years of age
- Women who abuse alcohol or other drugs
- Women who are pregnant
- Women whose partners are excessively jealous or possessive
- Women who have witnessed or experienced physical or sexual abuse as children
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A recent survey of physician attitudes found that "45 per cent of clinicians never or seldom asked about domestic violence when examining injured patients". The result is less than 15 per cent of female patients report being asked about abuse by doctors or telling their doctors about their abuse.[11]

Basic for Granting

In USA, the State laws define the relationships that must exist between the parties before a CPO will be granted. Recognised targets of a CPO include current or former spouses, family members who are related by blood or marriage, current or former household members.

Courts and legislatures have identified several types of acts as abuse sufficient to support the issuance of a CPO. Acts of abuse against the petitioner include threats, interference with personal liberty, harassment, stalking, emotional abuse, attempts to inflict harm, sexual assault, marital rape, assault and battery, burglary, criminal trespass, kidnapping, and damage to property (including pets).

Contents

CPOs typically require that the respondent shall:

1. Not molest, assault, harass, or in any manner threaten or physically abuse the petitioner and/or his/her child(ren).
2. Stay 150 yards away from the petitioner's home, person, workplace, children, place or worship and day care provided.
3. Not contact petitioner and/or his/her children in any manner (personally, in writing, by mail or telephone, or through third parties).
4. Vacate the residence at (location) by (date and time) (the police department shall stand by and shall give respondent 15 minutes to collect his or her personal belongings, which include clothes, toiletries, and one set of sheets and pillowcases; no other property may be removed from the premises without petitioner's permission; the police shall take all keys and garage openers from respondent, check to see that they are the right ones, and then turn keys over to the petitioner).
5. Relinquish possession and/or use of the following personal property as of (date and time).
6. Turn over to the police any and all weapons that the respondent owns or possesses and all licenses the

respondent has authorising the possession of or purchase of weapons.
7. Participate in and successfully complete a counseling program.
8. Relinquish custody of minor children to petitioner until further order of the court or the expiration date of the order.
9. Have rights of visitation with minor child (ren) under specified conditions.
10. Pay spousal and child support as designated.
11. Pay for specified repairs, medical or health insurance costs, attorney's fees, and court costs.

Enforcement

In the majority of states, violation of a CPO is a crime for which the police can arrest the offender, even if the violation did not occur in the presence of the officer. The statutory trend is to augment civil or criminal contempt enforcement with misdemeanor charges and to heighten the criminal classification for violation of a CPO. CPOs can and do remain in effect despite the parties reunification or the petitioner's invitation to the abuser to enter her residence.

Criminal Domestic Violence Prosecutions

For most of the twentieth century, victims of repeated acts of domestic violence who killed their partners could not prove self-defense because courts believed that the attack was not necessary, the use of deadly force was excessive, and the victim was the aggressor in the events immediately preceding the killing. In the 1970's, however, psychologist Lenore Walker studied several hundred women in an effort to explain the psychological and behavioral patterns that commonly appear in women who have been physically and psychologically abused by an intimate partner over an extended period. Analogising to scientific research on dogs. Walker theorised that the experience of repeated and unpreventable abuse, along with the social conditioning of women to be subservient, created in battered women a state of "psychological paralysis" that rendered them unable to seek escape or help, even when it might be available. Walker coined the term battered woman syndrome, which soon provided the basis for expert testimony designed to convince a jury that the defendant reasonably believed she had to kill to save herself, even during ebb in violence.

Invoking the syndrome, however, may not always advance justice for battered women who kill. Experts therefore have encouraged a redefinition of the "battered woman" because testimony concerning the experiences of battered women refers to more than their psychological reactions to violence and because battered women's diverse psychological realities are not limited to one particular "profile". As the debate over the proper role of domestic violence expert testimony continues in the legal and scientific literature, courts have begun to admit behavioural science evidence in domestic violence cases.

The role of law in domestic violence cases extends beyond CPOs and criminal prosecutions. Children must be supported, as well as protected; the rights and benefits of employment must be maintained; tort actions may be appropriate; and the validity of prenuptial agreements may be imperiled.

Batterers often assault their children with increase of abuse and kidnapping. The physical and emotional consequences for children who experience domestic violence include medical problems, substance abuse, suicide attempts, eating disorders, nightmares, fear of being hurt, loneliness, bed wetting, and delinquent behaviour such as fighting, prostitution, truancy, crimes against other people, running away, dropping out of school, teenage pregnancy, cognitive disorders, and low self-esteem.

Prenuptial Agreements

Domestic violence may influence prenuptial agreements in three ways. First, battering may provide a defense to the enforcement of an otherwise valid prenuptial agreement. Second, domestic violence may give rise to tort claims that may offset preclusions of equitable economic distribution found in many prenuptial agreements. Third, a prenuptial agreement can include a provision that the occurrence of domestic violence invalidates the terms of the contract.

Employment Issues

Many victims of domestic violence are harassed at work by their former or current spouses or partners. Victims also may miss work because of injuries, court dates, or the need to cooperate with criminal investigations. Job performance may be undermined by depression, fear, and other psychological effects of battering.

Employers may incur liability if domestic violence occurs in the workplace or if they fail to respond properly. Theories of liability may include the Occupational Safety and Health Administration's "general duty" clause, respondent superior, duty to warn, wrongful discharge in violation of public policy or an employee's privacy rights, and negligent hiring, retention, security, and/or supervision. Employees who are victims of domestic violence also are protected by workers' compensation statutes, unemployment insurance or benefit laws, and statutes that preserve benefits for persons cooperating with the judicial process. Perhaps the biggest challenge for employers dealing with domestic violence is to balance employer interests in protecting employees and ensuring workplace safety with employee interests in privacy and freedom from defamation and discrimination.

CONCLUSION

All medical and legal professionals must improve their abilities to identify and confront domestic violence. Appropriate and effective recognition and intervention require vigilance, knowledge of and a willingness to ask the right questions, and a sense of obligation to help society end this undesirable phenomenon. Knowledge of legal considerations should improve the collaboration of health care workers, legal professionals, and community programs seeking to control domestic violence – a major public health problem.

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Chapter 25

Torture and Medical Profession

Torture and medical profession have been closely linked for centuries. Fifty years ago in Nuremberg, Germany, 23 physicians and scientists stood trial for war crimes committed before and during the Second World War. They were accused of inflicting a range of vile and lethal procedures on vulnerable populations and inmates of concentration camps from 1933-45. Fifteen of the twenty accused were found guilty after the trials and of these fifteen, seven were given the death penalty and the remaining were imprisoned.[1]

In cases of state sponsored torture some doctors who have been employed by the Governments are known to have connived with the perpetrators in torturing the victims. The role of physicians in the Nazi horrors has been well documented. Doctors have been present to revive victims in instances where interrogators have been torturing their victims. Doctors have advised torturers on the victims' weak points, and advised against any torture that would result in an '*embarrassing*' death.[2] Of course doctors could always write a false report if the prisoner did die. The Ethics Committee of the Turkish Medical Association in 1995 has suspended ten doctors for preparing false reports to hide the torture of some teenagers.[3]

If human rights are to be integrated into agenda, and meet the complex challenges, health professional accountability becomes a key and non-negotiable objective.[4] Accountability in the context of a human rights framework is the only effective and coherent way to move beyond lip-service to effect systemic transformation and to ensure that struggle to attain socioeconomic rights can integrate health and human rights in a common paradigm.[5] There were five-core objectives suggested by Baldwin-Ragaven et al in 1999.[2] These are:

1. To achieve accountability to our patient and society.
2. Capacity to recognize human rights abuse when it happens.
3. To recognize and empower vulnerable groups so that all patients are treated with dignity and respect.
4. Health professionals to re-orient their practice towards larger social and political context.
5. Health professionals need to be aware of their own positioning in the society and how their values and loyalties may put them in an inconsistent or conflicting situation.[6]

Man has known torture since time immemorial. It is a deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons on another as punishment or to extract information. It occurs in three forms: Physical, mental and or sexual.[7] About 1 in 15 asylum seekers in the United States reported history of torture. From many studies it has been ascertained that 5-30 per cent asylum seekers are tortured.[8]

There is a growing evidence for widespread use of torture among political prisoners throughout the world. Physicians themselves may become victims of torture when the state attempts to subvert the doctor-patient relationship, for political purpose.[9]

The UN Convention against torture, adopted in 1984, is one of the least ratified major human rights treaties. Only 119 States had ratified the Convention by mid-2000. Majority of the doctors in India are aware of various national and international human rights institutions, but seem not to be aware of the human rights of the detainees. It is interesting to note that the doctors are aware of the long-term physical and psychological effects of torture and also agreed that physical examination is not sufficient to detect torture sequelae.

A small number of doctors expressed their unwillingness to get involved in the treatment of the victims of torture due to medicolegal consequences.[9] The lack of knowledge among undergraduate and postgraduate students regarding torture led to incompetence in dealing with these cases. Medical association should take the responsibilities of protecting the doctors who fearlessly testify cases of torture besides disciplining doctors who facilitate torture. Medical profession can no longer ignore the medicolegal and ethical problems. The skills of doctors with forensic expertise allow detection of human rights abuses and thereby its potential reduction. There is scope for the reduction of torture or ill treatment; the profession maintains high standards of medical practice and ethics.[10]

As members of the medical profession, a physician has an obligation to their peers around the world. The current state of physicians' involvement in the prevention of international torture and in the treatment of its victims is very important.[11] In the time of electronic communication, it is easy to communicate with the relevant professionals through out the world.

DEFINITION OF TORTURE

There is a need for a comprehensive definition of torture:

As per the United Nations (UN) Convention against torture, Article 1:

'*Torture*' means any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted on a person for such purposes as:

- Obtaining from him or a third person information or a confession,
- Punishing him for an act he or a third person has committed or is suspected of having committed, or

- Intimidating or coercing him or a third person, or for any reason based on discrimination of any kind; when such pain or suffering is inflicted by or at the instigation of or with the consent or acquiescence of a public official or other person acting in an official capacity.

In the year 1975, the **World Medical Association** adopted a declaration against torture and called it as *Declaration of Tokyo.* According to this, torture is defined as:

'A deliberate, systemic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority to force another person to yield information, to make a confession/for any other reason.[12]

The declaration clearly expressed that a doctor must in no way, for any reason, take part in the practice of torture or other form of cruel, inhuman or degrading procedures as the doctor's role is to alleviate the distress of his/her fellow persons and, '*no motive whether personal, collective or political shall prevail against this higher purpose*'.

METHODS OF TORTURE

Beating is by far the most common method of torture and ill treatment used by state officials today. Thus the torture methods often resorted are of three types:

- Physical torture
- Psychological torture
- Sexual torture.

Physical Torture

People are being brutalised and wrecked physically by various state agents to achieve one purpose or another. The method of physical torture is those, which inflict pain, discomfort in different parts of the body. Killing the victims is not the aim most of the time. The torturer also takes care that the torture inflicted upon the victim remains undetected by an ordinary examination. Therefore, torturers are trained to torture in such a way that these two precautions are well taken care of. However, despite all precautions, physical torture always leaves behind some clues that ultimately lead investigators to its discovery and to the criminals.[13]

Further it is noted that purpose of torture is to spread terror in the society or a country, to destroy a personality, to take revenge; and to get testimony incriminating others.[12]

Case Example

The police arrested Mr. M on 30th July 1996.[15] He was tortured by the helicopter technique (Fig. 25.1A), i.e. putting a rod between tied hands, leg together and then spinning around the rod for an hour. He was taken to courts on 2nd August 1996 for an application to be seen by a doctor. Mr. M was taken to a doctor on the 6th and released. On the 14th he was taken back to hospital with a complaint of chest pain, given a cough syrup, antibiotic and a bronchodilator and sent home. He died the following day. A doctor was appointed by the *Independent Complaints Directorate* to carry out the postmortem. A saddle pulmonary thromboembolus was detected. The case was referred to the author for an expert opinion regarding the possibility of death by thromboembolism two weeks after the torture[14] (Figs 25.1B and C).

Types of Physical Torture

Bearing in mind the methods in practice in India[12] and those adopted globally, various techniques involved in physical torture are:

- **Asphyxial torture** – This is usually done by suffocating the individual.
- **Beating** – Using rods, sticks, chains, cables or such other objects beating is done on various parts of the body from head to toe, including genitals. Method of suspending a person and beating him on the soles called *Falanga*, is quite popular in India.
- **Cold torture** – Pouring thin stream of ice cold water on the nude body or sensitive parts of the body, making the person stand on or walk bare footed or lie down on the ice block, or locking naked in an extremely cold air conditioned room are some of the techniques adopted here.
- **Ear torture** – This comprises of hitting the ears with open palm (*telefona*[12]), continuously, which will create rupture of the ear drum/tympanic membrane, causing severe pain and bleeding from ear resulting in deafness.
- **Electrical torture** – Applying electrical shock by electrodes over the sensitive parts of such as nipples, genitals, oral cavity, anal canal, and arm pits, etc.
- **Heat torture** – Burning by means of cigarette butts, cigar, hot iron rod or flames over the sensitive and concealed parts of the body
- **Irritant torture** – Here an irritant like chilly paste/ powder, Tiger Balm of Rajkot,[12] etc. is applied to the eyes or to other delicate parts of the body especially genitalia. They may be placed forcibly into the mouth or introduced into the anal canal, vagina, etc. The torture method may include making a person walk bare feet or lie down on broken glass pieces, thorny plants, or on pointed nails projecting up.
- **Keeping a person in abnormal position** – The abnormal position could be in standing or sitting or lying supine or prone or on one side or crouched with tying of hands and feet also.
- **Mutilation** – this is an extreme degree of torture where in multiple injuries are inflicted resulting in mutilation of the parts of the body or totally difficulty in establishing the identity of the person.
- **Pulling and/twisting of nails/hairs/tongue/teeth/ breasts/genitalia, etc** – This is quite a painful procedure and may be practiced alone or together in combined form.
- **Roller torture** – Comprises of applying roller over the parts of the body.
- **Suspension** – Here a person is suspended either by hands/ feet for several minutes to hours.

Psychological Torture

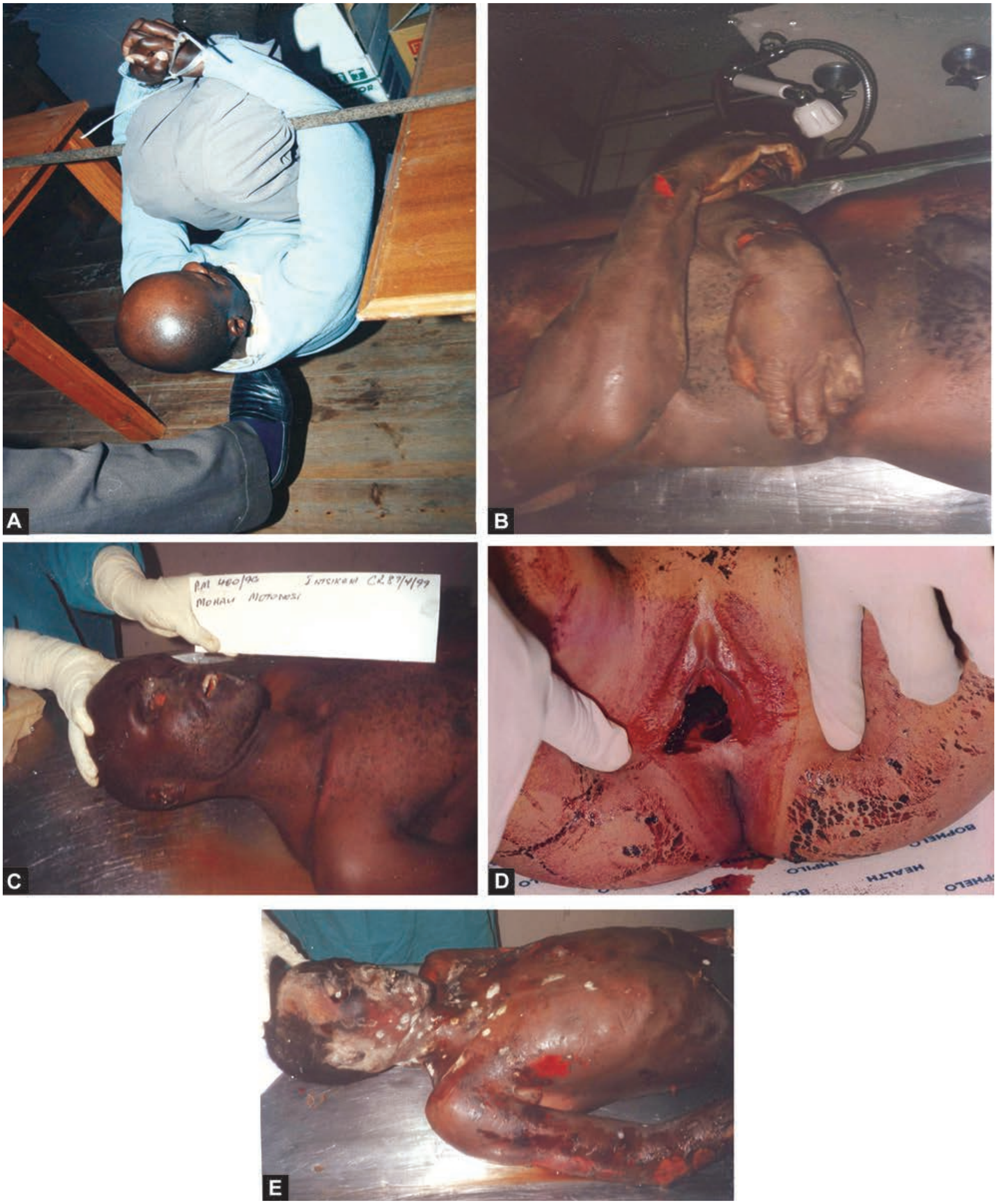
Various techniques of psychological torture may be enumerated as:[12]

- The deprivation technique
- The coercion technique
- The communication technique

The deprivation technique – This include social deprivation, sensory deprivation, perceptual deprivation, sleep deprivation, nutritional deprivation, hygienic deprivation and health service deprivation.

The coercion technique – Impossible choice/incongruent action, humiliations, threats, blind obedience of rules and sexual torture are some of the types under this category.

The communication technique – Counter-effect technique, double blinding technique, disinformation, and distortion of perception and conditioning of new reflexes.



Figs 25.1A to E: *Physical and sexual torture:* (A) Torture by helicopter technique; (B and C) Autopsy findings in same victim as in A; (D) Torture by sexual assault followed by homicide; (E) Forced to drink poison and allowed to die—body recovered in partially decomposed state

Sexual Torture

Sexual torture includes rape, penetration of vagina/ anus by long neck bottles, wooden or metal rods, dildos/ artificial phallus, or such other objects, which can result in injuries or mutilation of the genitalia, causing impotence consequently.[12]

Rape—Throughout history rape has been one of the most common but least documented acts of torture. Yet it has been an inescapable aspect of many conflicts, from the rape of the Sabine women in Ancient Rome to the allegations that the Serbs set up ‘rape camps’ during the recent war in Bosnia.

Rape is a deliberate weapon, and officially sanctioned by some of the countries during wartime. More recently, Pakistani troops were alleged to have raped 200,000 Bengali women during the battle for Bangladeshi independence in 1971.[27]

Mutilation of the female genital—Female genital mutilation is a common and popular practice in some of the African countries, and Egypt. Thousands of young girls are subjected to this torture and mutilation in Egypt alone.

Religious institutions and ancient social customs are primarily responsible for the genital mutilation of female children. The full social and psychological consequences of mutilating the genitals of female children have yet to be evaluated. Preliminary evidence, however, suggest that the psychological consequences of female genital mutilation is very similar to that of rape victims.[28]

Two hundred and eighty three torture victims were questioned about methods of torture and subsequent difficulties. Overall, the prevalence of sexual torture was 80 per cent in women and 56 per cent in men.[29] There are indications that sexual torture has a greater impact on the development of sexual dysfunction in comparison to the other types of torture.[29]

For both men and women the dominant subsequent emotion is usually deep shame. Their community and family as having been defiled and no longer being fit to be accepted.[30-31] Some may have been placed at risk of HIV as a result of sexual violation; particularly as in many countries the incidence of HIV is higher among soldiers, who are often the perpetrators of sexual violence. Victims may not voice their concerns about the possibility of HIV infection because of fears about confidentiality and stigma. It is important to offer testing for sexually transmitted infection and, if appropriate, for pregnancy.[32]

Gender bias torture especially sexual harassment of women at the work place is now a hard reality. This is the ultimate form of control especially in a position of authority. The fear of loss of job, hostility at work and social stigma still prevent women from complaining about sexual harassment.[33]

Post-traumatic stress disorder (PTSD) is more common in the rapes especially by strangers, of physical force being used, of weapon being displayed and injuries being sustained by the victim. These above features can readily lead to the victims developing long-term psychological sequelae after rape.[34]

Clinician should not focus exclusively on the amelioration of symptoms but should provide support, validation, and empowerment for sexual assault survivors who seek treatment.[35]

There is a significant proportion of homicide associated with sexual offences. Women are at high risk of being threatened or shot with firearms.[36] There is a case of decapitation with sexual assault (Figs 25.1D). The perpetrator had sex with a girl and then beheaded her.

In the examination of victims of rape, three things have to be taken into consideration by a medical officer.

Firstly, the over all well-being of the victim. This is important in the sense that there should not be a life-threatening situation such as profuse bleeding that will lead to death. Rape is an acute genital injury syndrome, and need a medical officer to carry out careful examination in a holistic way. Most of the doctors are working like genital technicians only to complete the form.

Secondly, the medicolegal management. This is to carry out the collection of all the evidence so that perpetrator could be brought to books.

The third aim of the examination to look after the future consequences related to rape such as pregnancy, HIV infection and other genital infections. Appropriate antibiotics should be prescribed; HIV test counseling and testing followed by antiretroviral treatment if available should be administered. Psychotherapy and rehabilitation of patient with follow up is a necessary step for the well being of the patient and their families.

PHARMACOLOGICAL TORTURE

This involves introduction/ feeding of various types of pharmacological substances or chemicals into the body producing repulsive symptoms.[12] There is a need of medical and scientific knowledge on the use of drugs in the techniques of torture. Doctors have been involved in training perpetrators on the use of drugs.

Case Examples

- A well-publicised case is that of Dr Wouter Basson, who has now been struck off the medical register and is on trial. He is a cardiologist who has been nicknamed, "*Doctor Death*" and spearheaded the apartheid government's germ and chemical warfare campaign. In one documented case Dr Basson supplied pharmacological agents to security forces who then injected them to their selected victims and when they were knocked unconscious, they were airlifted and the bodies thrown into the sea.[2]
- In 1997, a former policeman Eugene de Kock, known by his colleagues as *Prime Evil*, was sentenced to 262 years in jail for scores of killings carried out for the apartheid government.[2]
- In 1999, 5 Lesotho nationals identified as rustlers were abducted. They were then forced to drink a poison. Four died at the scene and one was admitted to the local hospital that passed away later. The four bodies were in an advanced state of decomposition when discovered (Fig. 25.1E).
- In the years 1994 and 1995, representatives of physicians for human rights studied the problem of physician complicity in torture in Turkey. The research consisted of a survey of forensic documentation of torture, interviews with individual physicians who examine detainees, analyses of official medical reports of detainees, and interviews with survivors of torture. The report provided evidence that torture of political and criminal detainees continues to occur in Turkey and that Turkish physicians are coercive to ignore, misrepresent, and omit evidence of torture in their examinations of detainees to certify that there are no signs of torture.

Sequelae of Torture

Torture is one of the most important preventable causes of psychological morbidity. Although a great deal has been written about the history and the methods of torture, and survivors have produced moving testimonies, there is still no adequate framework within which to describe the range of psychological reactions reported.

There have been attempts made to describe a single '*torture syndrome*'[16-20] but these are generally unconvincing. They lack a theoretical basis and appear to be no more than a list of symptoms commonly seen in survivors of torture.

These include impaired memory and concentration, headache, anxiety, depression, sleeplessness and /or sleep with nightmares and other intrusive phenomena, emotional numbing, sexual disturbances, rage, social withdrawal, lack of energy, apathy, and helplessness.[18,19,21]

Torture by definition creates a severe form of psychosomatic distress because of the person's lack of control over the basic bodily function.[22]

Usually torture sequelae are a combination of physical, psychological and social events.[12] The physical form comprises of severe pain, hemorrhage, infection, scars, mutilation of parts of the body, disfiguration, un-united or mal-united fractures, impairment of vision and hearing, muscle atrophy, closed compartment syndrome, chronic pain, vertigo, STD/HIV, and vague somatic symptoms.

Anxiety, depression, phobia, sleep disturbance, headache, and post-traumatic stress disorders (PTSD), psychosexual problem, psychosomatic problems, and convulsion, psychotic disorders, suicidal tendencies and personality changes**,** are some of the psychological sequelae.

The social sequelae are seen in the form of social stigma, difficulty in getting employment or loss of job, rejection by the family or society or the community.

Management of Torture Victims

This comprises of diagnosis, treatment and rehabilitation. Proper history, physical examination and investigations may help in the diagnosis. The medical man may have to use special skills and diagnostic tools like bone scintigraphy. The fundamental principles for treating the victims are – avoid reminding the patient that he is the victim of torture. Both the patient and the family are to be provided with both physical and psychological treatment simultaneously.

The United Nations Convention calls for education of all doctors and other health personnel.

Education should therefore be at the undergraduate level and should provide an insight into torture methods, the goal and objectives of torture and the sequelae of torture so that doctors can identify victims of torture. The main principles of treatment must also be taught.[23]

Mental disorders figure among the leading causes of disease and disability in the world. Depressive disorders are already the fourth leading cause of the global disease burden. They are expected to rank second by 2020, following ischaemic heart disease but ahead of all other diseases.

Therefore, a medical practitioner should understand not only the physical life event in its own right, but also the psychosocial problem.

Society often regards persons with mental disorder as a threat rather than as a person in need of care.[24] Soon after the release from torture cell, it is important to consult a psychologist for therapy.

Truth and Reconciliation Commission (TRC) notes that, the conditions in mental institutions in South Africa were horrendous and did nothing to foster mental health. Inmates were used as sources of income-producing labor and there are allegations that black patients were used as '*guinea pigs*' in research.[2] More than 40 per cent of countries have no mental health policy: over 30 per cent have no mental health program; around 25 per cent of countries have no mental health legislation. The magnitude of the mental health burden is not matched by the size and effectiveness of the response it demands. More than 33 per cent of countries allocate less than 1 per cent of their total health budgets to mental health, with another 33 per cent spending just 1 per cent of their total health budgets on mental health.[24]

The United Nations health agency report (new understanding, New Hope) seeks to break this vicious cycle and urges governments to seek solutions for mental health that are already available and affordable. Governments should move away from large mental institutions and towards community healthcare and the general healthcare system (WHO).[25]

Prolonged detention without trial has serious effects on mental health of the detainees. It has been equated to psychological torture. The families of detainees too suffer. The prison health services are not adequate. Discrimination against mentally ill is thought to arise in part from the perception that they are dangerous.[26]

Medicolegal and Ethical Aspects of Torture

Torture and Human Rights

International human rights treaties not only regulate the conduct of states and set limits on the exercise of state power; they also take action to prevent abuses of human rights.

States have a duty under international law to take positive measures to prohibit and prevent torture and to respond to instances of torture, regardless of where the torture takes place and whether the perpetrator is an agent of the state or a private individual.

Torture is not an intractable social problem or an inevitable part of the human condition. One can do much to address and prevent it.

The world has not yet fully measured the size of the torture and does not yet have all the tools to carry it out. But the global knowledge is growing and much useful experience has already been gained.

Human rights standards have been established regarding the health professional's role in torture and participation in the death penalty.

Torture and Forensic Experts

More general standards for forensic evaluations, however, are needed. The judgments of the forensic medical evaluator must be completely independent of influence by the state or third parties.

The single most important threat to the human rights of individuals comes from forensic medical examiner that frames medical judgment to serve state or powerful third party interest.

The forensic medical examiner should disclose the confidential report to proper authority and it is for the benefit of that individual.

There is a need of checks and balances for the state-run organisations. A body of health and human rights should be established, consisting of health care professionals, human rights experts, consumers' representatives and legal experts. It should be independent of government, professional organisations and statutory councils.

In conclusion, the job of forensic professionals is to document, obtain, preserve or interpret evidence.

Health professionals are often called upon to engage in evaluations for courts. In torture cases, forensic health professionals are asked to evaluate whether a person is tortured or not. It is a difficult task and risky in some of the states to be a whistle blower.

There exists an inevitable tension between the roles of a forensic expert who has evaluated a torture victim to retain loyalty to the individual.

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Chapter 26

Sexual Jurisprudence

Sexual jurisprudence is a subject subdivision in forensic medicine wherein the medical knowledge is applied to derive justice in cases of sexual offences. The full spectrum of what may be deemed sexual jurisprudence, is realised only when one considers the possible permutations and combinations of an infinite variety of matters related to human sexual behavior, and their physical, physiological and psychological consequences which often pose a difficulty to the medical practitioner and the law enforcement agencies.

Sexual jurisprudence deals with the medicolegal aspects of virginity, impotency, sterility, artificial insemination, pregnancy, abortion, delivery, etc. on one hand, and various types of sexual offences and sexual perversions, on the other hand. Related medical issues needing legal investigation have been discussed, to enable medical professionals to handle such situations independently and easily whenever an occasion arises. Figures 26.1A and B illustrate the normal anatomy of female genitalia and Figure 26.1C shows lithotomy position, the most convenient position to perform local examination and procedures.

VIRGINITY

A female is called a virgin (*Virgo intacta*) if she has never experienced any sexual intercourse.

Signs of Virginity

Principal signs of virginity are studied under (Figs 26.2A to D):[1-8]

a. Genital findings.
b. Extragenital findings.

Genital Signs

Labia majora–firm, elastic, rounded and lie in close contact with each other even on full abduction of the thighs.

Labia minora—soft, elastic, small, pinkish in color, and lie in close contact being completely covered by the labia majora.

Vestibule—narrow.

Posterior commissure and fourchette—intact and crescent-shaped (it is usually lacerated by sexual intercourse in children, and rarely in adults).

Vagina—narrow and tight with rugosed pinkish wall; slit-like orifice due to the apposition of its walls and presence of hymen (rugosity of wall may be lost after childbirth).

Hymen—intact and may admit hardly one finger in an adult.

Extragenital Signs

1. Breast—hemispherical, firm, plump and elastic.
2. Nipples—small and usually surrounded by a small areola, pinkish in fair skinned, while dark brownish in dark skinned girls.

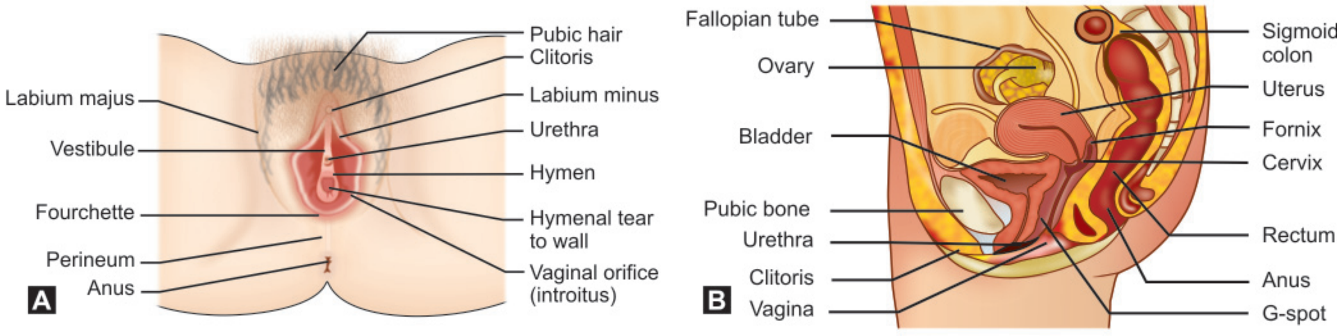
Hymen

Hymen, a membranous diaphragm at the vaginal introitus, is a thin fold (about 1 mm) of mucous membrane derived from the posterior vaginal wall, with an anterior opening.

Types of Hymen

Based on the shape of its opening, hymen may be classified into five types[1-6] (Figs 26.3A to F).

1. Annular—with an oval central opening
2. Crescentic—with a semilunar central opening
3. Vertical—with a vertical slit like opening
4. Septate—with two lateral openings partitioned by a bridge of hymenal tissue
5. Cribriform—with multiple small openings



Figs 26.1Aand B: (A) Normal anatomy of female external and (B) internal genital organs

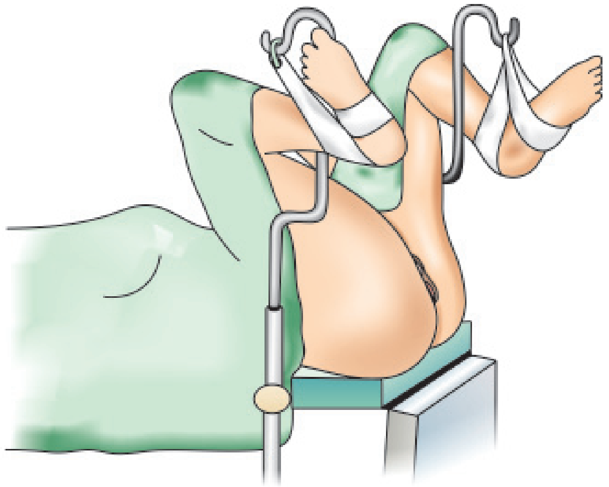


Fig. 26.1C: Lithotomy position for examining external and internal genital organs

Other Variants of Hymen

1. Absence of hymen—observed as a congenital deformity.
2. Infantile—with a small central linear opening.
3. Imperforate—without any opening (Fig. 26.4).
4. Fimbriated—the free margin of the hymenal opening shows symmetrical indentations or notches placed anteriorly.
5. Ring or fringe like or marginal type—with a narrow ring or fringe or band of thick membrane all around the vaginal orifice (can easily distend during sexual intercourse without rupturing the hymen).

Rupture of Hymen (Deflorated)

Hymen usually gets ruptured with the first act of sexual intercourse.[2,4] However in some cases hymen remains intact inspite of sexual intercourse. Such condition where hymen remains intact inspite of sexual intercourse is called *False Virgin*.[2,4,6] It can occur under the following conditions:

- Hymen is fleshy and elastic
- Hymen is thick, tough and with annular or big central opening.
- Hymen is situated higher up in the vagina.

Other Causes of Rupture of Hymen

Besides sexual intercourse, rupture of hymen may occur under the following circumstances:[1,2,4]

- Accidental fall astride on a projected object.
- Passing of foreign body into the vagina, e.g. sanitary pads, etc.
- Masturbation.
- Medical manoeuvres.
- Artificial manoeuvres—*Aptae viris*: to make a young girl fit for sexual intercourse.
- Ulceration from infectious diseases like diphtheria.

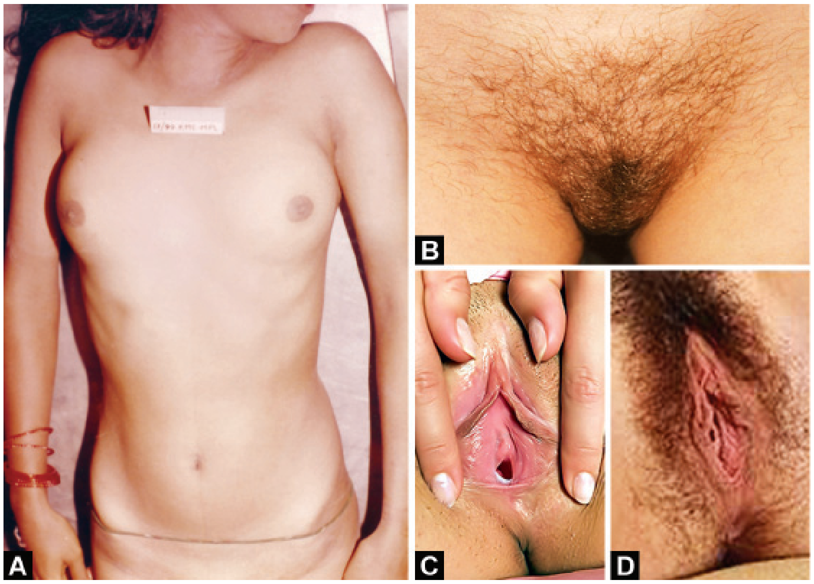
Changes in the Hymen after Rupture

- The ruptured hymen appears like *triangular projections* varying from 3 to 6 in number.
- The tear usually reaches up to the base. It heals up from edges in 4 to 6 days, but torn segments will never reunite.
- The torn segments gradually become thicker and smaller in size and appear as small fleshy pyramidal projections, known as *carunculae hymenales* (Fig. 26.5). After vaginal delivery torn segments (hymenal tags) may disappear or remain as remnants in the form of marginal attachments or as an irregular thick margin known as *carunculae myrtiformes.*

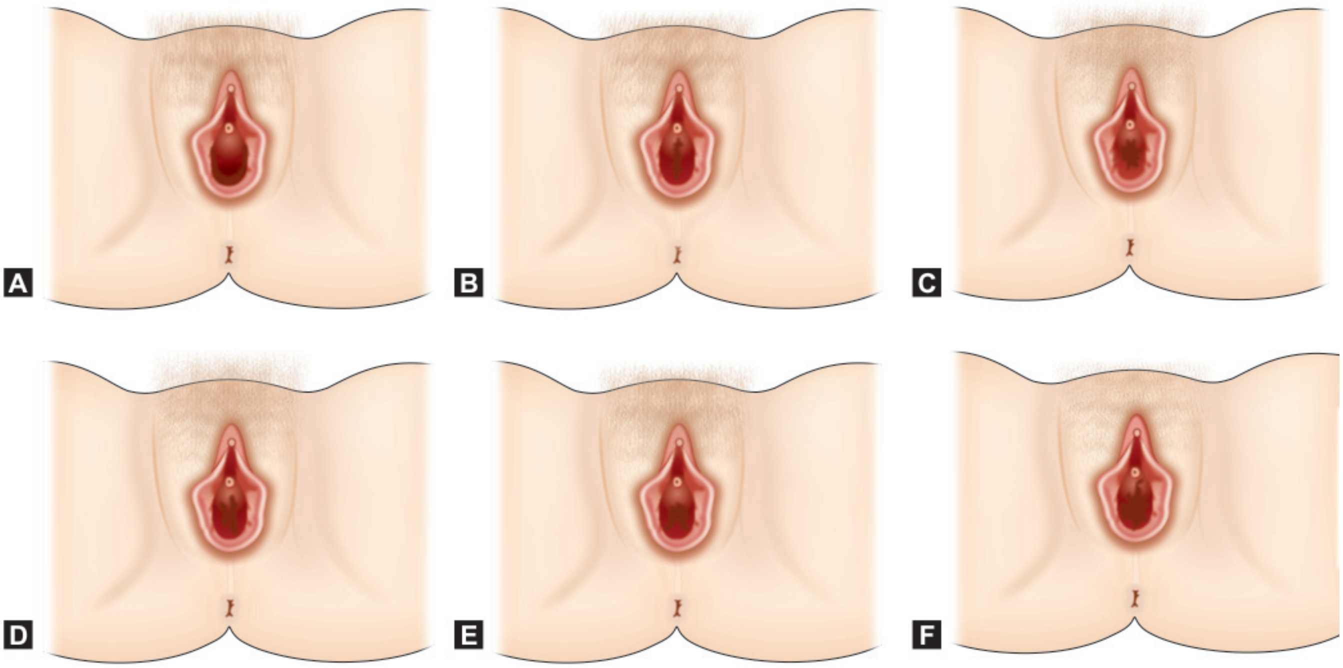
Sometimes the normal fimbriated hymen may be *mistaken* for torn hymen due to sexual intercourse and should be differentiated as mentioned in Table 26.1.

Hymen Examination

Hymen examination is an essential step in the examination of a rape victim. It is done with a special kit called 'hymen examination kit' that comprises of a set of glass rods of varying sizes with a spherical bulbous expansion at one end (Fig. 26.6).



Figs 26.2A to D: *Signs of virginity:* (A) Extragenital: Breasts—hemispherical, firm, plump with small nipple and small areola. (B) Genital: Closely approximated labia majora, covered by pubic hairs. (C) Intact hymen. (D) Genital signs of defloration: Gaping labia majora with protrusion of labia minora in a married and parous woman



Figs 26.3A to F: *Types of hymen:* (A) Crescentic/semilunar; (B) Fimbriated; (C) Annular; (D) Septate; (E) Deflorated—with two lateral lacerations; (F) Deflorated—multiple lacerations

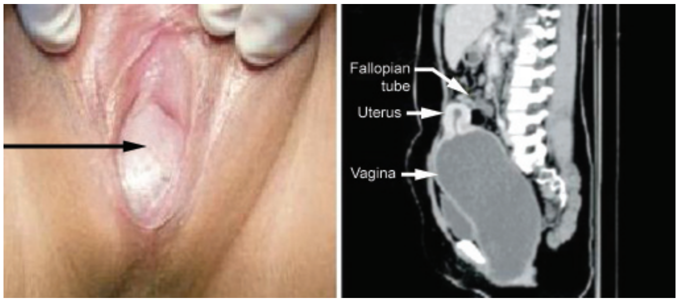


Fig. 26.4: Imperforate hymen, with radiological confirmation. Note—the radiograph shows dilatation of the vagina filled with menstrual blood and uterus with fallopian tubes pushed up to the top of the vagina

Procedure of Examination

The patient is placed in lithotomy position, labia are separated and the bulbous part of the glass rod (pre-heated to body temperature) is inserted into the posterior aspect of hymenal orifice. The bulb is gently rotated along the hymenal orifice in such a way that margin of the hymenal orifice is carefully lifted up by the bulb and examined.

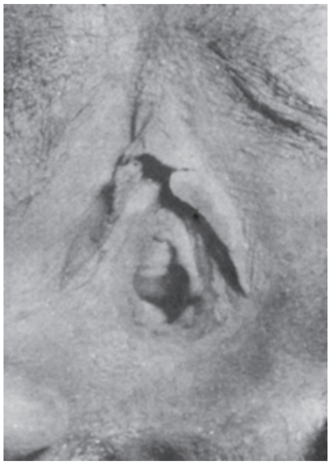


Fig. 26.5: *Deflorated hymen:* with healed tears at 4, 7 o'clock positions, and a notch at the 6 o'clock position. *Note: carunculae hymenales* formation

Medicolegal Importance of Virginity

The question regarding virginity will be considered in the following cases:

1. Rape
2. Defamation

Table 26.1: Differences between normal fimbriated hymen and torn hymen due to sexual intercourse

Fimbriated hymen	*Torn hymen*
Notches are symmetrical and placed anteriorly	Notches (tear) may be single or multiple (rarely), situated in the midline posteriorly or on either side.
Notches do not extend to the vaginal wall	Notches may be tears that extend to the vaginal wall
Mucosa overlying the notches is intact without any signs of inflammation around the notches.	Mucosa overlying the notches is torn with signs of inflammation in and around the tear, if fresh

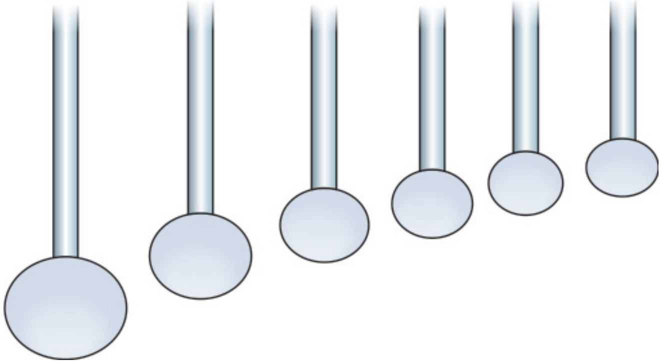


Fig. 26.6: Hymen examination kit

3. Nullity of marriage
4. Divorce

Defloration

Defloration signifies loss of virginity. The genital and extragenital signs in a deflorated female (Fig. 26.2D) are as follows:[4-10]

1. Hymen—ruptured (singularly important sign of defloration).
2. Labia majora—not apposed and gaping
3. Labia minora—not covered and protruding between labia majora.
4. Vaginal canal—dilated with loss of rugosity.
5. Posterior commissure—usually ruptured
6. Breast—enlarged and flabby
7. Nipple—large and surrounded by wider areola.

Medicolegal Importance of Defloration

- The diagnosis of virginity is difficult and in many cases a physical examination of the genital organs may not be objective and helpful. The presence of unruptured hymen offers presumption of virginity, but is not an absolute proof.[9]
- A false virgin may claim that she is a true virgin.[2,3]
- The hymen very rarely is absent congenitally.

IMPOTENCE/ERECTILE DYSFUNCTION (ED) AND STERILITY

Impotence (erectile dysfunction) may be defined as inability to perform sexual intercourse. Sterility means inability to procreate. In case of male, sterility indicates inability to impregnate whereas in female, it means inability to conceive. An impotent need not be sterile and vice versa however, both conditions can coexist.[1-6]

Frigidity refers to a woman who fails to respond to sexual stimulation. There is lack of desire for sexual intercourse.

Incidence—According to NIH, it is estimated that 15-30 million of men suffer from ED in United States population.[7]

Causes of Impotence

They are of varied nature and vary in male and female, and are discussed separately (Figs 26.7 and 26.8):

In Males

1. Congenital or acquired malformations such as:
   - Bilateral hydrocoele (Fig. 26.7).
   - Bilateral inguinal hernia (Fig. 26.8A).
   - Micropenis, (Fig. 26.8B) or complete loss or absence of penis.
   - Severe forms of hypospadiasis or epispadiasis (Fig. 26.8C).

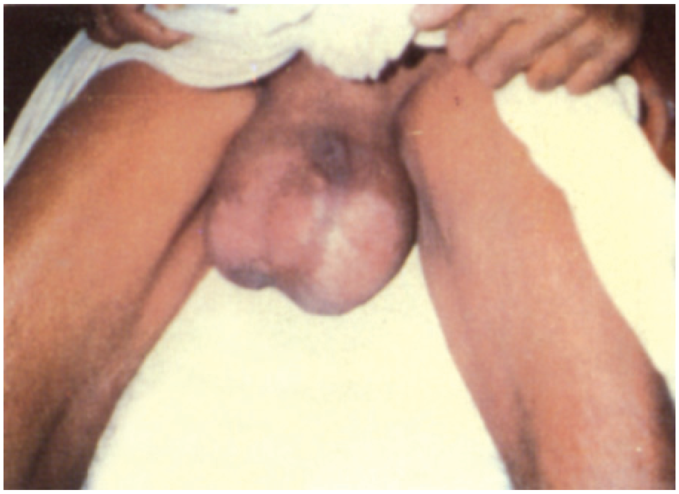


Fig. 26.7: Causes of impotency in male: bilateral hydrocoele (*Courtesy:* Dr Udaypal Singh, KMC, Warrangal, Andhra Pradesh)

2. Age: Before puberty, boys are considered to be impotent though there is no age limit for it. It depends more on the physical development of the individual.
3. Diseases—Local and general (refer below).

Causes of Sterility in Males (Figs 26.8A to E)

1. Congenital or acquired malformations
   - Loss or absence of both the testicles.
   - Cryptoorchidism
   - Severe forms of urethral fistula.
2. Age: Before puberty, boys are considered to be sterile though there is no age limit for it. It depends more on the physical development of the individual.
3. Diseases, which includes local and general diseases are enumerated below:

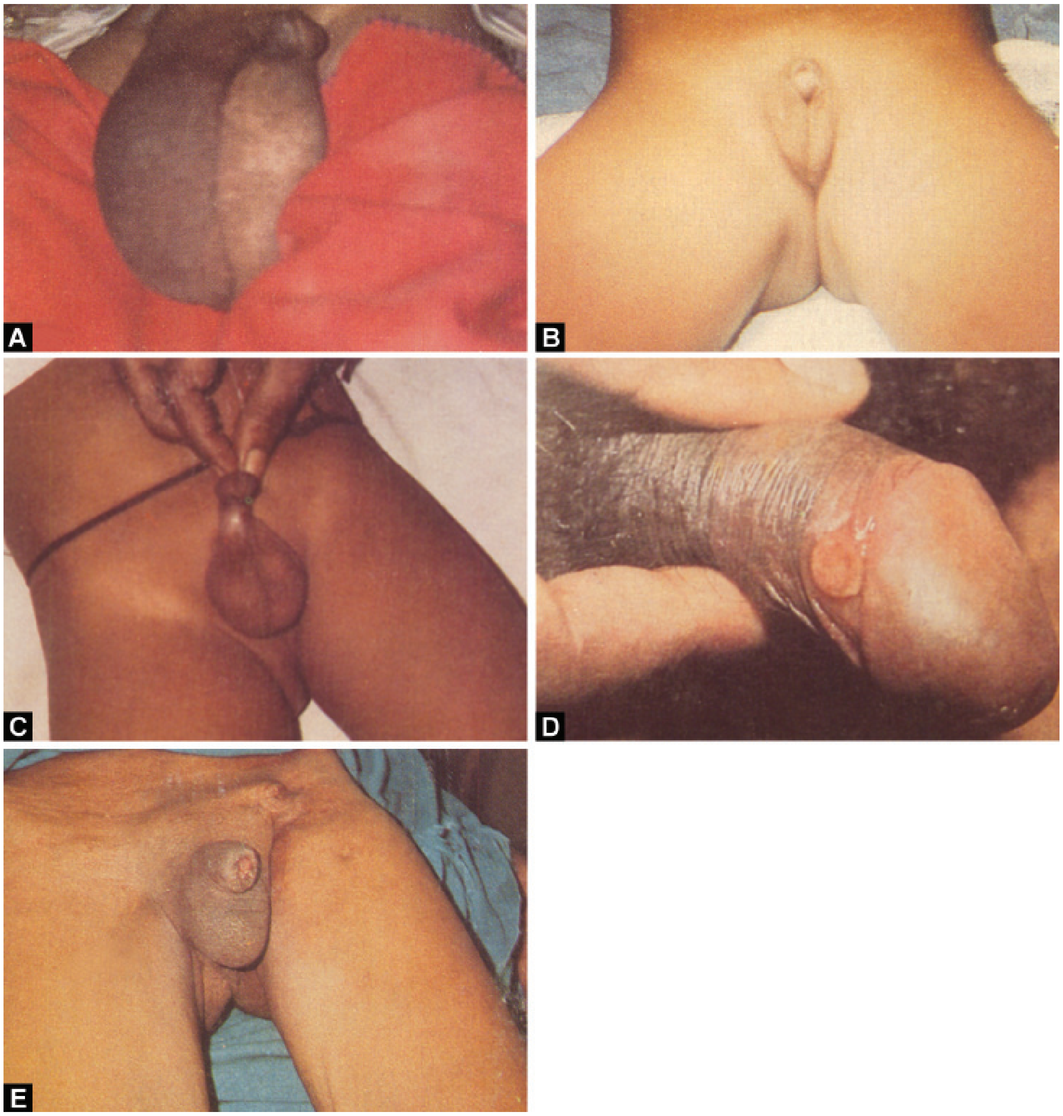
Local diseases

a. Elephantiasis of scrotum and penis.
b. Stricture of urethra.
c. Large size hydrocoele and hernia (Figs 26.7 and 26.8A).
d. Diseases of testis and epididymis, e.g. tuberculosis, syphilis, atrophy of testis following mumps.
e. Partial amputation of penis (Fig. 26.8E).

General diseases

a. Exhausting constitutional diseases like diabetes mellitus, tuberculosis, chronic nephritis, hemiplegia, paraplegia, etc.
b. Hormonal imbalance and chromosomal abnormalities like Addision's diseases, hypopituitarism, etc.
c. Certain diseases of the brain and spinal cord like Tabes dorsalis, transverse myelitis, syringomyelia and injuries to the brain and spinal cord.

4. Prolong or habitual use of certain drugs such as bromide, lead, cocaine, chloral hydrate, barbiturate, Cannabis indica, Dexedrine, opium, heroin, LSD, tobacco, etc.
5. Mental or psychic causes can result in temporary impotence. This may be caused by fear, too much passion, anxiety, hypochondriasis, sense of guilt and aversion. A fear of incompetence against a very virile sexual partner may cause temporary impotence. A man may be potent with a particular woman, but impotent with another woman. This condition is called *impotence quad hanc or psychological impotence.*



Figs 26.8A to E: Causes of impotency in male: (A) Bilateral inguinal hernia (B) Micropenis (C) Hypospadias, (D) Primary syphilitic chancre, (E) Partial amputation of penis (*Courtesy:* Dr Udaypal Singh, KMC, Warrangal, Andhra Pradesh)

In Female

1. Congenital or acquired malformations of genital organs such as absence or atresia of vagina, absence or underdeveloped ovary or uterus, tough and imperforate hymen.
2. In addition, adhesion of vaginal wall due to diphtheria or ulcers may render sexual act impossible. Kraurosis vulva in elderly female will produce vaginismus.
3. Diseases—local diseases such as vaginitis, gonorrhea, leucorrhoea, displaced position of uterus, rectovaginal fistula, tumors of labia and vaginal canal, obstruction of fallopian tubes, diseases of the ovaries, etc.
4. Age—A girl usually attends puberty by 11 to 13 years in India when menstruation begins and achieves menopause by 45 years. In female, the reproductive period normally extends from puberty to menopause.
5. Mental or pshycological causes:[8]
   a. *Hysterical fits*—in this condition, any attempt to perform sexual intercourse will result in severe fits. This may be because of hatred to male sex, fear or excessive passion.
   b. *Vaginismus*—in this condition any attempt to perform sexual intercourse results in severe spasmodic contraction of pubococcygeus (PC) muscles, perineal muscles, vaginal canal, adductor muscles, etc. (Fig. 26.9), which in turn would never allow penile penetration. It may also result from psychological trauma following rape.
   Type: Recently vaginismus in classified into two types:[8,9]
   - *Primary vaginismus*—refers to the experience of vaginismus with first time sexual intercourse attempted.
   - *Secondary vaginismus*—refers to the experience of vaginismus a little later in life, after a period of pain free normal sexual intercourse and typically following temporary pelvic problems.

The Question of Impotence Arises under the Following Conditions[1,4,6]

1. *Civil Cases:*
   a. Nullity of marriage—divorce, nullity and dissolution of marriage can be legally claimed and granted on the grounds of impotence when it is present at the time of marriage and incurable or curable only by surgery to which the individual refuses to submit.

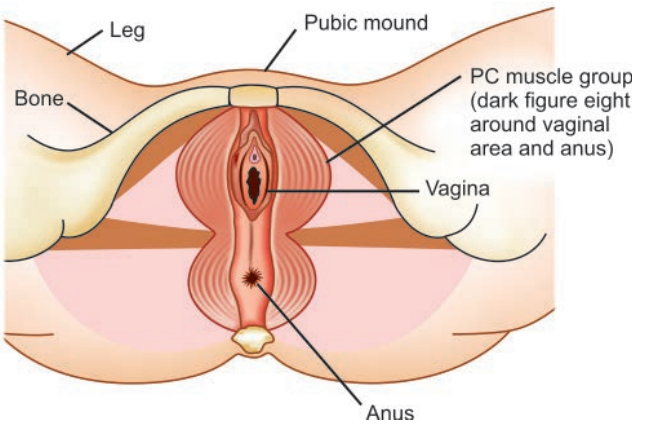

Leg
Pubic mound
Bone
PC muscle group (dark figure eight around vaginal area and anus)
Vagina
Anus

Fig. 26.9: Pubococcygeus (PC) Muscles—group of female pelvic floor muscles which surround both vagina and anus. They tighten involuntarily when vaginismus is experienced

   b. Adultery or any other unnatural sexual offences
   c. Contested paternity or legitimacy where the alleged father pleads impotence or sterility as a defence.
   d. Cases wherein a sterile woman brings in a *suppositious child* claiming right over her husband's property, when the husband is claimed to be impotent.
2. *Criminal Cases:* Impotence and sterility are often put forward as a plea of defense in cases of sexual offences.
   a. Adultery—charges for adultery where impotence is a defense plea.
   b. Rape—charges for rape where impotence is a defense plea.
   c. Unnatural sexual offence—charges for unnatural sexual offence where impotence is a defense plea.
   d. Injured person claiming impotence following injury caused by negligent act of another, thus claiming for compensation.
   e. Blackmailing and defamation.

ARTIFICIAL INSEMINATION (AI)[1-6]

Artificial introduction of semen into vagina, cervix or uterus to bring about pregnancy is termed *Artificial Insemination* (AI).

Types

1. Artificial insemination homologous (AIH)—when the semen of the husband is used.
2. Artificial insemination donor (AID)—where the semen of some other person is used.

Indications

1. When the husband is impotent but fertile.
2. When the husband's sperm count is not up to the optimum level of fertility.
3. When husband is suffering from congenital anomalies like epispadiasis or hypospadiasis and is unable to deposit semen in the vagina.
4. Rh-incompatibility between husband and wife.
5. To avoid transmission of hereditary diseases.
6. *Nullity of marriage*—marriage may be nullified under the following conditions:
   a. When either party is under-age for marriage contract.
   b. When either party is already married.
   c. When one party is of unsound mind or mentally defective or suffering from incurable disease at the time of marriage.
   d. Where the marriage was not consummated due to impotence or wilful refusal.
   e. Where the woman was pregnant by another man at the time of marriage.

Medicolegal Aspects

The practice of artificial Insemination makes many infertile couples happy. It is practiced all over the world.[3-6] Insemination with husband's semen is justifiable, but AID is generally not socially accepted. There is no statutory law in India for artificial insemination as yet.[5,6] Nevertheless, the national guidelines are framed by the Indian Council of Medical Research.[9]

The following are the legal aspects of AID:[1-6]

1. Adultery—the donor and the recipient cannot be held guilty of adultery in India as there is no act of sexual intercourse (Section 497 IPC).
2. Legitimacy—the father is not the actual father and as such, the child is illegitimate and cannot inherit the property. This drawback may be overcome by a statutory law mentioning that the child born through consentual artificial insemination is legitimate.
3. Nullity of marriage—AI as such is not a ground for divorce. But if it is done due to impotence, or done without the consent of the husband, it will become a ground for divorce or nullity of marriage.
4. Status of the child-a child born of AID remains illegitimate unless it is adopted. But if the parents do not declare AI, the child remains a natural child for all practical purposes.
5. There is a remote chance of incestuous relationship between the donor and the recipient's offspring.
6. Sociological aspects—the husband may feel humiliated for his deficiency in procreation and may develop psychiatric problems. If the child is mentally retarded or physically deformed, the husband may feel resentment as he is not the actual father but is partially responsible for this deformity. Mother may become neurotic as the child belongs to her alone, but not the husband. She may also develop an obsession to know the donor whose name may not be divulged by the doctors. If the child comes to know his history of birth, he may have a great shock and may even have mental trauma.

Precautions

Though special precaution need not be taken for AIH, the following precautions are required to be adopted in case of AID:[5-7]

1. The knowledge and full consent of the donor is mandatory. Similarly, consent of the recipient wife and her husband is also necessary.
2. The identity of the donor must remain secret to the recipient and her husband.
3. The results of insemination and the names of the recipient and her husband should remain secret to the donor.
4. The donor must be mentally and physically healthy and should not have any hereditary or familial diseases.
5. The donor must not be a relative of either spouse.
6. He should be fertile and his age should not exceed 40 years.
7. The race and morphological appearance of the donor should resemble the husband of the recipient as far as possible.
8. The donor should give a written declaration that he will not claim parenthood for the child.
9. Rh-compatibility between the donor and the recipient should be tested.

TEST TUBE BABY (*IN VITRO* FERTILISATION/IVF)

In this process, the ovum of the wife is removed from the ovary through the abdominal wall and is fertilised by the sperm of her own husband in a laboratory. At the stage of blastocyst, the developing embryo is implanted into the uterus through the uterine cervix and develops there till full term foetus. It was *Dr. Steptoe* who pioneered the birth of the first test tube baby in *Bolton*, *England* in mid 1970s.[13]

SURROGATE MOTHER[14,15]

This is a woman who bears a child either by artificial insemination from husband of a sterile woman or by implantation of *in vitro* fertilised ovum at the *blastocyst stage*. The *surrogate mother* bears the child and on delivery, she hands over the child to its biological father and his wife.

The practice of surrogate motherhood has been commercialised thus, raising various legal issues. At times the surrogate mother refuses to handover the baby to the biological parents; and sometimes the couple get separated or divorced before delivery thus compelling the surrogate mother to abort. Some couples prefer surrogate motherhood inspite of being fertile just for the sake of maintaining a good physical appearance. There is usually a contract for the surrogate mother not to claim guardianship of the child. Thus surrogate mother is a mother by substitute.

Semen Bank

Human semen can be preserved for future donation by means of slow cooling and by addition of glycerol. However, there is a legal problem when the woman becomes pregnant after the death of her husband and claims her *posthumous child* to be the product of insemination from the semen bank of her husband, thus demanding the child to be declared as the legal heir to her husband.[16,17]

STERILISATION

Sterilisation is a procedure to render a person sterile but without any interference to potency or sexual function.[1-6]

Types

1. *Compulsory:* This is performed by an order of the State on eugenic grounds for those who are mentally defective and as a punishment for those sexual criminals. It is not practiced in India.
2. *Voluntary:* Voluntary sterilisation may be done on the following grounds:
   - *As a family planning measure.*
   - *For therapeutic purpose*—This is performed to prevent danger to the health or life of the woman by future pregnancy. It is done as a therapeutic measure for certain diseases. The indications are:
     a. Repeated cesarean operations.
     b. Chronic diseases of the heart, lungs or kidneys, or carcinoma of breast or testicles where removal of ovaries or testis is performed.
     c. Severe physical or mental defects.
   - *As eugenic measure*—It is also done on eugenic grounds to prevent transmission of hereditary diseases.

Methods

Sterilisation could be permanent or temporary.

a. Permanent methods are *vasectomy* in male and *tubectomy* in female, and exposure to deep X-rays of gonads in both.
b. Temporary methods are the use of oral hormonal pills, condom, diaphragm, spermicidal jellies and intrauterine contraceptive devices including loops, copper T, etc.

Legal Safeguards for Permanent Sterilisation[18]

The following precautions should be observed before undertaking the surgery:

a. Written consent of both the couples should be taken.
b. When performed for family planning purpose, the age of the husband should not be below 25 and the wife below 20 years and they must have at least two children one of whom should be a male child.
c. When performed for eugenic or therapeutic purpose, a senior colleague should be consulted.
d. It is preferable to have a seminal check-up after vasectomy. The couple should be advised to abstain from sexual intercourse for at least 3 months or till the semen examination shows absence of spermatozoa.

PREGNANCY

Pregnancy is a phase in the reproductive period of a woman which results as a consequence of fertilisation of an ovum by a sperm.

Diagnosis

Pregnancy can be diagnosed from *subjective* and *objective* signs.[2-6]

Subjective Signs

- Amenorrhea (cessation of menstruation)
- Morning sickness: nausea and vomiting on getting up from bed in the morning
- Perverted appetite
- Increased frequency of micturition
- Progressive enlargement of abdomen
- Quickening—a peculiar sensation of fetal movement felt by the mother (felt from 16th week of pregnancy)
- Excessive salivation
- Constipation

All these subjective symptoms are of mere presumptive value and not much of diagnostic significance as they can also be found in many other pathological conditions.

Objective Signs

They include *probable signs* and *positive signs* of pregnancy.

Probable Signs

a. *Changes in the Vagina*
   - The normal pink colour of vaginal mucosa changes into violet and ultimately into blue colouration due to increased vascularity resulting from pressure of the gravid uterus (*Jacquemier's or Chadwick's* sign).
   - Flattening of anterior vaginal wall by the upwardly titled cervix.
   - Thickening of hypertrophied mucosal folds.
b. *Changes in the Cervix*
   - Increased vascularity imparts certain changes to the cervix such as softening (Goodel's sign), fullness and roundedness with circular external os.
c. *Changes in the Uterus*: The increase in the size due to the growing foetus makes it an abdominal organ from fourth month of pregnancy. The fundus of the uterus can reach the level of xyphisternum at full term (Fig. 26.10). Gestational changes in the uterus are enumerated below:

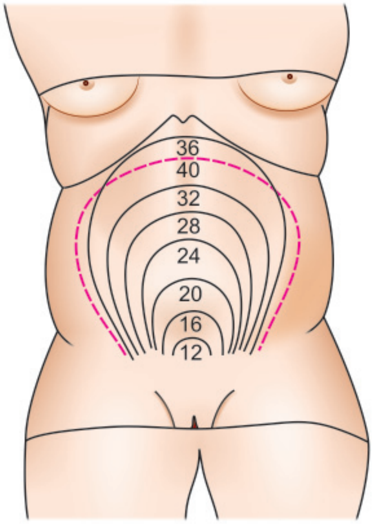


Fig. 26.10: Fundal height at various weeks of gestation

- Increase in size— progressive increase in the size is observed with the growth of the fetus within.
- Hegar's sign is the characteristic softening of isthmus of the uterus, detectable by manual examination, from sixth week.
- Braxton Hick's sign is an intermittent uterine contraction, seen only after fourth month.
- Uterine souffle—a blowing sound synchronous with maternal pulse, due to the blood circulation through the enlarged tortuous uterine arteries heard by auscultation of the abdomen from sixth month onwards.
- Ballottement (tossing up like a ball)—by this test, the palpating hand or finger can give a jolt or push to the fetus per abdomen (external ballottement) or per vaginum (internal ballottement) only to feel the hitting back of the fetus on the palpating fingers immediately. It can be elicited positively during fourth and fifth fetal month.
- Foetal part can be palpated through abdominal wall. This is appreciated only after sixth month.

d. *Changes in the Breast:* Mammary changes are prominent in primi and they are as follows: (Fig. 26.11)
  - Enlarges in size, becomes firm, tense and tender.
  - Nipples become prominent, with 10-12 small pigmented nodules around called *Montgomery' tubercles.*
  - Pigmentation of areola, making it darker
  - Secondary areola formation, i.e. the pigmentary changes go beyond areola (primary areola) on the normal skin around for about 2-5 cm or more.
  - Superficial veins may become more distinct.
  - Secretion of *colostrums (witch's milk)* from fourth month.

e. *Laboratory tests:* Several laboratory tests have been reported, but each of them have their own limitations, as these tests can give positive results in conditions other than pregnancy.

I. **Biological tests**: These tests are based on reactions of chorionic gonadotropins in pregnant woman's blood or urine on test animals. These include: Asheim Zondeks test, Friedman's test, Hogben test, etc. However, introduction of newer immunological, rapid reporting, highly sensitive tests have made the biological tests almost outdated.

II. **Immunological tests**: These tests are based on antigen-antibody reaction upon human chorionic gonadotropin hormone (HCG) passed by the pregnant mother in her urine. They are:
  - Gravindex slide test
  - Prognosticon tube test, etc.

An antibody against HCG is obtained by injecting HCG into rabbit, and then collecting the serum. Sheep RBC or latex particles coated with HCG.

Procedure:

- A few drops of the morning urine of a pregnant woman are first treated with anti-HCG serum, and then with coated sheep RBC or latex particles.
- If the urine has HCG, a reaction takes place between the HCG in urine and the anti–HCG in serum. Thus, red cells/latex remain unagglutinated, and the test is reported as *positive.*
- If the urine has no HCG, anti-HCG in the serum added would be available in the suspension and react with the HCG coated on sheep RBC or latex particles, producing agglutination. This is reported as *negative* test.
- Time required for reporting 2-3 minutes.
- Fallacies—false-positive results may be reported with hydatidiform mole, chorion-epithelioma, ectopic gestation, etc.

Positive Signs of Pregnancy

(Synonyms —Absolute/Conclusive/Certain/Sure Signs of Pregnancy):

1. ***Foetal movements:*** these are felt by keeping the palpating hand on the abdomen from fourth month and also seen by naked *eye* examination from fifth month.

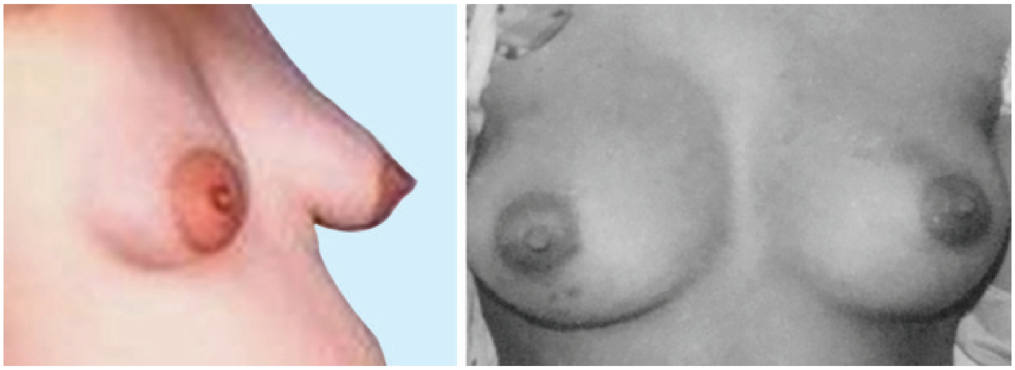


Fig. 26.11: Breast changes in pregnancy. *Note*—large, soft, pendulous nature with large nipples and dark areola and Montgomery tubercles

2. ***Foetal heart sound***: forms an important and definite sign of pregnancy, heard from 18 to 20 weeks. Normal heart rate is 160 per minute at fifth month and 120 per minute at ninth month.
3. ***Radiological diagnosis:*** shadow of fetal skeleton in the radiograph and ultrasound scanning of the abdomen is diagnostic of pregnancy, which is usually seen from 15th to 16th weeks. It is also diagnostic of twin pregnancy, fetal abnormalities, intrauterine death of fetus (***Spalding's sign***—crowding of cranial bones) (Figs 26.12 and 26.13), hydatiform mole, etc.

Medicolegal Importance

1. When a woman is condemned to death or sentenced to undergo rigorous imprisonment, she might submit a petition to the court, stating that she is pregnant. In India, during pregnancy, a woman cannot be hanged, until she delivers and the child is six months old.
2. When a woman after her husband's death may feign to be pregnant, so that she might be entitled to the estate left by her deceased husband on behalf of the prospective heir.
3. When a woman after claiming to be pregnant, brings an accusation in the court for breach of marriage or seduction against a certain person.
4. When an unmarried woman, a widow or a woman living separately from her husband, wants to get rid of charges of *adultery* brought against her on grounds of her pregnancy.
   - When a woman alleged to be pregnant asks for compensation from a person.

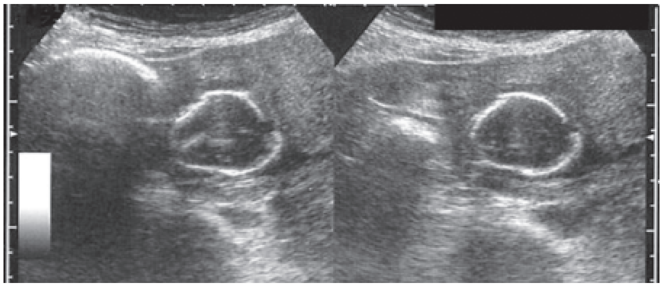


Fig. 26.12: Spalding's sign—ultrasound images of the foetal skull—overlap of bones—IUD of foetus

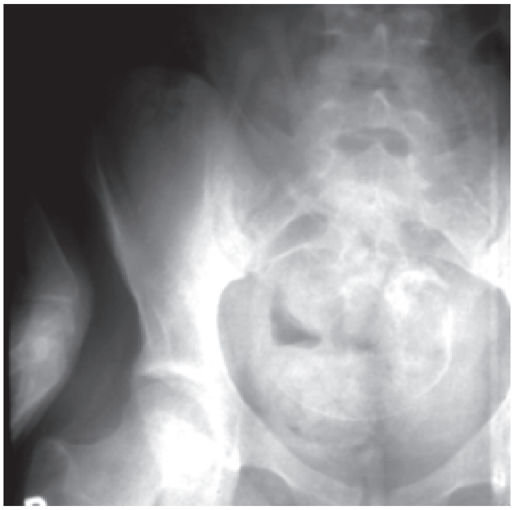


Fig. 26.13: Spalding sign foetal skull bones and spine and ribs overlap–IUD of foetus (*Courtesy:* http://myweb.lsbu.ac.uk/dirt/museum/856-826.html)

- When the pregnancy of a widow or an unmarried girl is suspected to be the motive of her suicide or murder.
- A woman who is pregnant can claim greater compensation in case of death of her husband in a railway or aeroplane accident.
- In case of attempted criminal abortion or infanticide.
- ***Pseudocyesis*** (*Synonyms : spurious pregnancy, phantom's pregnancy, feigned pregnancy*): This is a condition wherein a woman who has no issues nearing menopause and intensely desiring an offspring, presents with all subjective signs of pregnancy including an abdominal distension which may be due to deposition of fat, ascites or tumour.
- ***Duration of pregnancy***: Accepted average period of pregnancy is 280 days (i.e. 10 lunar months or 10 times intermenstrual period) from first day of last menstrual period (LMP).
- ***Period of viability:*** Child born at or after 210 days of pregnancy is considered a viable child, as it is capable of an independent survival outside the mother's uterus (*Refer Chapter on Infanticide*)

DELIVERY

Delivery refers to the birth of the child, on completion of 280 days of pregnancy (full term).

If delivery occurs earlier than 280 days, it is called premature delivery, while if it occurs after 280 days, it is called postmature delivery.

Signs of delivery: Signs of delivery could be *recent* or *remote*. It again varies in the *living* or in the *dead.*$^{1-6}$

Signs of Recent Delivery in the Living

Signs mentioned below are characteristic of a full term delivery other than a premature one. They are likely to disappear within 10 days in a healthy woman.

General indisposition: She will be apathetic, pale, and ill-looking with slight increased pulse and body temperature.

Abdomen: Abdominal wall is pendulous, lax, wrinkled with striae gravidarum.

Breasts: These are full, enlarged and tender exuding colostrum or milk. The areola is dark, nipples prominent and Montgomery's tubercles are present.

Uterus: The uterine changes may be enumerated as:

- 0-1 day – relaxed flabby mass at umbilical level.
- 2-3 days later – hard, cricket ball-like mass in the lower abdomen.
- In 6 weeks – normal.

Cervix: It is soft, patulous with torn or lacerated edges. The internal os begins to close within 24 hours. *External os* is soft and patent; admits two fingers for a few days initially, followed by one finger at the end of one week, and complete closure in 2 weeks. However, it is transverse, enlarged and patulous in parous uterus, while in nulliparous, it is small, round and dimple like in the centre of cervix.

Genital tracts:

- Vulva – is swollen, may be bruised and lacerated
- Labia majora – are swollen, congested and may be bruised and tender
- Fourchette and perineum – may show laceration

Table 26.2: Changes in size and weight of the uterus after delivery

Days	*Size (l x b x t cm)*	*Weight(gm)*
0-1 day	25.0 × 20.0 × 10.0	1000
2-3 days	17.5 × 10.0 × 05.0	500
4-7 days	14.0 × 08.0 × 05.0	350
8-15 days	12.0 × 06.0 × 02.5	125
6 weeks (Normal)	07.0 × 05.0 × 02.0	80

l × b × t = Length × breadth × thickness

- Lochia – is a discharge from the uterus, and its presence is a characteristic sign of all recent deliveries. It has got a peculiar odor and is of three types:

  Lochia rubra – First 3 to 4 days, bright red in color, blood mixed, with large clots.

  Lochia serosa – in next 4 days it turns pale and serous.

  — Lochia alba – on about ninth day it turns yellowish gray or lightly greenish, gradually diminishes in quantity and then disappears completely.

Signs of Recent Delivery in the Dead

In addition to findings mentioned above in the living, following may be observed in the uterus:

1. *Size of the uterus* – it depends on how long the victim lived after delivery. Table 26.2 highlights changes in the size and weight of the uterus depending on number of days of survival of the woman after delivery.
2. *Peritoneal covering is wrinkled.*
3. *Cut section shows* – dark coloured, irregular area of placental attachment covered with blood clots. The diameter of this area can give clue about the number of days after delivery as shown in Table 26.3.
4. *Fallopian tubes* – congested
5. *Ovaries* – both are congested and one of them show a large corpus luteum.

Signs of Remote Delivery in the Living

Pregnancy usually leaves tell tale permanent marks on the body of the woman provided, it is a full term pregnancy. These findings are:

Abdomen: Abdominal wall will be lax with *linea nigra* and *linea ablicantes*.

Breasts: Will be large, soft, and pendulous, with large nipples, dark areola and Montgomery's tubercles.

Genital tract:

- Vulva – gaping
- Vagina – vaginal orifice is partially exposed, vagina will be capacious, and with no rugosity, and walls not approximated.
- Hymen – absent or if retained seen as tags called *carunculae myrtiformes*.
- *Cervix:* external os appears like transverse slit.

Signs of Remote Delivery in the Dead

In addition to the findings mentioned above in the living, there will be:

Table 26.3: Diameter of the placental area

Day/weeks	*Diameter (cm)*
0-3 days	12-15
4-7 days	03-04
6-12 weeks	01-02

- *Uterus* – slightly enlarged with thicker walls, larger cavity and more weight.

Medicolegal Importance

It is necessary to determine whether the woman has delivered or not in solving the cases, such as:

- Legitimacy
- Legal action for disputed chastity
- Feigned delivery
- Abortion and infanticide
- Concealment of birth
- Blackmail – a woman may feign pregnancy for sometime and then produce some child, alleging that, it is her child. The motive here is to extract money by blackmailing. Such a child is called "fictitious child" or "suppositious child"
- Affiliation cases – these are cases wherein woman having illicit sexual intimacy with a man may become pregnant, and deliver a child and then sue him for maintenance of the child.

PATERNITY

Paternity is the '*fatherhood*' of a child.

Diagnosis of Paternity

Determination of paternity is usually done by certain tests and these tests are called *paternity tests* [1-6] such as:

1. ***Parental likeness*** – a child may resemble the parents in feature, figures, complexion, gesture, gait, colour of iris and hair, mannerism, etc. and with this we infer that the child is of such a parent. However, mere resemblance is not reliable. It is only considered to be of corroborative value.
2. ***Atavism*** – at times the child may not resemble the parents but grandparents. This is called atavism. It is also of corroborative value.[19]
3. ***Blood group tests*** – blood group of an individual is of a hereditary transmission origin from a parent to the offspring. Hence, determination of blood group of a child and parents is of help in establishing the paternity. Table 26.4 summarizes the possible children for specific blood group parents.
4. ***Determination of nonpaternity*** is also established if the alleged/*putative father* is:
   - Impotent or sterile
   - Had no access to his wife
   - Blood groups of the child and father are inconsistent
   - Racially not similar to that of child.

Medicolegal Importance

- In case of legitimacy and disputed paternity
- In case of fictitious child
- ***Superfecundation:*** This is a condition wherein fertilisation of two ova, discharged in the same ovulatory period, occurs by different acts of coitus resulting in birth of *twins*. If by

Table 26.4: Possible (blood groups) children for specific blood group parents

Blood groups of parents	*Possible groups of the child*	*Blood groups of child not possible*
O + O	O	A, AB, B
O + A	O, A	B, AB
O + B	O, B	A, AB
O + AB	A, B	O, AB
A + A	A, O	B, AB
A + B	A, B, AB, O	None
B + B	B, O	A, AB
AB + AB	A, B, AB	O

any chance the woman had coitus with two different men of different race, twins born will be of different race.[1,4,6,9]

- ***Superfoetation:*** this is a condition wherein fertilisation of two ova, discharged in two different ovulatory periods, occurs by two different acts of coitus, resulting in *twins* at birth, one of which will be always older than the other.[1,4,6,9]

LEGITIMACY

A child born during the continuance of a legal marriage is considered a legitimate child. A child born to a couple who are not married legally is considered to be illegitimate or a bastard.

Presumption in Favour of Legitimacy

The law in India has accepted certain presumptions in favour of legitimacy, based on the principle that the law is averse to declare a child a *bastard*. These presumptions are:[1-7,10-13]

- Child born to a woman who is living with her legally wedded husband, but the offspring is in reality a product of her illicit intimacy with a paramour, is still considered as legitimate child, till the contrary is proved in the court.
- A child born to a woman within 270 days of divorce is considered as legitimate child until the contrary is proved.
- A couple indulges in sexual intimacy prior to marriage and consummation takes place. However, they get married later and the child is born soon after marriage, such child is also presumed to be legitimate until the contrary is proved (example of William Shakespeare in England).

Medicolegal Importance

Legitimacy may have to be decided in cases of:

- Affiliations – according to the law, father of an illegitimate child, must arrange to maintain it.
- Inheritance – a legitimate child alone can inherit the property
- *Fictitious child*.

SEXUAL OFFENCES

Sexual offences are almost of infinite variety of physical acts by a person with another person or animal, either executed or attempted in the furtherance of sexual gratification.

Classification

Sexual offences are of three types:[1-6] (i) Natural sexual offences, (ii) Unnatural sexual offences, and (iii) Sexual deviations or perversions (Table 26.5). Unnatural sexual offences and sexual deviations together are often referred as *sexual paraphilias*.

Natural Sexual Offences

All such physical acts executed within the order of nature's accordance in furtherance of sexual gratification are considered as natural sexual offenses. They include:

- Rape
- Incest

Unnatural Sexual Offences

All such physical acts executed against the order of nature's accordance in furtherance of sexual gratification are considered as unnatural sexual offenses. They include:

- Sodomy, Lesbianism, Buccal coitus, Bestiality.

Sexual Deviations (Sexual Perversions)

All such physical acts executed which are not only against the order of nature's accordance, but also against human biology in furtherance of sexual gratification are considered as sexual deviations. They include:

- Eoneism, exhibitionism, fetishism, masochism, masturbation, nymphomania, necrophagia, necrophilia, satyriasis, sadism, transvestism, troilism, undinism, voyeurism, etc. (see Table 26.5).

RAPE

Legal Definition

A man is said to commit "rape" if he has sexual intercourse with a woman under circumstances falling under any of the six following descriptions:[20,21]

- *First*– Against her will.
- *Second* – Without her consent.
- *Third* – With her consent, when her consent has been obtained by putting her or any person in whom she is interested in fear of death or of hurt.
- *Fourth* – With her consent, when the man knows that he is not her husband, and that her consent is given because she believes that he is another man to whom she is or believes herself to be lawfully married.
- *Fifth* – With her consent, when, at the time of giving such consent, by reason of unsoundness of mind or intoxication or the administration by him personally or through another of any stupefying or unwholesome substance, she is unable

Table 26.5: Sexual offences encountered routinely

Natural	*Unnatural*	*Sexual perversions*
Rape Incest	Sodomy Lesbianism Buccal coitus Bestiality	Eoneism Exhibitionism Fetishism Masochism Masturbation Nymphomania Necrophagia Necrophilia Satyriasis Sadism Troilism Undinism Voyeurism

to understand the nature and consequences of that to which she gives consent.

- *Sixth* – With or without her consent, when she is under sixteen years of age.

Explanation: Penetration is sufficient to constitute the sexual intercourse necessary to the offence of rape.

Exception: Sexual intercourse by a man with his own wife, the wife not being under fifteen years of age, is not rape.

In the Manipur State, rape law has reduced the ages of valid consent to sexual intercourse by unmarried and married woman to 14 and 13 years, respectively.

Rape and Law

a. Explanation of rape definition — Rape is said to have been committed when a man has sexual intercourse with a woman:
   - With or without her consent, when she is below the age of 16 years.
   - With consent when she is:
     — His own wife, but below the age of 15 years.
     — Mentally ill.
   - With consent when she is above 16 years but the consent is obtained by:
     — Fear – threatening to kill her
     — Fraud – pretending to be her husband
     — Intoxication – with intoxicating agents like alcohol.

b. Rape and gender – in law, rape is an offense, which can be committed only by man.
   - *Reasons*—In sexual intercourse, man is considered to take an active role rather than a woman.

c. Rape and degree of penetration – Penetration is sufficient to constitute the sexual intercourse necessary to the offence of rape. The depths of penetration, seminal emission, rupture of hymen, etc. are not considered as important factors in justifying the offense of rape.
   - *Reasons*—In a child victim actual penetration may not be accomplished due to the disproportion of the sex organs, but other injuries might have resulted due to the force used, e.g. perineal tears, contusions of the labia, etc.

d. Rape and resistance – depending on the age, build, health and social status, a victim can usually offer resistance prior to the actual act resulting in marks of struggle or struggle evidence such as nail scratches, abrasions, bruises, bite marks, etc. These marks of struggle constitute good corroborative evidence in favour of rape.

e. Consent for sexual intercourse – consent becomes valid only if the following criteria are fulfilled:
   - She must be 16 years or above by age
   - She must give it prior to the act
   - She must give it voluntarily and freely
   - She must be "compos mentis" and not intoxicated. Thus, even a prostitute can plead for being raped against

   a man who had coitus with her without her consent.

f. Age and rape
   - Age of assailant – in Indian law, a male of any age is considered eligible for sexual intercourse (in England, male above 14 years is only deemed to be fit).
   - Age of victim – no age in a female is free from the fear of rape. However, child victims are often preferred by a rapist and reported frequently for the reasons such as:
     – They offer little resistance
     – They can be seduced easily
     – They can be threatened successfully and keep the event secret
     – For a "false belief" of curing the venereal diseases, as practiced in some remote villages in rural India even today.

Punishment for Rape

Rape is a cognizable offense. IPC section 376 defines the punishment of rape.[20] Whoever commits rape shall be punished with imprisonment of either description for 7 years but which may be for life or for a term which may extend to 10 years and shall be liable to fine unless the victim is his own wife and not under 12 years of age, in which case, he shall be punished with imprisonment up to 2 years or with fine or both.

Dangers of Rape[1-16]

- Shock due to fear, may turn fatal or when the victim survives, may make her mentally deranged temporarily or permanently
- Haemorrhage, due to genital injuries, may be fatal when severe
- Accidental death, e.g. Suffocation – the assailant may cover his hand over her mouth and nostrils preventing the victim from shouting or screaming for help; can result in suffocation and death
- Homicidal death, e.g. Strangulation – in order to conceal the event, the assailant may kill his victim after rape
- Psychological trauma; parasuicide/suicide – out of frustration of being raped, the victim may end her life.

EXAMINATION OF CASE OF SEXUAL ASSAULT

Apart from the medical responsibilities, the duties of medical investigator include the examination of the victim (alive or dead) and the accused to gather evidence to corroborate the charges and to apply in the adjudication of the complaint. The method of performing the examination may differ from case to case; a general plan of examination consisting of examination of the scene, medical examination of the victim and the suspect or accused is discussed below:[22]

Examination of the Scene

Although examination of the scene is primarily the responsibility of police, it will be worthwhile for a forensic pathologist to visit the crime scene personally, especially in cases where the victim is dead.

Medical Examination of the Victim

Avoid unnecessary delay. Examination of the victim (Figs 26.14A and B and 26.15A and B) of rape constitutes three steps (remember 3Gs):

- General Procedures/Preliminaries
- General Examination (Fig. 26.14A)
- Genital/Local Examination (Fig. 26.14B)

I. General Procedure/Preliminaries:

- *Requisition:* Requisition for examination of the victim can be obtained from the Investigating Officer (IO) or from Judicial Authority. Examination can be carried out only after the receipt of the requisition from the concerned legal authorities. Ideally it should contain the bio data of the victim including residential address, name of the Police Station along with FIR number, identity of the escorting police, a brief history of the case and queries from the IO.
- *Date, time and place of examination:* should be recorded.
- *Identity of the victim:* The victim should be physically identified by the escorting police and by any

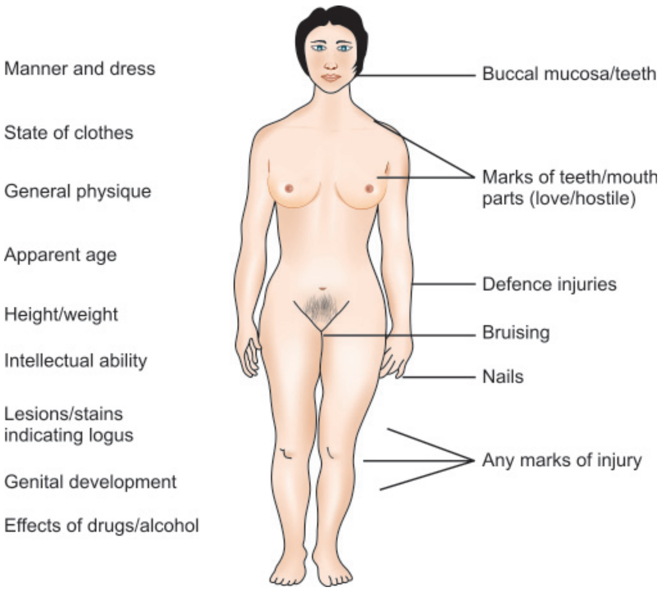

Manner and dress
State of clothes
General physique
Apparent age
Height/weight
Intellectual ability
Lesions/stains indicating logus
Genital development
Effects of drugs/alcohol
Buccal mucosa/teeth
Marks of teeth/mouth parts (love/hostile)
Defence injuries
Bruising
Nails
Any marks of injury

Fig. 26.14A: Examination of the victim of rape—general physical examination

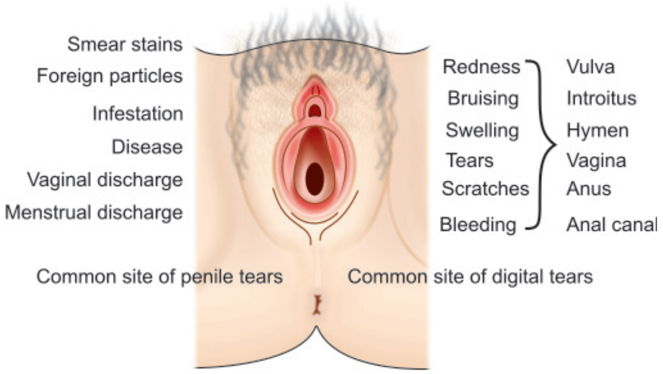

Smear stains
Foreign particles
Infestation
Disease
Vaginal discharge
Menstrual discharge
Redness
Bruising
Swelling
Tears
Scratches
Bleeding
Vulva
Introitus
Hymen
Vagina
Anus
Anal canal
Common site of penile tears
Common site of digital tears

Fig. 26.14B: Examination of the victim of rape—genital findings

accompanying guardian before the medical officer prior to the examination.

- *Consent:* Victim of sexual assault cannot be examined without her consent. Depending on the age of the victim, informed written consent for medical examination can be given by herself or by her guardian.
- The examination of the victim should always be done in presence of a female third person, e.g. nurse, female attendant.
- *Second opinion:* Never hesitate to take second opinion from a qualified person if necessary.
- Report all findings properly
- *Prepare three copies:* Two copies for submission and one for the office file. A sample of standard proforma of examination of a case of rape is provided in Proformas 26.1 to 26.3.

History

- – *History of the incident and post-incident events:* Date, time, place of alleged occurrence, alleged suspect's name if known, description of the alleged suspect, detailed circumstances of the incident, drug and/or alcohol influences, damage to clothing and injuries to person.
- – *Sexual history:* Any past history of involuntary/voluntary intercourse including date, time and place of the last act.
- – *Medical history:* Menses: If menarche, regularity, interval, duration and last menstrual period, vaginal discharge; Pregnancies; STDs, Illnesses including medical care and physician; Surgical operations including sterilisation.
- – *Personal history:* Change of clothing, Vaginal douching or taking bath after the incident; Habits: use of alcohol, drugs, etc.

II. General Examination:

- *Examination of clothing:* The victim should ideally undress herself whenever possible or otherwise assisted by the third party. The person is made to stand over a wide white paper/cloth (Catch paper/cloth) to collect any trace evidence that may dislodge while undressing (Figs 26.15A and B).
  - – Manner and state: Disturbed, shabby, etc.
  - – Damage: Tears, loss of buttons.
  - – Stains: Body fluids (Blood, semen, saliva, urine, faecal matter), mud, etc.
  - – Other trace evidence: Hairs, fibers, grass, etc.
  - – Note the demeanour and emotional state, gait.
  - – Collect stains and any other trace evidence present on the body adopting standard procedures (Figs 26.15A and B).
  - – Record vital signs.
  - – Look for presence of *signs of struggle*, violence which may present in any form (Fig. 26.16) such as abrasions (nail scratches), bruises, bite marks, lacerations, incised and stab wounds, etc. Any injuries present should be properly documented, incorporating sketches or photographs whenever possible, giving exact location, detailed description: size, shape, type of injury, age of injury, etc.

III. *Genital/Local Examination:* (see Figs 26.14B; and 26.15B)

Prerequisites:

- Position: proper examination is possible only by making the patient adopt the lithotomy position (Fig. 26.1C).
- Proper illumination: can help good observation.
- Local anaesthesia: use of this may be beneficial when the victim is complaining of severe pain.
- Examination proper: findings differ depending on the victim who could be a virgin, deflorated woman, or a child (Figs 26.14 to 26.22).

Findings in a Virgin Victim

All findings are described as typical findings of rape and become corroborative evidence in law and these are (Figs 26.16 and 26.17A to C):

- *On the vulva* – redness, bruises, swellings, tears, scratches, bleeding, etc. (Figs 26.17A to C).
- *With the hymen* – recent rupture is of maximum corroborative value. Note the site and degree of tears. Hymen examination kit should be used (see Fig. 26.6).
- *In the vagina* – bruises, tears, bleeding, discharges (venereal origin), foreign particles, etc. (menstrual flow may be there if she is in her menstruating period).

Proforma 26.1: Examination of rape victim

Requisition from SI of..............................Police Station with his Letter No. .... Dated .... for examination of ....in charge of PC No. ....

1. Name and address:
2. Age as stated by:
3. Occupation:
4. Married or not:
5. Number of children if married:
6. Persons accompanying and their relationship:
7. Consent—Obtained from parents in the case of minor girls. Always get signature. However explain that the physical findings observed during examination will be used as evidence during trial whether or not it is in his interest and he is free to refuse being examined if he chooses.
8. Nurse or other female present
9. Marks of identification
10. History as given by the police
11. History as given by parents/relatives
12. Statement of the female with regard to the following:
    - Date, time and place of occurrence
    - Exact position of the parties
    - Did she struggle or cry for help?
    - Was she menstruating or not?
    - Was she conscious the whole time?
    - Did she urinate or not? Pain?
    - Did she change her clothes?
13. Date and time of lodging a complaint, explain delay
14. Date and time of physical examination
15. Mental disposition. Excited or calm
16. Gait. Does she walk as if in pain?
17. Clothes—look for blood, semen, hair, tears, loss of buttons, mud, grass, etc. Describe location and extent of each.
18. Physical development—height, weight, build.
19. Marks of general violence—look for abrasions or contusions of face, back of the shoulder, arms, and thighs.
20. Breasts—look for contusion, abrasion, and bitten nipples.
21. Pubis, perineum, thighs—look for stains, matting of hair, scratches.
22. Vulva—look for bruises, abrasions
23. Hymen—present or replaced by carunculae, if present—type, position of natural opening, whether torn/intact, if torn—position, extent and age of tear.
24. Fourchette—intact/torn.
25. Vagina—look for bruises, tear, nature of discharge
26. Veneral disease—gonorrhea/syphilis—get specialist’s opinion if it is necessary and possible.
27. Vaginal smear—for spermatozoa, blood.
28. Preserve following material for chemical examination
29. Clothes are dried to prevent decomposition of stains. Put in a cardboard box, seal, and label.
30. Take vaginal fluid with a swab. Rub on two sides. One side may be examined immediately. The other one is dried well, covered with cotton and wrapped in paper, sealed and labeled. Get authorization from the investigating officer and send the materials for chemical examination.

- *In the perineum*—tears (especially the fourchette), scratches, bruises, etc (see Fig. 26.17C).

Findings in Deflorated Woman Victim

Typical findings described in the virgin victim may not be elicited in a deflorated woman victim. However, presence of the following is important in such cases:

- Semen in the vagina (in fornices or vulva or garments worn, confirmed by vaginal smear) (see Fig. 26.22).
- Evidence of struggle is more important.

Findings in a Child Victim

Typical findings described in the virgin victim may not be elicited in a child victim, due to anatomical disproportion in genitals

Proforma 26.2: Examination of accused of rape charge

Requisition from SI of ....Police Station, with his Letter No .... Dated .... for the examination of .... and in charge of PC No. ....

1. Name and address
2. Age ....years
3. Occupation
4. Consent for examination obtained from (get signature)

   Note: Explain to the person that the physical findings observed during examination will be used as evidence during trial whether or not it is in his interest and he is free to refuse being examined if he chooses.
5. Identification marks
   a.
   b.
6. History as given by the Police
7. Statement of the individual
8. Gait
9. Clothes (look for blood, semen, tears, mud, grass, etc.) Did he change the clothes or wash his parts?
10. Date and time of examination
11. Physical development

    (Look for any general violence on the body as bite marks, scratches, contusions, etc. indicative of resistance from the female)
12. Pubic region and thighs (look for matting of hair, stains).
13. Penis—look for any of the following evidence of impotence (general examination is required if he pleads impotence - as defense.
    a. Evidence of venereal disease (get expert's opinion)
    b. Smegma (retract prepuce and see)
    c. Frenulum (torn/intact)
    d. Paraphimosis (present/not)
    e. Glans penis (look for abrasions)
    f. Foreign hair underneath the prepuce (preserve if any for comparing with pubic hair of the female).
15. Microscopic examination of discharge
16. Preserve clothes stained with blood and semen for chemical examination.

    Note: When the physical examination is over and the necessary articles have been preserved for chemical examination, a certificate is issued in either case (victim and suspect).

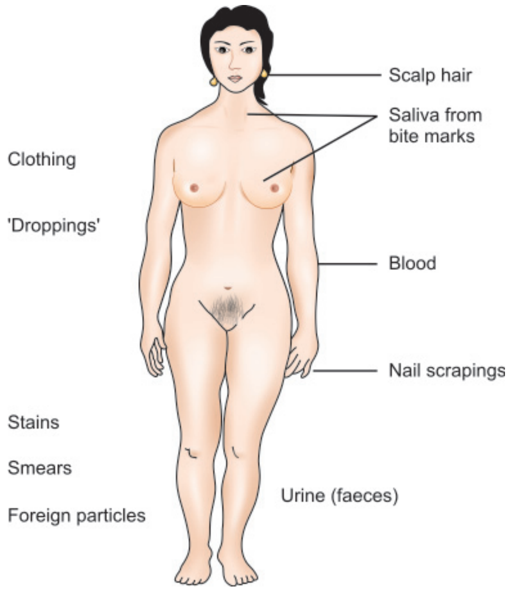

Scalp hair
Saliva from
bite marks
Clothing
'Droppings'
Blood
Nail scrapings
Stains
Smears
Foreign particles
Urine (faeces)

Fig. 26.15A: Examination of the victim of rape—specimens (trace evidence) general/physical

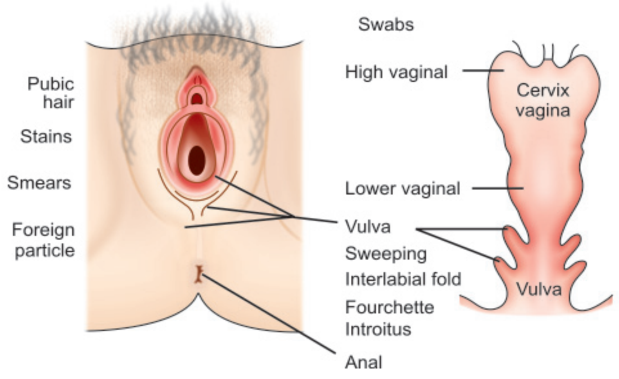

Swabs
Pubic
hair
Stains
Smears
Foreign
particle
High vaginal
Cervix
vagina
Lower vaginal
Vulva
Sweeping
Interlabial fold
Fourchette
Introitus
Anal
Vulva

Fig. 26.15B: Examination of the victim of rape—specimens (trace evidence) genital

of victim and accused. However, presence of following is important in such cases:[1,4,5]

- Inflammation/abrasion/bruises of vulva
- Inflammation of urethra
- Hymen – intact/torn/destroyed

Proforma 26.3: Report/certificate on sexual offences

Appearances found on the person of a male/female named,................... aged ....years, an inhabitant of...................sent by SI of .... Police Station, with his Letter No......dated.................accompanied by PC No. ....for examination and report for certain injuries or other findings said to have been caused of ....and to be due to rape/sodomy.

Identification marks

a.

b.

The person was first seen by the undersigned at.....on.......................and the examination was conducted at ....on...............when the following were found (state the essential findings)...

Station:	Signature of MO
Date:	Name
	Designation
	Address
	Rubber Stamp

Note:

- It is not advisable to state whether rape had been committed or not. Medical evidence should always be analyzed in the light of circumstantial evidence and is done during trial.
- The certificate is issued as soon as the examination is over. The detailed report prepared during examination is kept with the doctor and can be used for refreshing memory during trial.
- As soon as the report of the chemical examination is available, it should be forwarded to the Investigating Police Officer, after recording the essential findings in our note.
- When the age is disputed, determine age as directed elsewhere.

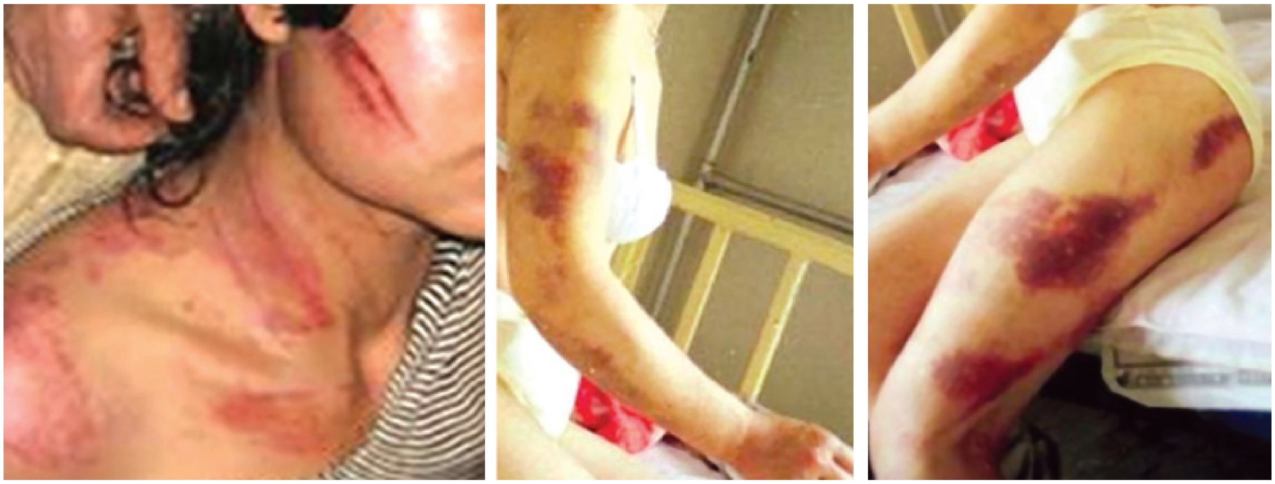
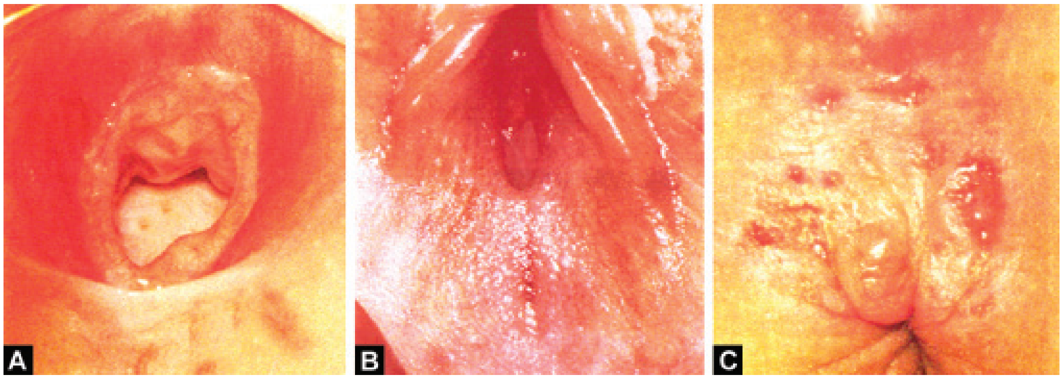


Fig. 26.16: Victim of rape. Note the *struggle marks* on the body of the victim



Figs 26.17A to C: Genital examination findings of rape injuries in a virgin victim: (A) Redness, bruising, ecchymosis, swelling of vulva, tearing of hymen at 7 o'clock position; (B) Scratches and redness and swelling of labia minora; (C) Swelling, redness, scratches, bruise in the fourchette-perineum[24] (*Source:* http://www.medical-library.org/journals5a/rape–pictures.htm)

- Perineal tears – uncontrolled bleeding/clotted blood.
- Discharges of lesions of STD such as gonorrhea, syphilis, etc.

Collection of Evidence: Rape is an excellent example for *Locard's Principle of Exchange,*[1,4,5] which states that every contact leaves a trace. Physical and biological evidence play a pivotal role in the objective and scientific reconstruction of the events in question. The evidence should be collected from the victim, from the crime scene and from the suspect employing standard techniques. Each sample should be packed using appropriate packaging materials, labelled, sealed and stored as per specification before transporting it to the laboratories. Documented chain of custody of the evidence should be maintained strictly at every level to ensure the authenticity of the evidence.

1. Stains and foreign materials present on the clothing or body.
2. Fingernail scrapings.
3. Brushing/combing of the person's hairy region: head, body and pubic.
4. Samples of the person's hair: head, body and pubic.
5. Urethral, perianal, vulval swabs, vaginal content aspiration and swab, and cervical swab to be collected under direct visualisation. Ideally it should be collected prior to the examination to avoid contamination.
6. Sample of blood.

Laboratory Investigation: In majority of sexual assault cases, the physical evidence generally encountered is: Blood, Semen and Saliva. There is an array of analytical tests for these physical evidences; however, it is beyond the scope of this book to consider each and every test. Several tests for detection of semen are mentioned below for the benefit of the readers, as even with limited resources, some of the tests can be conveniently performed.

DETECTION OF SEMEN

The type of physical evidence most frequently associated with sexual assault cases is semen. The very presence of semen is indicative of the occurrence of sexual activity.[1-6, 26]

Process of Collecting Biological Samples

a. *Dried Stains:* Application of absorbent swabs moistened in distilled water or normal saline.
b. *Wet Stains:* Under direct visualisation any liquid secretions in the body cavities can be collected by aspiration or insertion of dry absorbent swabs.

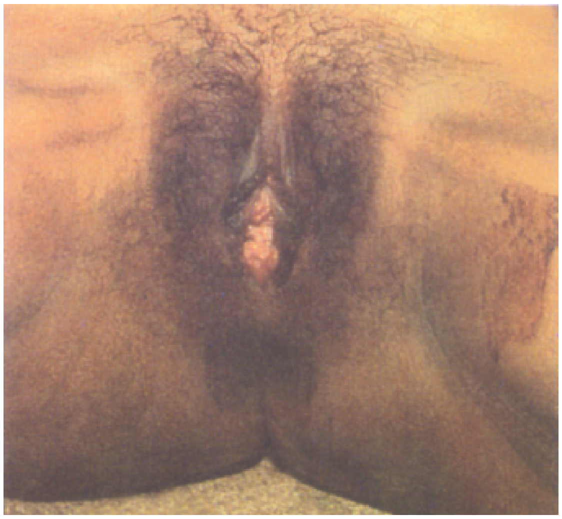


Fig. 26.18: Victim of rape: Genital injuries with blood stains on the thigh (*Courtesy:* Dr KWD Ravi Chandar, Prof and HOD, Forensic Medicine, MMC, Mysore)

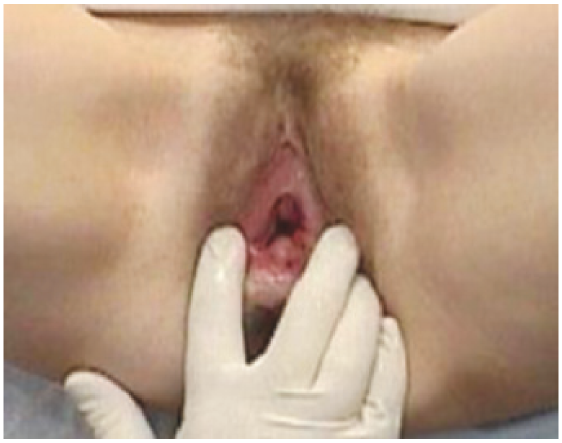


Fig. 26.19: Victim of rape: Rape and murder. Forceful sexual intercourse results in ecchymosis in parts of genital organ, and tearing of hymen at 5, 6 and 7 o'clock position

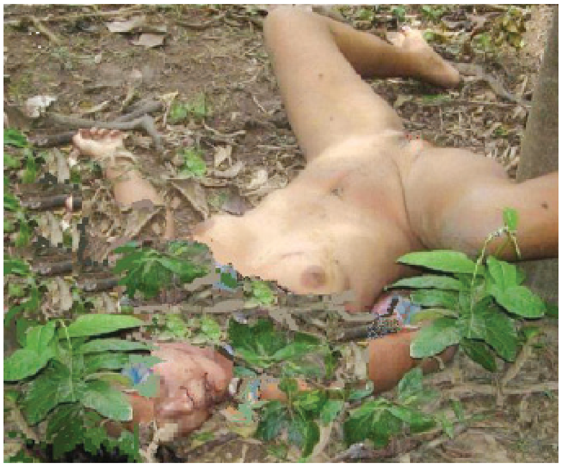


Fig. 26.20: Victim of rape and murder: Nudeness, presence of vegitation and soil, bleeding perineum are diagnostic of rape

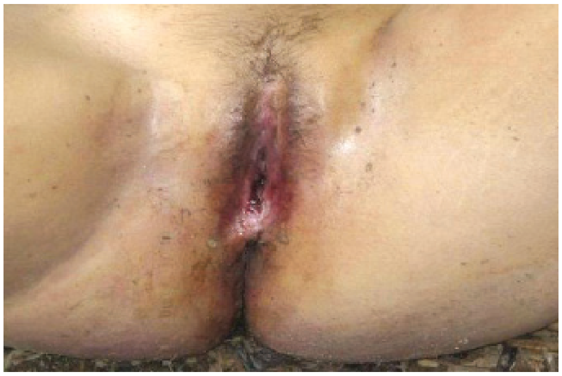


Fig. 26.21: Virgin victim of rape (same as in Fig. 26.20): swelling, gaping of vulva and perineal tears corroborates sexual assault

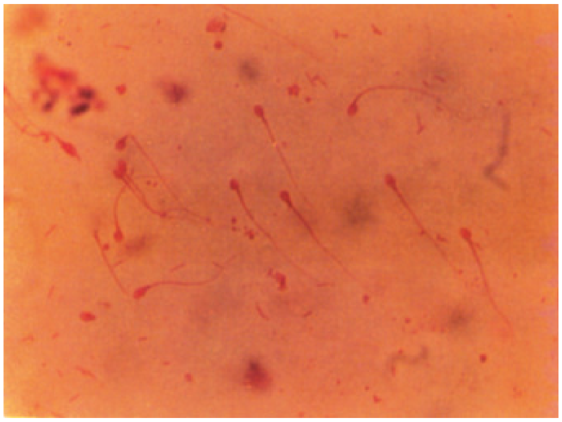


Fig. 26.22: Vaginal smear showing spermatozoa (*Courtesy:* Dr Udaypal Singh, KMC, Warrangal, Andhra Pradesh)

Screening Tests

For identification of occult seminal stains, to confirm the sampling location, and as presumptive test for semen.

1. *Ultraviolet Light Scanning:* Helps in identifying occult semen stains. Dried seminal stains fluoresce under UV illumination.
2. *Chemical Tests:*
   - *Barberios' test:* Detects the presence of spermine, one of the constituents of semen. One drop of the stain extract is placed on a glass slide under a cover slip. Then it is charged with a drop of saturated aqueous solution of picric acid (1 g picric acid in 30 ml of distilled water) and allowed to diffuse uniformly. Examine under 100× objective of a microscope for needle shaped yellowish spermine picrate crystals indicating presence of semen.
   - *Fluorescent test:* Detects the presence of choline, one of the constituents of semen. One drop of stain extract is placed on a glass slide under a cover slip. Then it is charged with a drop of Fluorescent reagent (1.65 gm potassium iodide, 2.54 gm of iodine and 30 ml of distilled water) and allowed to diffuse evenly. Examine under 100 × objective of a microscope for brown rhombic or needle shaped crystals of choline per-iodide, indicative of semen.
   - **Seminal Acid Phosphatase (SAP)**
     It is an enzyme present in varying amounts in different body fluids. Seminal fluid has a high concentration of SAP- 400-8000 King Armstrong (KA) units and its activity in human semen is 500-1000 times greater than in any other human body fluid. The enzyme has the property to cleave a variety of organic phosphates, based on which several tests are available, e.g. Brentamine test, P-nitrophenyl phosphate, alpha-naphthyl phosphate, thymolphthalein monophosphate, etc. These tests are sensitive but not specific for semen owing to the presence of the enzyme in other tissues including vaginal fluid. Besides, there is considerable variation depending on a number of factors- pregnancy, phase of menstrual cycle, bacterial vaginosis, etc. Presence of more than 25 kA units per ml of extract from 1 sq cm of the stained area is considered to be positive reaction and consistent with the presence of semen.
   - **Other markers** like creatinine phosphokinase, lactate dehydrogenase isoenzymes, etc. have also been employed for detection of presence of semen in the stain extract.

Confirmatory Tests

The seminal stain is processed for extraction, which yields a supernatant and a cell pellet. The cell pellet is used for detection of spermatozoa and for DNA analysis while the supernatant portion is used for detection of noncellular markers in semen and to develop genetic profiling or grouping.

1. **Microscopic Examination**: Depending on the time elapsed since the crime, spermatozoa may be alive and motile or dead. Identification of one or more spermatozoa is conclusive proof of the presence of semen and affirms sexual contact.
   - **Motile sperm**: It is best accomplished when the examination is done at the time of collection of the evidence. The technique requires preparation of a wet mount slide (vaginal or cervical swab sample) placed on a slide with a drop of saline covered by a cover slip and examined under a phase-contrast microscope.
   - **Nonmotile sperm**: Can be detected from the examination of stained smear preparation. Smear is prepared at the time of collection of the evidence from the swabs or from the cell pellet of the stain extract. Commonly employed staining methods include Gram's, Hematoxylin and Eosin, Papanicolaou (PAP smear) and Oppitz (Christmas-tree-stain) stains. To prevent artefact from the selective degradation of cellular debris, the cell

extract can be treated with a mixture of proteinase K and sodium dodecyl sulphate before staining and microscopic examination. The slides are preferably examined under 400X light microscope.

2. **Noncellular semen markers**: To detect the presence of specific and unique seminal plasma markers.
    - **P30 (Prostate Specific Antigen/PSA)**: It is a glycoprotein derived from the prostate epithelial cells and is found in seminal fluid, male urine and blood, but not found in any female tissue or body fluids. Also there is no significant difference of P30 level between vasectomised and nonvasectomised individuals.[27]
    - **Monoclonal Antibody Mouse Antihuman Semen-5 (MHS-5)**: It is secreted by the seminal vesicle epithelium and is not found in any other body fluid besides semen and has no cross reactivity with other body fluids.

Individualisation of Semen Evidence: On confirmation of the presence of semen in the extract, further tests are subjected to individualise the semen. It is achieved by typing the questioned biological stains in various genetic markers system and compared to reference samples obtained from individuals who may be possible donors of the stain. Based on the results obtained, an individual is either included or excluded as being the stain donor. Genetic markers are inherited biochemical substances that exhibit variation (polymorphism) in the population.

BLOOD GROUP TYPING

ABO group typing can be done only among secretors. Secretors are those individuals who secret soluble ABO agglutinogens in their body fluids. They comprise about 80 per cent of the population. Seminal ABO group typing can be done by several methods: absorption-elution, absorption-inhibition or mixed agglutination. Blood group isoagglutinins can be determined by Lattes Crust method. As with other marker assays, temporal, qualitative, methodological and physiologic variables may limit the usefulness of the blood group contribution to the genetic profiling. The advantage of traditional grouping is—the procedures are cheap, universally available and it may be the only option in case where the stain contains few or no cells.

Enzyme Typing

Enzyme markers commonly used in genetic profiling of semen are *phosphoglutamase (PGM)* and *peptidase (Pep A)*. These enzymes are found in semen and vaginal secretions regardless of ABO type or secretor status.

PGM is polymorphic in all populations and can be subdivided into 10 distinct subgroups. **Pep A** is polymorphic in many racial groups and is commonly employed as a discriminator in cases where the perpetrator is suspected to be black.

DNA Profiling

The primary advantage of DNA profiling in sexual assault investigation is its ability to accurately individualise semen that contains only minimal number of spermatozoa. Besides it can also differentiate multiple donors in mixed stains.

POSTCOITAL INTERVAL (PCI)

It is the time lapsed since last act of sexual intercourse (time interval between the deposition and collection).[28] PCI is determined from the persistence of the seminal materials—spermatozoa, P30, SAP, PGM and Pep A depending upon their deferential stability. Thus, a significant level of P30 tends to be lost within 24 hr of deposition in the vaginal vault (as measured by immunodiffusion or crossed-over electrophorosis), SAP is normally lost 48 hours postcoitus, PGM drops below threshold by 6 hours, Pep A is not usually recovered after 3 hours and spermatozoa do not normally persist after 72 hours. In deceased individuals these seminal compounds can last for several days, depending upon the environmental condition and the rate of decomposition.

SIGNIFICANCE OF MEDICAL EXAMINATION OF SEXUAL ASSAULT

Medical evidence of rape can be derived from:

- Presence of stains of body fluid and foreign materials in the clothing and body including genitalia of the victim or vice versa.
- Presence of marks of struggle or violence in the body and clothing inflicted by the accused or vice versa.
- Evidence of injuries in and around genitalia of the victim.
- Presence of semen in the vagina of the victim.
- Evidence of implanted venereal disease on the body of the victim.

However, in the legal context, to constitute the offence of rape there need not be full penetration of the vagina by the penis with emission of semen and rupture of hymen. Even the slightest penetration or even touching the vulva without producing any injury to the genitals including hymen or leaving any evidence of seminal emission will be sufficient to constitute the offence. Thus the medical proof of sexual intercourse is not the legal proof of rape.

The multitude of individual variations and complexity of mechanism that comes into play at the time of commission of the crime may weaken the medical evidence, yet it is not excluded in the court of law. In many instances such as, the proof of seminal emission in a victim who is below 16 years of age or injuries in the genitals provides the proof of rape. Besides, establishing that sexual contact has in fact occurred, the role of medical evidence in sexual assault crimes not only helps to associate the victim and suspect with one another, but also to corroborate or dispute accusations by the parties involved.

Examination of the Accused

When the accused of a rape case is produced for medicolegal examination, it is conducted under section 53 and 54 CrPC.[29] Except some differences, the procedure is almost the same as with examination of rape victim (remember the 3G's) and report (see Proformas 26.1 to 26.3):

- General procedures (Prelimiaries)—refer above
- General examination–refer above (Figs 26.24 to 26.26)
- Genital/local examination.

Genital Examination (Fig. 26.25A): Elicit the following:

1. Development of genitalia
2. Injuries suggestive of forcible sexual intercourse such as:
    - Bruises, nail scratch marks, etc. by the victim in resisting the act of rape
    - Tearing of prepuce, frenulum, etc. due to forcible penetration.
3. Swabs from the urethra, shaft and glans are collected in addition to the above-mentioned physical evidence.
4. *Lugol's iodine test*

    Principle: During sexual intercourse, vaginal epithelial cells, which are transferred to glans penis are confirmed by this test.

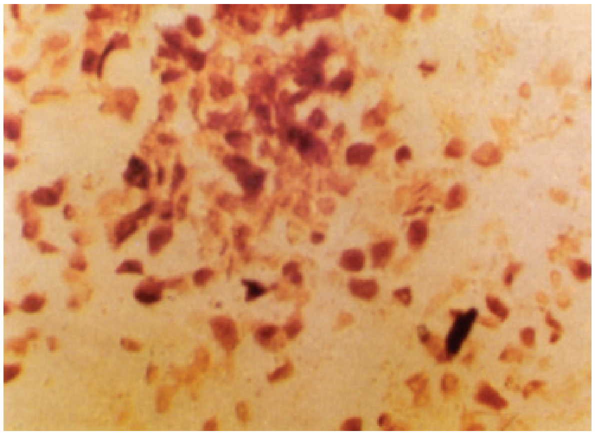


Fig. 26.23: Vaginal epithelial cells stained brownish with Lugol's iodine (*Courtesy:* Dr Udaypal Singh, KMC, Warrangal, Andhra Pradesh)

Procedure: It is a simple test, wherein the glans penis is mopped with a filter paper and the paper is then exposed to Lugol's iodine vapor. A brownish discoloration on the paper is suggestive of presence of vaginal epithelium, which is rich in glycogen content, responsible for color change on the paper (Fig, 26.23) shows vaginal epithelial cells stained brownish by Lugol's iodine.

Absence of smegma: Normally the glans penis when covered with prepuce will allow deposition of smegma in it. During sexual intercourse smegma gets washed off in vaginal secretions. Hence, testing the washings of glans for absence of smegma corroborates rape (smegma can be washed off at bath). If smegma is present it rules out sexual act, within 24 hours) (Fig. 26.25B).

Feigned Rape

Condition wherein a woman pretends to have been raped is known as feigned rape. The concept of rape is deceptively simple, and women who make false allegations often structure their complains in such a way that they seem to meet the requirements of rape but ignore its reality.

Investigating Suspected False Rape Allegation:

1. Motive for this being several are enumerated as:
   - When her illicit sexual activities are exposed.
   - When she becomes pregnant out of illicit sexual relationship
   - Taking revenge on a boy.
2. History not consistent.
3. Crime scene does not support story.
4. Damage to clothing is inconsistent with any injuries.
5. Injuries not consistent with history, defense wounds, do not involve sensitive tissues.
6. Injuries are of self-inflicted type.
7. Fingernail scrapings of the victim reveal her own skin tissue.
8. Confirmatory laboratory findings are negative.
9. Dubious personality and lifestyle.

Medicolegal Questions on Rape

- Rape and resistance (refer above)
- Raping a woman in deep sleep—it is impossible to rape a woman in sleep, as usually sexual intimacy can wake up the woman.
- Anaesthesia and rape charges—often after anesthesia, a woman may feel that she has been raped. Hence, it is better to give anesthesia in the presence of a nurse.

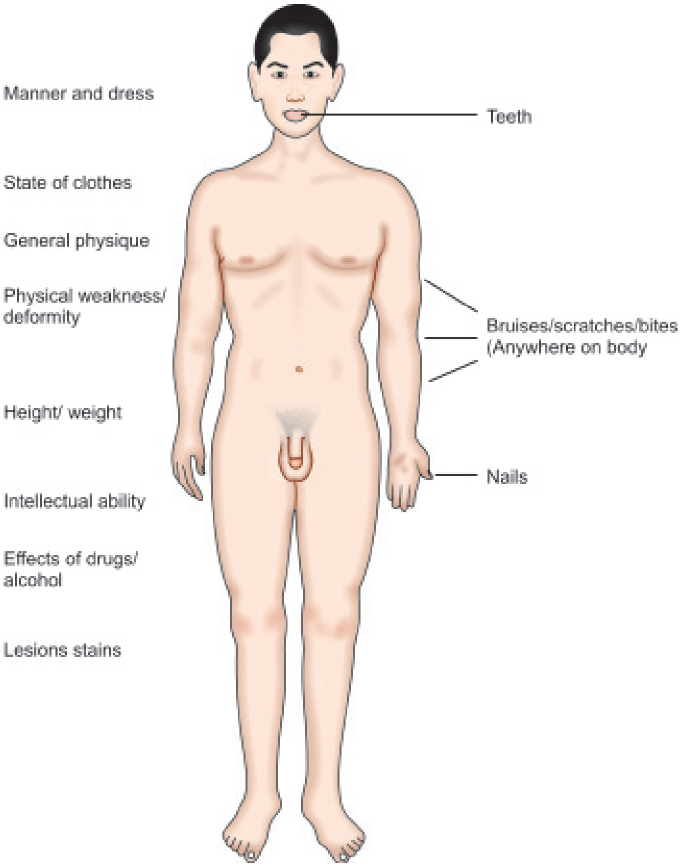

Manner and dress
State of clothes
General physique
Physical weakness/ deformity
Height/ weight
Intellectual ability
Effects of drugs/ alcohol
Lesions stains
Teeth
Bruises/scratches/bites (Anywhere on body
Nails

Fig. 26.24: Examination of the accused (male) of rape—general/physical examination

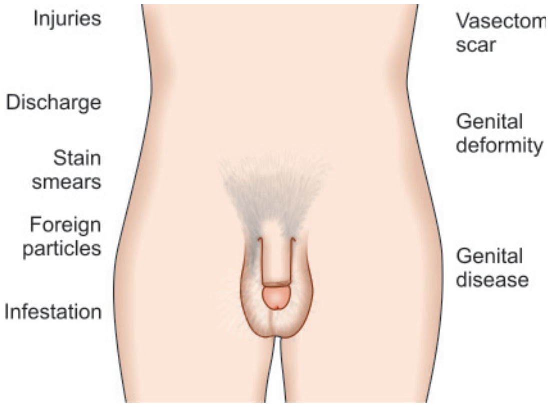

Injuries
Discharge
Stain smears
Foreign particles
Infestation
Vasectom scar
Genital deformity
Genital disease

Fig. 26.25A: Examination of the accused (male) of rape—genital examination

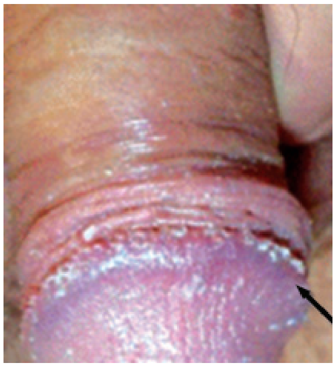


Fig. 26.25B: Smegma on the glans penis (Arrow)

- Rape by false impersonation (fraud)—cases of sexual intercourse by false impersonations are often reported, especially if the woman had never or only once seen her husband.
- Rape by misrepresented facts—especially with young innocent girls, this is quite often a possibility.

Case Example An innocent young girl with suppressed menses was convinced by doctor that the act he is going to perform would cure the problem, performed sexual intercourse with her, which not only amounts to rape but also infamous conduct (adultery) and punishable.

- Death during sexual intercourse – though this is rare, it is not impossible.
- Death following rape (refer above).

Standard proforma of examination of a rape case is provided in Proformas 26.1 to 26.3.

INCEST

Incest means an offence wherein sexual intercourse is practiced between a man and a woman who are within restricted relationship in a family (blood relation) and society such as father and his daughter, mother and her son or brother and his sister, etc.

Medicolegal Importance

It is prohibited and consent given by woman here is not a defense. This is not punishable in India, unless it amounts to rape.[20,29] However, such cases may occur more in number than what is reported.[1]

UNNATURAL SEXUAL OFFENCES

Definition

IPC Section 377 defines unnatural sexual offences as voluntary sexual intercourse against the order of nature with any woman, man or animal.[20]

Punishment given Imprisonment for 10 years and fine.

Classification

All unnatural sexual offences are classified as sodomy, buccal coitus, lesbianism and bestiality. Types of unnatural sexual offences are depicted in Table 26.5.

SODOMY (Anal Intercourse, Greek Love, Buggary)

Definition Sodomy is defined as anal intercourse performed by a male with another male/female who may be a child/adult, with or without consent and by force.

Historical aspect In the old *Biblical* town called *Sodom* in ancient Rome, this type of sex was more common in practice even among married couples, to avoid unwanted pregnancy and thus the term sodomy emerged. It was introduced and became a common practice in India during the Moghul period.[30]

Terminologies: Following four terminologies are important in connection with sodomy, [1-6]

- Active agent—is a sodomist male who performs the anal intercourse actively.
- Passive agent—is a sodomist male or female who offers the anus, and plays a passive role.
- Pederasty—is an adult performing sodomy act involving a male or a female child as his passive agent.
- Catamite—is a child who plays a passive role in sodomy.
- Gerontophilia—an old male or female who acts as passive agent.

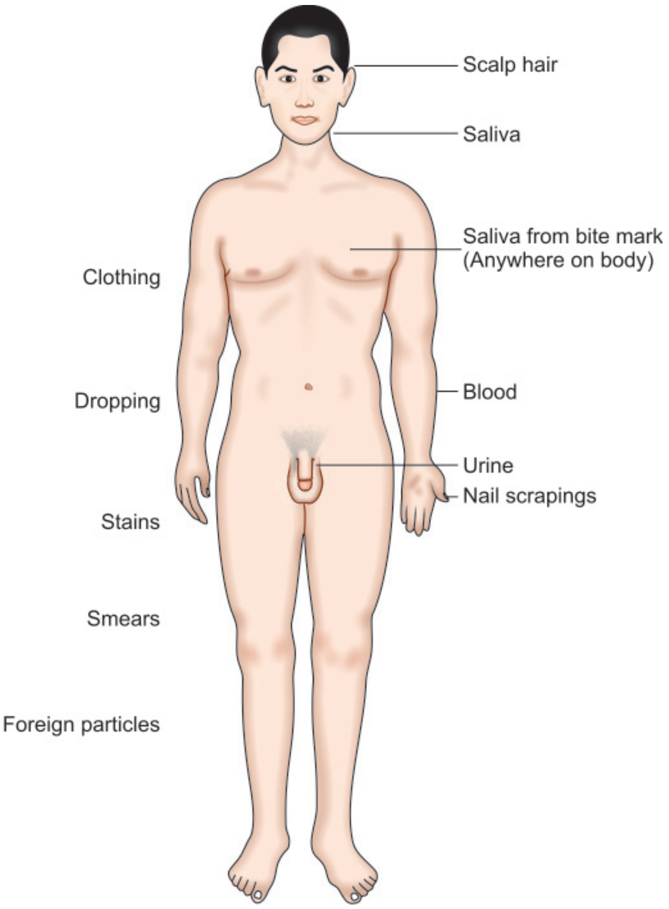


Fig. 26.26: Examination of the accused (male) of rape—specimens (trace evidence)

Incidence: It is common in men who are homosexuals (true invests), sailors, prisoners, military barracks, men's hostels, etc. These men often act alternatively as active and passive agents mutually.

Examination of a Case of Sodomy

Examination of a case of sodomy includes three steps (remember 3 Gs):

- General procedures/preliminaries
- General examination
- Local examination.

General procedure comprises of the following.

- *Informed consent for examination*—must be obtained prior to examination.
- *Details on the event*—elicit the history of the case from the victim and record in the same version as told.
- *Penetration*—ask if the victim felt the penetration into the anus.
- *Use of lubricant*—ask the victim whether any lubricant was used.

General examination comprises of the following:

- Examination of clothing (refer examination of rape victim).
- Physical examination (refer examination of rape victim).

Local examination comprises of the following.

- Examination of passive agent
- Examination of active agent.

Examination of Passive Agent

It can be dealt under two heads.

- Precautions
- Clinical findings.

Precautions

- Ask the patient to undress
- Keep a male/female nurse during examination
- Put the patient in "knee-elbow" position (Fig. 26.27A)
- Use an anal speculum for examination.

Clinical findings: These may vary depending on type of passive agent, who may be a habitual passive agent or forcibly victimised (non-habitual) passive agent.

1. Findings in habitual passive agent (Fig. 26.27B) will be as follows.
   - — Shaving of the anal and perianal hairs
   - — Loss of normal puckering around anus
   - — A funnel-shaped depression between buttocks around anus
   - — Skin around anus thickened and smooth-due to frequent friction
   - — On per rectal (PR) examination
     - Loss of muscle tone (no radial constriction of anus on pinching the skin around anus) presenting a patulous anus
     - Presence of scars of old tears or fissures
     - Presence of lubricant/semen/ venereal discharges.
2. Findings in forcibly victimised (non-habitual) passive agent (Figs 26.27C and 26.28). These will be as follows:
   - Anus may appear to be swollen with temporary loss of tonicity of the anal sphincter
   - Contusion/laceration of the posterior and mucocutaneous tissue
   - Seminal or semen mixed with faecal matter or bloodstains may be seen.
   - If the victim is child, penetration is usually forceful, resulting in tears, and at times prolapse of portion of anal canal, seen.
   - On per-rectal (P/R) examination—if one finger enters no intercourse may be opined, but if two fingers can be inserted eliciting pain, it is suggestive of anal intercourse.

Examination of Active Agent

Look for following findings:

- Peculiar smell of anal gland secretions
- Traces of faecal matter and lubricant used are often detected on the coronal sulcus, frenulum, prepuce, etc.
- Abrasions, bruises, lacerations of prepuce, frenulum, glans penis, etc.
- Presence of STD lesion/discharge.

Note: The shape of the glans penis may be found to be tapering, elongated and constricted, in a habitual sodomist (active agent). All above findings are considered corroborative evidence of sodomy.

Medicolegal Importance

- In India, sodomy is punishable
- In certain western countries it is not so, which has even led to male marriage (gay wedding)
- AIDS is suspected to have commenced as a consequence of practice of sodomy.

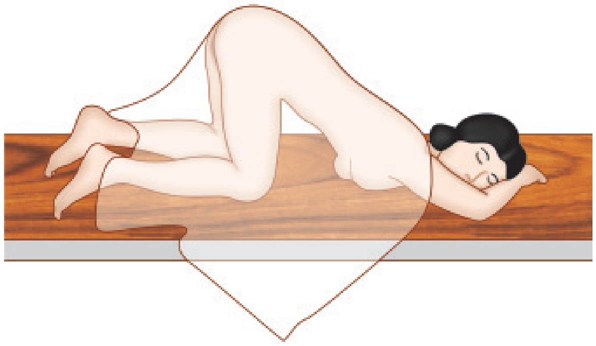


Fig. 26.27A: Knee-elbow (Dorsal lithotomy) position

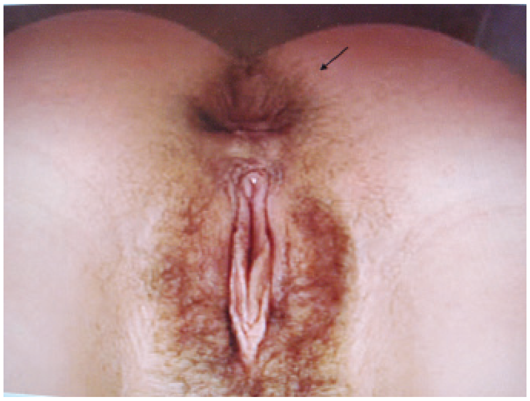


Fig. 26.27B: Findings of sodomy in a habitual passive agent (knee-elbow position). *Note:* loss of anal puckering, shaving of the area and, marked funnel-shaped depression and smoothening of skin around the anus (Arrow)

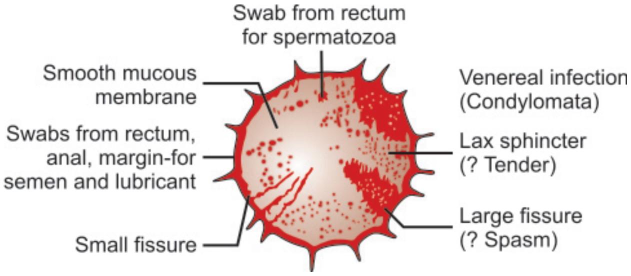

Swab from rectum for spermatozoa
Smooth mucous membrane
Venereal infection (Condylomata)
Swabs from rectum, anal, margin-for semen and lubricant
Lax sphincter (? Tender)
Large fissure (? Spasm)
Small fissure

Fig. 26.27C: Details of evidence of sodomy on examination

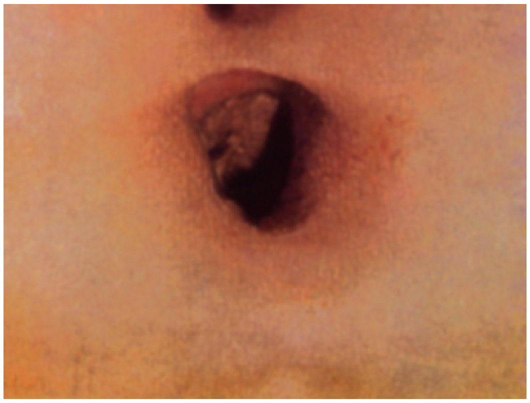


Fig. 26.28: Anal dilatation with anal and perianal injuries is strongly suggestive of sodomy in this child victim

Fig. 26.29: Sex toys: Dildos, vibrators, vaginal tampons, and artificial vagina and artificial phallus (*Source:* http://commons.wikimedia.org/wiki/file:Pleasuretoys-glass-dildo.png; *Source:* http://en.wikipedia.org/wiki/artificial_vaginal#materials)

LESBIANISM (Female Homosexuality, Lesbian Love, Tribadism)

Historical aspect: In ancient Greece, it was commonly practised in Isle of Lesbos, hence the term lesbianism.

Definition: Lesbianism is defined as a mode of deriving sexual gratification by two consenting females, by mutual genital manipulations manually with fingers, lips or extragenital mechanical devices—sex toys (Fig. 26.29).

Incidence: It is common in women who are homosexuals (true inverts) in ladies hostels or prisons, among nymphomaniacs, over-attached girls, etc.

Examination of a Case

All steps of examination are same as for examining the case of rape. Findings in favour are:

- Bite marks (love bites), nail scratch marks, abrasions, etc. on mutual genitalia, perineum, breasts, etc.
- Injury to vaginal canal (especially if dildos are used)
- Women may be more masculine.

Medicolegal Importance

- The act is punishable when practiced in public places.
- Suicidal or homicidal tendencies common among lesbianistic girls due to "morbid jealousy" whenever one of the partners gets married.
- Failure of marriage and domestic life certainly occurs in those who are married.

BUCCAL COITUS (Sin of Gomorrah, Coitus Per Oris, Fellatio, Cunnilingus)

Definition: Sexual gratification of a male by performing the act of intercourse into the oral cavity of a sex partner with consent or by force (usually a female partner).

Incidence: Usually practised with an innocent child victim. It is often practised as a part of "sexual foreplay" also.

Examination of a Case

All steps of examination are as for a rape case.

Diagnostic Findings

- Faint teeth marks or abrasion on the penis,
- Seminal stains on victim's face, mouth, etc.
- Struggle evidence on both when done forcibly.

Medicolegal Importance

- Punishable if done by force
- Punishable if done in public with a consenting partner
- Death of a victim especially a child, due to accidental respiratory tract obstruction by the ejaculating bouts of semen leading to mechanical asphyxia.

BESTIALITY

Definition: Bestiality is defined as sexual gratification by having sexual intercourse with animals.

Incidence: Common among "shephard boys" and nymphomaniac females.

Causes: Certain causes are:

- Sexual excitement when left alone with animals (pet ones)
- False belief among Indian villagers that it is a remedy for curing gonorrhea.

Common animal victims: Pet domestic animals like sheep, cow, calf, ass (female), bitch, large birds such as ducks, goose, chicken, etc. by a male offender. Male dogs, monkeys, cats, etc. by a female offender.

Examination of a Case

Examination of both accused and animal is important.

Signs in Accused

- Penis/vulva will be found to be contaminated with animal discharges or secretions.
- Marks of injury due to animal biting, scratching, kicks, etc.
- Presence of animal hair on the garments worn.

Signs in Animals

- Presence of human semen or sperms in animal passages, injuries on animal genitalia and other natural orifices.
- Venereal discharges in animal passages

Medicolegal Importance

- Punishable if guilty by medicolegal examination of accused.

SEXUAL DEVIATIONS (Sexual Perversions)

Definition

Sexual deviations are certain physical acts or behavioral abnormalities performed without hesitation, to achieve sexual gratification without the actual sexual union.

Types of Sexual Perversions

Table 26.5 enumerates the types of sexual perversions encountered routinely.

Incidence: It is found to be more common among men. However, it is also true that normally every person has a seed of sexual deviant behavior within the self. As most of these deviations are harmless to the victim, it is rarely bothered to be brought into the notice of law and police. There are certain dangerous perversions, which can result in death of both the performer and his victim, creating problems, e.g. sadomasochism.

Psychological Aspect

A brief account on *Freud's psychosexual stages of development*[31] can make one understand psychological aspect of how a person turns out to be a sexual pervert. As per this every human being with his physical maturity, undergoes a psychological maturity also concerned with sex.

It is proposed that, this *psychosexual development* (PSD) maturity occurs from infancy to puberty in the form of five phases, which are narrated as a "*sexual maturity ladder" and comprise of*:

- Oral phase
- Urethral and anal phase
- Phallic phase
- Latency phase
- Genital phase

Figure 26.30 illustrates the concept and each phase is discussed individually.

Oral phase: This phase commences from birth of the child and spreads over to first year. In this phase, the infant develops libidinal impulses through oral cavity, i.e. mouth, tongue, lips, etc. through acts such as sucking, licking, kissing, biting, crying, etc. These are the only modes of expression at this age.

Urethral and Anal phase: This extends between 1 and 3 years of age. Here, the libidinal impulse is through anal and urethral passages. The child learns the joy of defaecating and micturating along with the other psychosexual maturity already attained.

Phallic phase: In this phase (3-5 years), the child develops the actual curiosity in knowing about the opposite sex. Here, the child tries to realize the differences in each sex, e.g. a boy trying to expose his genital organs (exhibitionism) to a girl and then ask her to show her private parts to him, trying to find out how a girl passes urine by squatting by hiding himself (voyeurism) ultimately trying to achieve pleasure by knowing the facts.

Latency phase: In this phase (5-12 years), child apparently stops all sexual preoccupation and develops a super ego.

Genital phase: This extends from 12 to 14 years. In this phase, the person reaches the ultimate PSD resulting in all matured heterosexual affections and sexual gratifications, pleasure of manual manipulation of genitals (masturbation), etc. which continues to be maintained throughout life.

Onset of Sexual Perversion

During the PSD there is a possibility that the maturity can get arrested at any one of these five phases, while physical growth continues further. Such a person will hence restrict all his or her sexual needs at the level at which the phase of PSD has arrested and he/she will try to get the sexual gratification by all the methods his PSD allows, resulting into sexually abnormal person.

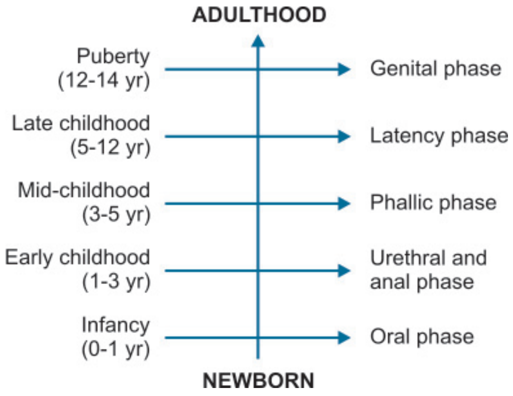


Fig. 26.30: The sexual maturity ladder

Thus, for example, a male whose PSD is arrested at oral phase may turn out to be a person enjoying sex by practicing oral sex, while one whose PSD arrests at phallic phase will try to achieve sexual gratification by adopting-masturbatory or voyeuristic or exhibitionistic acts, etc.

Eonism/Transvestism

Eonism is also known as transvestism, is a sexual perversion wherein sexual gratification is achieved by wearing the dress of the opposite sex. This is more common in males and he would dress like a female, perform masturbation to ejaculate and thus get sexual gratification.

However, the trends of modern fashion of wearing pants and shirts like a male by a female cannot be considered as transvestism.

Exhibitionism

The exhibitionist gets the pleasure by exposing private parts in public or in front of a opposite sex individual, sometimes performing masturbation. This perversion is common among men. Usually he may be suffering from some other mental disorders.

When exposure is aimed to attract a particular woman then, often there is an expectation of similar exposure by the woman or it may be a reflection of a hidden desire to observe the private parts of the woman. This act is punishable under sections 290 and 291 of IPC.

Fetishism

Fetishism is a type of sexual perversion common among males. Here the individual gets sexual excitement and gratification by merely seeing or feeling of female body parts, certain articles belonging to a woman, such as a sari, footwear, stockings, undergarments, hankies, hairpins, etc.

At times a fetish may steal these articles and masturbate and ejaculate on them to get sexually gratified.

Masochism

Masochism is a rarer sexual perversion, wherein sexual gratification is obtained on being tortured by the sex partner. Virtually this is just the opposite of sadism (refer below).

Masochistic asphyxial death (sexual asphyxia) is a dangerous perversion to practise, occurs, when the pervert creates a state of hypoxia (low oxygen level in blood) in him to get orgasm, through partial hanging through a special device, which is self-designed and self-regulated. After experiencing orgasm the constriction force around the neck is usually released.

As this is often practised in privacy, all alone, and that release of constriction around the neck is not possible if there is a fault in the device under his control, death is inevitable. A masochist is considered to be mentally aberrated person and possibly his childhood experience of cruelty and adversity gets reflected in the masochistic self-torturing.

Sadism

Sadism is a sexual perversion wherein sexual gratification is achieved by inducing pain on the sex partner.

Historical aspect: It has originated from the French author Marquis D'Sade' who is known for his novel, which became popular for his methods of inducing pain and torturing for deriving pleasure and sexual gratification.

Incidence: It may be found in both sexes, but is more common among male.

Methods Adopted: Biting, pinching, injuring or ill-treating the partner in a very cruel way. In extreme cases even a murder may be committed to derive sexual gratification. This is called *"lust murder"*. Rarely one may get the sexual gratification by tearing out the genitals or other parts of the body and virtually eating them raw. This is called *"necrophagia"*.

Lust Murder

This is the consequence of extreme sadist practice by a male. In this type of perversion, sexual arousal begins with torturing of the sex partner and with death of the partner full gratification is obtained. Often the body of the victim is mutilated. In some cases after full arousal in this way, the pervert performs sexual intercourse with the dead body of the victim till ejaculation and gratification occurs.

Sadomasochism

Commonly know as *bondage*. It is a combined form of sadism and masochism in the same individual. Often it is observed that a sadistic husband will have a masochistic wife, or vice versa.

Medicolegal Importance

- This is a dangerous perversion, as the victim may turn out to succumb into lust murder.
- The sadist must be hospitalised for his mental problem.
- However, in India sadism is a punishable sexual offense whenever reported.

Necrophagia

This is another rare from of sadistic perversion seen among male, who gets sexual gratification by tearing out the genitals or other parts like breasts, buttocks, etc. of his partner after her death by his teeth and may virtually eat them raw.

Necrophilia

Necrophilia is a desire of obtaining sexual gratification by performing sexual intercourse with a cadaver of opposite sex. Males often suffer from such desire and such a male is usually psychosexually incompetent. He feels that this is a most suitable way, as there is no rejection and no one else will come to know about his weaknesses. Often he visits a prostitute for fear of social stigma and fear of exposure of his sexual incompetence. This condition is a result of mental aberration accompanied by personality defect. However, no such pervert will commit murder of a female to have the sexual intercourse and achieve gratification.

Voyeurism

Popularly known as *Peeping Tom* or *Scotophilia*. It consists of achieving sexual gratification by secretly watching others getting undressed, taking bath, or performing sexual intercourse, etc. Orgasm is usually preceded by masturbation, e.g. watching blue film, troilism, etc.

Blue Film

Blue film is a movie taken while a couple is performing sexual union and exhibited to the audience in a theatre or video parlour. Here both the couple performing as well as the audience watching this in the theatre may be considered as sexual perverts, former ones exhibitionists and the latter voyeuristic.

The art of taking photographs of nude men and women and also clicking while couples perform sexual intercourse in different perspective and then publishing them in the form of books constitutes *pornography*.

This is banned in India for reasons not only because it is immoral, but also because it might encourage unhealthy activities such as promiscuous sex, prostitution, sexually transmitted diseases, etc. in the nation.

Troilism

Troilism is an extreme degree of voyeurism wherein a perverted husband gets sexual gratification by watching his own wife performing, sexual intercourse with another man.

Masturbation

This type of perversion comprises of an act of deliberate, manual handling or self manipulation of genital organ by fingers or mechanical devices till ejaculation in a male and orgasm in a female, i.e. achieving sexual gratification occurs.

Incidence: Masturbation is a common mode of sexual perversion observed both among men and women.

Medicolegal Importance: According to newer concepts, this is not considered a perversion, but a harmless sexual activity towards attaining sexual maturity. However, masturbating in public is immoral and punishable. Death may result in cases wherein an electrical device is used for the purpose and is not handled carefully or is in a faulty condition.

Nymphomaniac

Nymphomaniac is a type of perversion of excessive sexual desire in a woman, wherein the woman is in need of sex frequently, irrespective of whether it is by normal or abnormal sexual acts, such as masturbation, sexual intercourse, oral sex, sodomy, lesbianism, bestiality, etc.

Satyriasis

Satyriasis is excessive sexual desire, arousal and drive in men. These subjects are liable to commit sexual offenses like rape or practise other abnormal sexual acts such as sodomy, fellatio, bestiality, masturbation, etc. to get gratification and are dangerous to the society.

Frotteurism

Frotteurism is a mischievous act usually practiced by a male sex-pervert in a crowded place to derive sexual gratification by pressing/rubbing his genital part through the dresses worn, against body parts of a female in front of him. As per sections 290 and 291 IPC, this is a punishable offense in India.

Undinism

Undinism is a rare type of sexual perversion, common among men wherein the pervert achieves sexual gratification by watching the act of passing urine or defaecating by woman. The perversion may take bad shape when the pervert achieves the sexual gratification not only by watching the act of passing urine by a woman but combined with exhibitionism, masturbation, voyeurism, etc.

Indecent Assault

Any offense committed on a female with an intent or knowledge to outrage her modesty. This is usually sexually motivated and obviously done without her consent.

This includes:
- Kissing of any part of female body
- Touching, pressing the breasts or private parts, including thighs
- Exposing a female's genital parts, breasts, etc.
- A medical practitioner can be accused of indecent assault if he examines a female patient by stripping her clothes without her consent.

LAWS IN RELATION TO SEXUAL OFFENCES IN INDIA[20,21,29]

1. Section 375 IPC defines rape.
2. Section 376 IPC describes punishment for rape.
3. Section 377 IPC defines unnatural sexual offenses as well as describes punishment.
4. Section 53 CrPC states that when a person accused of sexual offence referred to a doctor for examination by Police Officer not below the rank of Sub-Inspector, he need not take consent from the accused for examination. If he refuses to get examined, mild degree of force can be used to examine him.
5. Section 54 CrPC provides an opportunity to an accused of a sexual offence to get examined physically to disprove the charges.

ABORTION (MISCARRIAGE, FOETICIDE)[32-35]

Definition

Abortion is defined as the premature expulsion of the products of conception prior to completion of the total period of gestation.

Products of Conception

It is designated by several terms and they are as follows:
- Fertilised ovum—from fertilisation till implantation, which usually occurs within 1st to 2nd weeks of conception (Fig. 26.31).
- Embryo—denotes the developing product of conception after its implantation in the uterus up to the end of second month, when usually placenta develops (see Fig. 26.31).
- Foetus—the product of conception from the 3rd month till birth.

Classification

Depending on the way of induction, abortion is classified into two groups:
1. Natural abortion
2. Artificial or induced abortion which could be again of two types:
    - Justifiable or therapeutic abortion (Medical termination of pregnancy–MTP)
    - Criminal or non-justifiable abortion.

Differences between natural and criminal abortions are depicted in Table 26.6.

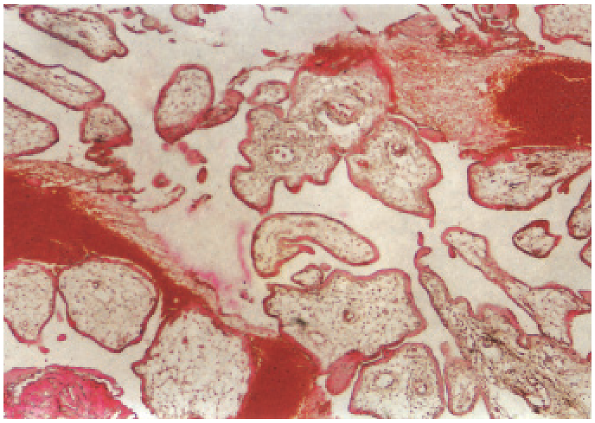


Fig. 26.31: Products of conception (clots expelled) suggestive of placental structures (chorionic villii) confirmed histopathologically

Natural Abortion (Spontaneous Abortion)

Natural abortion is an abortion that occurs spontaneously without any induction procedures and usually coincides with a menstrual flow.

Incidence

It is more common in the earlier period of gestation (2nd-4th months).

Causes

Causes of natural abortion could be paternal or maternal or fetal in origin.
1. *Paternal*—usually due to syphilis, tuberculosis, old age or general debility.
2. Maternal
    - Uterine causes—cervical incompetence and other pathological conditions of uterus.
    - Constitutional causes—syphilis, acute fever, diarrhea, diabetes, jaundice, anemia, etc.
    - Nervous/mental causes—shock, sudden fright, sudden joy or sorrow, etc.
    - Violence/trauma—fall from height, blows or kicks on the abdomen, etc.
    - Acute/chronic poisoning with lead, copper, mercury, etc.
3. Foetal

- Inflammation and fatty degeneration of the placenta
- Faulty development
- Syphilis

Differential Diagnosis of Blood per Vagina

- In a pre-pubertal girl—it could be injury, rape, foreign body, etc.

Table 26.6: Differences between natural and criminal abortions

Particulars	*Natural abortion*	*Criminal abortion*
Predisposing factor	Diseases of uterus, placenta may be present	History of pregnancy due to illicit sex may be present
Infection	Rare	Frequent
Injuries in and around genitals	Absent	Often present
Mucosa of genitalia and uterus	Changes of pregnancy may be present	Ulceration, edema, congestion, etc. in vagina/cervix due to local application of irritant substance may be present
Foreign bodies	Never found	May be found

Table 26.7: Differences between frank blood and menstrual blood

Criteria	*Frank blood*	*Menstrual blood*
Source	Arteries/veins	Denuded endometrium
Clotting	+ ve	– ve
Odour	– ve	+ ve (Foul smell)
Microscopy	RBC, WBC, platelets, etc.	Endometrial glands and vaginal epithelial cells

- In women in the child bearing age (14-45 years) – it could be injury, menstruation (Table 26.7), rape, criminal abortion, lochia, fibroid uterus, dysfunctional uterine bleeding (DUB), etc.
- In postmenopausal age– DUB or malignant tumours.

Medical Termination of Pregnancy/MTP (Justifiable Abortion, Therapeutic Abortion)

Medical termination of pregnancy is an abortion induced by a medical man in good faith to save the life of the mother for proper therapeutic need.

The following conditions must be fulfilled to perform a justifiable abortion.

1. Written consent of both the woman and her husband or legal guardian.
2. Consultation with another medical man, usually a specialist or one who is superior in qualification and experience.
3. Proper indication of performing the abortion:
    - Systemic diseases – cardiac failure, severe hypertension, severe toxemia of pregnancy, advanced renal diseases, epilepsy, etc.
    - Gynecological indications
        - — Carcinoma of the genital organs
        - — Threatened abortion with severe bleeding
        - — Uterine sepsis due to attempted criminal abortion
    - Conditions causing fetal abnormality- infective conditions like rubella, significant exposure of mother to teratogenic agents, etc.

Abortion Law in India

MTP Act, 1971 and MTP rules, 1975 have liberalised abortion in India.[32] As per this Act, a qualified registered medical practitioner is allowed to undertake termination of pregnancy under following circumstances:

Therapeutic indications: Pregnancy would be a risk to the life of the mother or may cause physical or mental illness of the mother.

Eugenic indications: There is significant possibility of the child being born with physical and mental abnormalities.

Social indications: Failure of family planning methods in married women.

Humanitarian indications: Pregnancy caused by rape, pregnancy in lunatics, etc.

MTP Act Rules 1975 to Be fulfilled

1. Written informed consent of the woman before performing abortion is mandatory. The woman to be aborted should be above 18 years of age. However, consent can be obtained from the parents or guardian when she is a minor or mentally unsound.
2. Up to 12 weeks pregnancy, any qualified practitioner can perform an abortion. However, he must have undergone compulsory training.
3. Beyond 12 weeks and up to 20 weeks pregnancy, opinion of second practitioner must be obtained to justify performing the abortion.
4. MTP must be done only in well-equipped hospitals authorised by the government.

Methods of Inducing MTP

This depends on duration of pregnancy in each case.

1. In first three months of pregnancy following are opted:
    - Dilatation and curettage (D and C)
    - Vacuum suction and curettage

    Under both these methods, the cervical canal is first dilated by no.4-Hegar's dilator under local or general anaesthesia and then products of conception removed by either scooping out by a uterine curettage or sucked out by connecting to vacuum suction evacuation equipment.
2. In pregnancy beyond three months following are opted:
    - Induction by prostaglandin E_1 and E_2, which bring about uterine contractions and expel the fetus.
    - *Amniocentesis* – is a minor surgical procedure wherein using an amniocentesis needle, amniotic fluid is withdrawn and replaced with equal volume of 20 per cent saline/50 per cent glucose per abdominally. This results in expulsion of uterine contents with in 48 hours after injection.
    - *Abdominal hysterectomy*— is a surgical procedure of removing the fetus, preferred after 4 months of gestation.

Criminal Abortion

Criminal abortion is an unlawful expulsion of the products of conception at any stage of gestation by an unqualified person or a qualified doctor.

Motive for Criminal Abortion

1. To get rid of a child following illicit intercourse with a view to avoid the shame and disgrace in case of:
    - Widows
    - Unmarried girls
    - Married women conceiving during long absence of their husbands or living separately from their husbands.
2. To prevent inconvenient addition to the family.
3. The heir presumptive may procure abortion of the female, whose husband's property he is to inherit in the absence of a child of her own.

Methods Adopted in Inducing Criminal Abortion

Usually mechanical violence is used by several means (Fig. 26.32) such as:

Extrauterine Means

- Violent exercise, such as horse riding, cycling, jolting and lifting of heavy weights.
- Severe shock as in kicks and blows over the abdomen and falling or jumping from a height.
- Blood letting, by application of leeches to the vulva, perineum or inner aspect of thighs

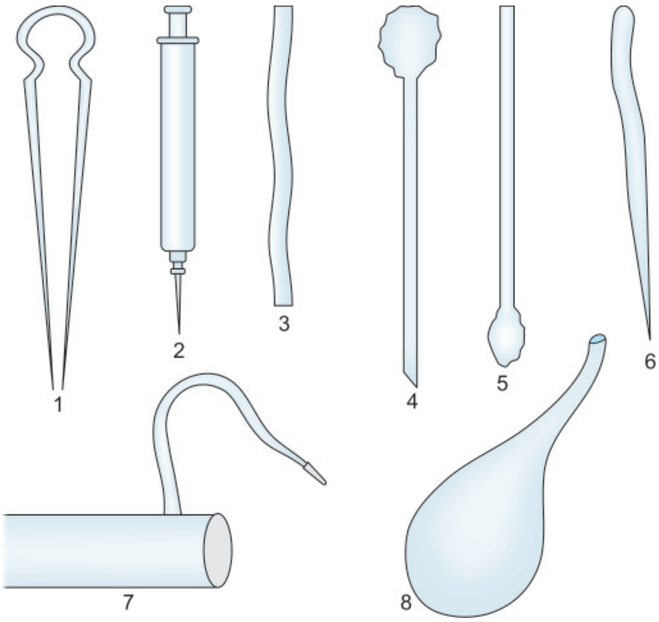


Fig. 26.32: Materials used for causing criminal abortion: (1) Hair pin, (2) Syringe, (3,4,5) Abortion stick, (6) Root of abortifacient plant, (7) Douche, (8) Part of Hagginson's syringe

- Kneading or firmly massaging the anterior abdominal wall
- Tight compression of the lower abdomen.
- Drugs such as ecbolics, emmenogogues.

Uterine Means

- Vaginal douching-alternative hot and cold water douching of vagina is proved to result in abortion
- Injection of soap water into the vagina can induce abortion
- *Abortion stick*—is a wooden stick measuring 10 to 15 cm in length and 0.5 to 1 cm in diameter. Usually a branch of vegetable irritant plant such as: calotropis, marking nut, plumbago, etc. are used for the purpose. One end of this stick, which is wrapped, with a piece of cloth or cotton wool, is soaked in or smeared with an irritant or any abortifacient substance. This is passed into the uterus, per vaginally, resulting in expulsion of product of conception.

  Danger of the stick: It could result in any of the following:
  - Shock
  - Haemorrhage
  - Sepsis
  - Perforation
  - Peritonitis
  - Absorption of poison from vaginal or cervical mucous membranes leading to generalised poisoning
  - All these complications may terminate fatally, resulting in death of the patient.
  - Electricity—110 volts of electric current via negative pole applied to posterior vaginal cul-de-sac and positive pole to lumbosacral region can lead to abortion.
  - Use of abortifacient drugs such as: Drastic purgatives like croton oil, magnesium sulphate, aloes, etc. which are drastic purgatives acting on rectum. Ecbolics like ergot, quinine, posterior pituitary extract, etc. which act by increasing uterine contractions result in abortion.

Cause of Death in Criminal Abortion

Causes are several (Fig. 26.33) and are classified as immediate, delayed and remote causes.

1. Immediate causes:
   - Reflex vagal inhibition following instrumental evacuation or from sudden dilatation of vagina and cervical canal by the passage of instrument.
   - Air embolism from faulty syringing.
   - Soap embolism from improper introduction and absorption of soap solution.
   - Shock and haemorrhage from injury to vaginal, uterine or pelvic vessels.

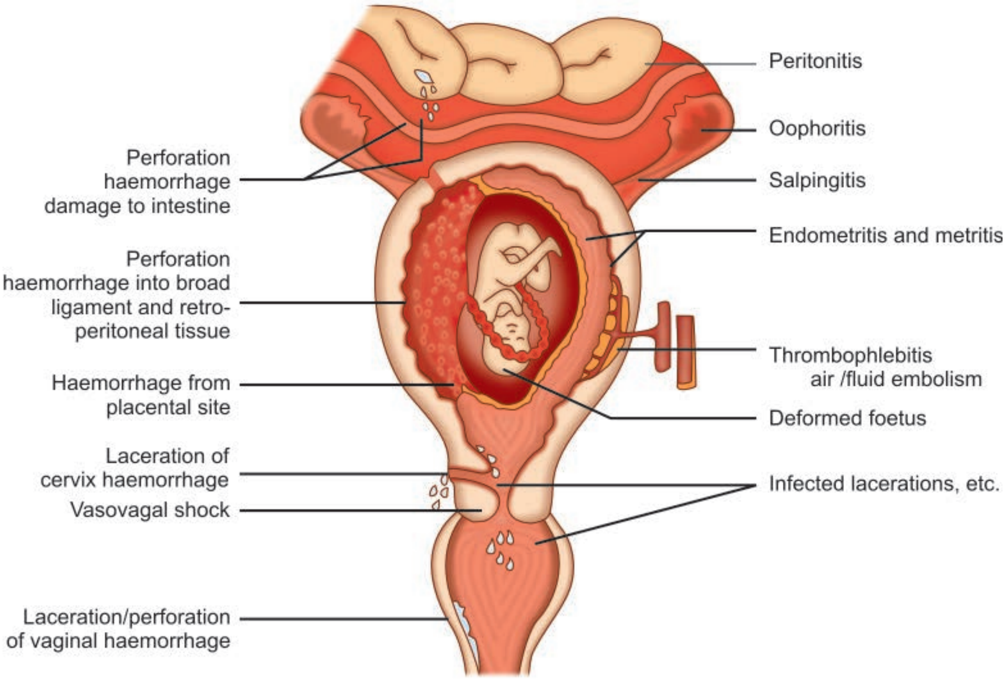

Peritonitis
Oophoritis
Salpingitis
Endometritis and metritis
Thrombophlebitis air /fluid embolism
Deformed foetus
Infected lacerations, etc.
Perforation haemorrhage damage to intestine
Perforation haemorrhage into broad ligament and retro-peritoneal tissue
Haemorrhage from placental site
Laceration of cervix haemorrhage
Vasovagal shock
Laceration/perforation of vaginal haemorrhage

Fig. 26.33: Diagram illustrating complications of criminal abortion

2. Delayed causes (2-3 days):
   - Septicemia following infection.
   - Pyemia from pelvic abscess.
   - Generalised peritonitis following perforation of uterus or of the bowel by the instrument.
   - Tetanus.
3. Remote causes (beyond 3 days):
   - Renal failure.
   - Pulmonary embolism from dislodged thrombus.
   - Systemic poisoning by absorption of abortifacient drugs.
   - Secondary infection—pneumonia, empyema, meningitis, etc.

Signs of Criminal Abortion

1. Profile of victim – usually unmarried girls or widows
2. On examination the patient may show following:
   - Laceration of the vagina and cervix
   - Marks of violence on the abdomen of healthy women
   - Evidence of sepsis – like pelvic cellulitis, peritonitis, etc.
   - Injuries on the foetus
   - Evidence of burns over the back may be found in a criminal abortion induced by electrocution
   - Presence of the parts of the abortion stick or foreign body in the uterus or vagina
   - Reddening and blistering of vaginal mucosa when irritants are used
   - Perforation of the uterus, when abortion stick is used.

Examination of a Case of Criminal Abortion

Includes following steps:

- Procedural formalities
- Examination of the alleged woman
- Examination of the material alleged to have expelled out.

Procedural Formalities:

These include the following:

1. *Requirement before examination:*
   - Requisition from the magistrate
   - Identification of the female
   - Written consent of the female
   - Result of the examination should be appraised before hand.
2. *Precautions:* It is better to keep a female witness (nurse) or the husband of the woman undergoing MTP.
3. *Other particulars:* Note the following:
   - Short history of the case
   - Date and time of abortion
   - Physical and mental conditions of the patient
   - Examination of the clothing
   - Age of the victim is to be determined.
4. *Enquire into facts* about the woman aborted, means adopted to procure criminal abortion, and the material expelled out of the uterus.

Examination of the Alleged Woman (Victim)

In the living: This should be elicited as early as possible, otherwise many of the important signs will disappear within seven days. The signs of recent abortion, i.e. findings within first 4 to 5 days and other findings are same as those of recent delivery. These are already discussed above under the heading 'delivery'.

In the dead: In addition to above signs we may find following internal postmortem findings:

- Foreign body inside the uterus (Fig. 26.34)
- Extravasated blood under the bruised portions
- Mucous membranes of stomach and intestines are congested if an irritant poison was swallowed to procure abortion
- Uterus is enlarged and its cavity contains portions of decidua. Some of the products of conception may be found. Placental site with the gaping placental sinuses may be seen. Ovaries are congested and on section, show the presence of corpus luteum. Uterus may show injuries of different degrees (Figs 26.35 to 26.37).

Examination of Material Alleged to have been Expelled out

- This may be blood clot, shreds of membrane, a hydatidiform mole, fibroid tumor, polyp, an embryo or an immature foetus.
- Traces of ovum are searched for in the material after washing it in a basin of water. Abortion cannot be definitely established, unless the products of conception are found.
- If it is a foetus, its intrauterine age is determined. The viability of the child is also noted (210 days – viable age).
- Note the length, weight, and stage of development of the baby and whether it is stillborn or live born. Blood group test may determine its maternity. Whether abortion is done or not, even its attempt on a woman is illegal. The abortion attempted on a woman, who is later proved to be not pregnant is illegal too [abortion- if attempted or carried out on therapeutic or medical grounds in the interest of the mother, is however not considered to be illegal].
- After the death of a pregnant woman, if body is highly decomposed, the contents will come out spontaneously due

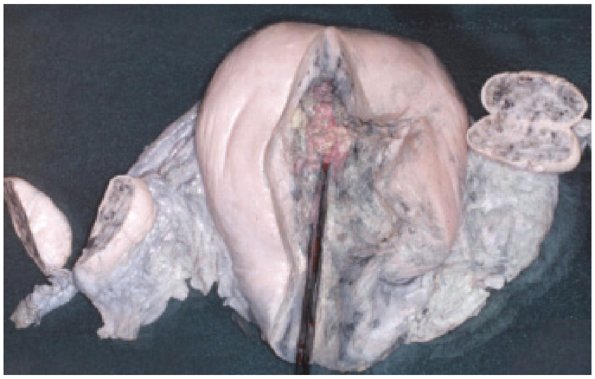


Fig. 26.34: Foreign body (twig of an abortion stick) inside the uterus, noticed at autopsy in a victim of criminal abortion

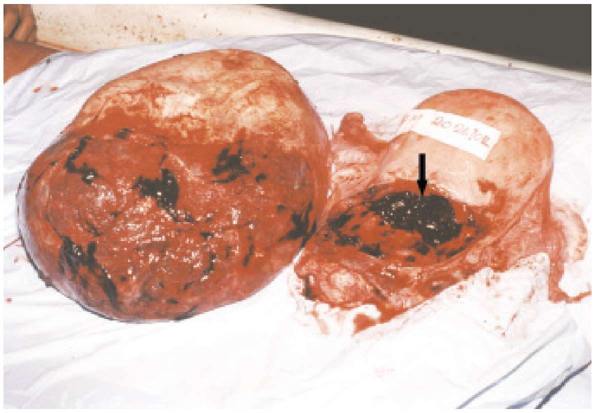


Fig. 26.35: Rupture of uterus (right side) and extruded amniotic sac with foetus (left side) as noticed during autopsy, victim of criminal abortion (*Courtesy:* Dr Zachariah Thomas, Dept. of Forensic Medicine, Medical College, Trivandrum, Kerala)

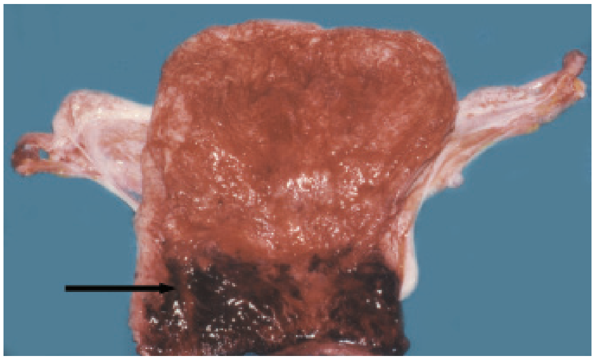


Fig. 26.36: Autopsy finding of uterus—atonic flabby with retained clots in postpartum haemorrhage case (*Courtesy:* Dr Zachariah Thomas, Dept. of Forensic Medicine, Medical College, Trivandrum, Kerala)

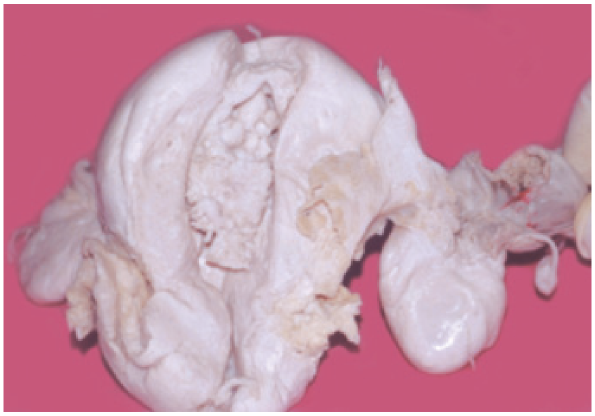


Fig. 26.37: Ruptured ectopic pregnancy with decidual cast inside the uterine cavity (*Courtesy:* Dr Zachariah Thomas, Dept. of Forensic Medicine, Medical College, Trivandrum, Kerala)

to the pressure of gases of putrefaction (postmortem delivery). Even the uterus may prolapse.

Medicolegal Aspects

In a case of an attempt to cause abortion or miscarriage, it is not necessary to prove that the woman was pregnant. But in awarding punishment, the following points are usually considered by the court:

- Whether the woman was pregnant or not (proof of pregnancy) is required for actual abortion, but not for attempt to do it.
- Whether the woman was quick with the child. The offence is grave when the woman was quick with child.
- Whether the abortion or its attempt has been done with or without the consent of the woman. In either case, punishment is by life imprisonment.
- Whether death of the child was caused by the act.
- Whether death of the woman was due to the abortion or its attempt.

Medicolegal Importance of Placenta

1. It gives the duration of pregnancy.
2. Abnormality or disease condition of placenta can result in spontaneous abortion.
3. Injured placenta may point to criminal abortion.
4. Pieces of placental tissue retained in the uterus will confirm the diagnosis of abortion.
5. Poisons or chemicals used for the abortion can be detected from the placental tissue.

Relevant Sections in Laws on Abortion in India[20,21]

- *Section 312, Indian Penal Code (IPC)* states that abortion induced not for the purpose of saving life/good of the woman, be punished with imprisonment for 3 years. If the woman is "*quick with child*" the period is extended for 7 years more.
- *Section 313 IPC,* states that if abortion is done without the consent of the woman, punishment is life imprisonment
- *Section 314 IPC,* states that if the woman dies after an abortion, which is done with her consent, punishment is life imprisonment.
- *Section 315 IPC*, states that preventing a child born alive or causing it to die after birth, is also punishable.
- *Section 317 IPC*, exposure or abandoning infant are both punishable.
- *Section 318 IPC* – concealment of birth and secret disposal of foetus are also punishable.

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Chapter

27

Infanticide, Foeticide and Child Abuse

INTRODUCTION

The tender age in life such as intrauterine foetal life, infancy and childhood are often exposed to various unfavourable circumstances comprising of diseases and trauma. Broadly they are categorised in to three types: (i) foetal death means death of a foetus at any time prior to birth, (ii) infant/newborn/neonatal death meaning death of a child under one year age due to natural and unnatural causes, and (iii) child abuse: means maltreatment of a child for sexual and nonsexual purposes.

The word infanticide is derived from Latin word *infanticidium*, meaning killing of a child/infant (meaning newborn, baby, toddler, tot, etc).[1] Infanticide has been practiced in every continent of the world by people of all cultural complexity, from hunters and gatherers to high civilisation, including our own ancestors. Rather than being an exception, then, it has been the rule. There is ample historical evidence to document the incredible propensity of parents to murder their own children under an assortment of stressful situations. In nineteenth century England, for example, infanticide was rampant throughout the country.[2]

Two reasons for infanticide stand prominent in history. They are poverty and population control. Darwin believed that infanticide, "especially of female infants," was the most important restraint on the proliferation of early man. While female infanticide has at times been necessary for survival of the community-at-large, there have also been instances where it has been related to the general societal prejudice against females which characterizes most the male-dominated cultures.

The data on female death is truly amasing. Estimates indicate that 30.5 million females are "missing" in China, 22.8 million in India, 3.1 million in Pakistan, 1.6 million in Bangladesh, 1.7 million in West Asia, 600,000 in Egypt, and 200,000 in Nepal.[2]

It is clear that the onerous costs involved with the raising of a girl, end eventually providing her an appropriate marriage, dowry, was the single most important factor in allowing social acceptance of the murder at birth in India. Females are considered as only consumers and that a serious financial burden to poor families. They were therefore often killed at birth.

Incidence

"In 1966, the United States had 10,920 murders, and one out of every twenty-two was a child killed by the parent".[2] Today, infanticide is still most commonly seen in areas of severe poverty. Killing of an infant was described as a crime that was committed by the mother giving birth to that child in medieval times; such likelihood remains true even today. Although men are more likely to murder in general, statistical review of prosecutions show that killing of infant is usually committed by the mother. When mothers killed their children, however, the victim was usually a newborn baby or younger infant within the age of one year.[2] Some research shows that for murders of children over the age of one year in the United States, white fathers were the perpetrators 10 per cent more often than white mothers, and black fathers 50 per cent more than black mothers.[1,2]

Killing of female infant occurs mostly among the poor, in rural population. In India, the main factors that is responsible for the increase in the incidence of killing female infant or foetus is believed to be factor of low status of women, son preference, and the practice of dowry across all casts groups.[2,3]

Other risk factors can include young maternal age, low level of education and employment, and signs of psychopathology, such as alcoholism, drug abuse or other criminal behavior. The most common method of killing children over the ages has been head trauma, strangulation and drowning. Most of the murders today are committed with the use of the mother's hands, either by strangulation or physical punishment.

INFANTICIDE

Definition

Infanticide is defined as the deliberate, unlawful, destruction of a child under the age of one year, by act of omission or act of commission.

However, in India, from the legal point of view, irrespective of the age of the child, the offences against children are dealt with in the same lines as adult. Thus, in our law there is no distinction between infanticide and child murder. Thus, infanticide is considered as killing of a child below the age of one year, and legally it is amounting to homicide.

Investigation in a Case of Death of a Newborn and Infant

Examine the case by meticulous autopsy and establish answer for the following questions:

- Whether the child is viable or not?
- Whether the child—live born or stillborn or dead born?
- Time of survival if live born.
- Cause and manner of death.

Whether the Child is Viable or Not?

Viable child is a foetus, which has completed of 210 days (7 months) of intrauterine life (IUL) and capable of leading a separate existence after birth. Further questions like live birth, duration of survival and cause of death etc. arise only if it is above the age of viability.

Medicolegal Importance

If on autopsy, the child or newborn is found to be not viable then the charge of infanticide stands withdrawn. Thus, every doctor examining a case of infanticide must establish whether the child or fetus examined is viable or not.

Establishing the viability: Examination of the infant by proper autopsy techniques is the accurate method to establish the viability and following two methods are adopted.

- By considering the foetal developmental changes suggestive of attaining the viability
- By Haase's formula.

Foetal developmental changes: Foetus must be examined for the developmental changes suggestive of viability, as depicted in Table 27.1 and Figures 27.1A to G.

Haase's rule/formula: If the crown-heel length (vertex to heel length) of a foetus is known, its intrauterine age (IUA) can be determined as:

Principles

- Up to 5 months, age of the foetus = Square root of length of foetus (in months)
- > 5 months = length of the foetus (in cm)/5 = (months) (Tables 27.2A and B).

Thus:

- Up to 25 cm of crown-heel length of the fetus, intrauterine age (IUA) in months is calculated by taking square root of the length. Thus, if crown-heel length is 9 cm, the IUA is determined as:

  IU Age = $\sqrt{9}$ = 3 months.
- Beyond 25 cm of crown-heel length, intrauterine age (IUA) of the foetus is calculated by determining one-fifth of the Crown-heel Length, i.e. dividing the Crown-Heel Length by 5, gives IU Age in months. Thus, if the crown-heel length is 40 cm

  IU Age = 40 × 1/5, i.e. 40 ÷ 5 = 8 months

Table 27.1: Foetal autopsy: External and internal developmental changes of viability

	Features	*Observations*
I. External	Length at 7 months IUL	35-45 cm
	Weight at 7 months IUL	1060 gm
	Eyes: Eye brows/lashes	Formed
	Eyelids	Open
	Pupillary membrane	Absent
	Limbs	Present
	Hands and feet	Present
	Fingers and toes	Present
	Nail growth	Present
	Umbilical cord	Present
	Placental weight	350-400 gm
	Sex	Formed
	Hairs	Lanugo
	a. Body	+ve
	b. Scalp	+ve
	Skin—vernix caseosa	Present
II. Internal	Ossification centres talus	Appeared
	2nd piece of sternum	Appeared
	Brain convolutions	Formed
	Intestines	Present
	Gallbladder bile	Present
	Meconium in colon	Present
	Caecum	In right iliac fossa
	Testis	Between kidney and inguinal canal

Measuring Crown-heel Length

It is done by measuring the actual length of the foetus placed stretched on a flat surface from top of the head to the heel, utilising a metallic scale actual/flexible measuring tape. It can also be measured using osteometric board, used for measuring the lone bones in osteometry.

LIVE BORN, STILLBORN AND DEAD BORN CHILD[4-7]

Live Born Child

A child is considered live born, if any of its part is out of mother's reproductive passage, though it has not breathed or completely born (Figs 27.2A and B).

Stillborn Child

A stillborn is one who is born after 28 weeks (IU age) of pregnancy, and it did not show any signs of life, at any time

Table 27.2A: Changes in foetal length and weight at birth and after birth[5-15]

S. No.	*Parameters*	*Observations*
1.	Length at birth	Range: 45-34 cm and Median: 50 cm[5-9]
2.	Length at the end of 6 months	60 cm
3.	Length at the end of 1 year	68 cm
4.	Length at the end of 4 years	100 cm (double of length at birth)
5.	Weight at birth	Range: 2500–4000 gm and Median: 3400 gm[10-14]
6.	Weight at the end of 5 months	6800 gm (doubles birth weight)
7.	Weight at the end of 12 months	10200 gm (triples birth weight)

Table 27.2B: A rough method of calculating the age of the foetus

Haase's rule:

- Up to 5 months, age of the foetus = Square root of length of foetus (in months)
- > 5 months = length of the foetus (in cm)/5 = (months)

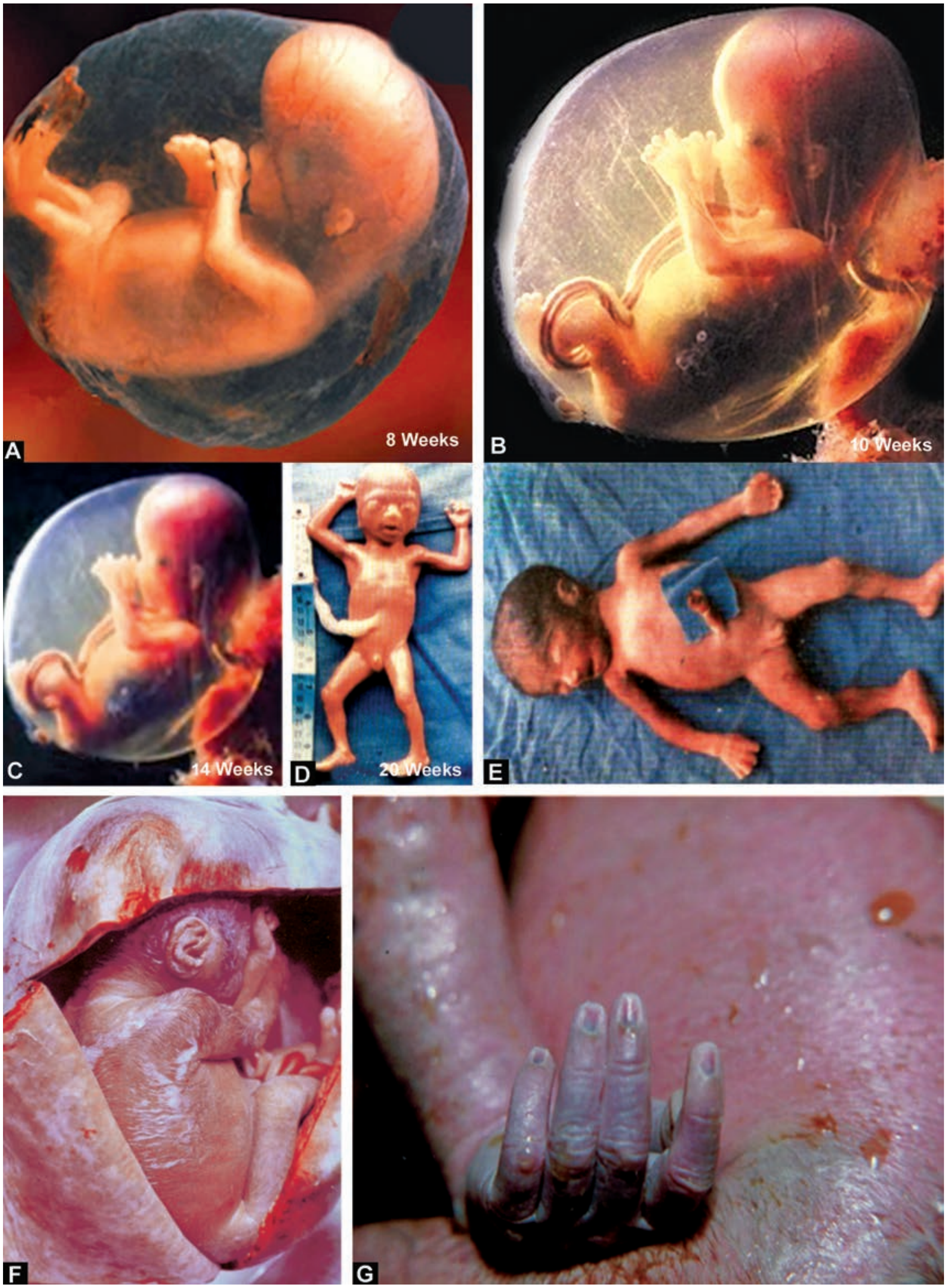
For confirmation of age between 6 and 12 years:

- Best is dental examination.

For confirmation of age between 14 and 25 years:
Fusion of bones:

- 14 years : Patella completely ossifies
- 14-25 years : Sternum fusion takes place below upwards
- 15-16 years : Elbow joint
- 16-17 years : Ankle joint
- 17-18 years : Hip joint
- 18-19 years : Knee, shoulder, inner end of clavicle centres appear.
- 18-20 years : Iliac crest fuses.
- 21 years : Fusion of ischial tuberosity and inner end of clavicle.
- 22 years : Epiphyseal union of sternal end of clavicle

Bertillon's system : For age >21 years.



Figs 27.1A to G: *Foetus of different intrauterine age and developments:* (A) 8 weeks, (B) 10 weeks, (C) 14 weeks, (D) 20 weeks IUL, (E) Full term, (F) Full term foetus in the cut opened uterus with lanugo hairs, vernix caseosa, and in a universal flexion position, (G) Foetal hand showing fully grown nails at the finger tips, full term. *Courtesy:* (Figs 27.1F and G) Dr B Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

after being completely born. In this the foetus was alive in uterus but dies in the birth canal after the birth process has initiated.

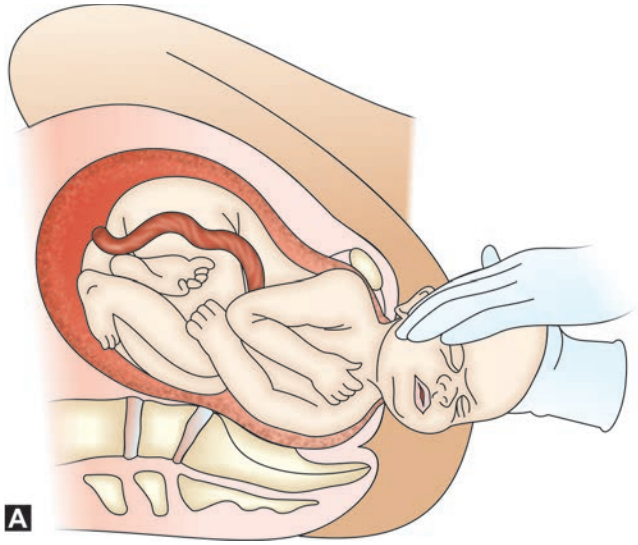
Dead Born Child

A dead born child is one which had died in uterus before the birth process has started and shows rigor mortis, maceration or mummification at birth.

Establishing Whether Live Born?

In civil cases: Following are considered signs of live birth:

- Baby's cry (vagitus vaginalis/vaginus, vagitus uteralis/uterinus, i.e. baby's cry inside the vagina or uterus respectively during delivery).
- Muscle twitching/movements of limbs, *etc.*



Figs 27.2A and B: (A) Concept of live birth: Full term is on the process of birth—head and face out of birth canal—is considered as a live born, (B) Just born baby

In criminal cases: Signs of live birth have to be demonstrated by autopsy examination of the newborn as usually law presumes that "Found dead is born dead".

External Autopsy Findings

- General findings
- Ear changes
- Changes in the chest
- Changes in the umbilical cord (Figs 27.3A to F).

General findings: Absence of vernix caseosa and presence of clothing are suggestive of live birth.

Ear changes: Absence of embryonic connective tissue or presence of air in the middle ear is suggestive of live birth.

Changes in the chest: Table 27.3 summarises the changes in the chest of the child, which help to decide whether respired or not and there by live or dead born.

Changes in the umbilical cord: Presence of ligature/marks of crushing by the artery forceps is suggestive of live birth. Table 27.4 summarises other changes in the stump attached to the child, which are not only suggestive of live birth, but also help to assess time since birth.[17-19]

Table 27.3: Changes in the chest

Features	*Respired*	*Unrespired*
Shape	Arched/drum shaped	Flat
Circumference	More and greater than the abdomen	Less than abdomen
Intercostal space	Increases/wider	Narrow
Diaphragm	At the level of 6th or 7th rib	At the level of 4th or 5th rib

Table 27.4: Time since birth by changes in the umbilical cord

Changes observed	*Time since birth*
1. Shrinkage of blood vessel lumen	Just born
2. Drying up at cut end	24 hr (1 day)
3. Inflammatory line at the base of the stump	48 hr (2 days)
4. Obliteration and mummification changes in artery	72 hr (3 days)
5. Obliteration in veins	120 hr (5 days)
6. Detach-fall off	144 hr (6 days)
7. Complete healing of the umbilicus	240 hr (10 days)

Internal Autopsy Findings

Signs of live birth observed in the viscera such as: lungs, heart and gastrointestinal tract are as follows.

Changes in the Lungs

Changes are mainly because of respiration and are depicted in Table 27.5.

Confirmatory Tests for Respiration in Lungs

- Plaquet's test
- Hydrostatic test (floatation test)
- Histological examination of lung (Figs 27.4A and B).

Plaquet's Test

In this test, weight of the lung and body weight are compared. Normally the ratio of lung: body weight is 1:35. In unrespired lung this ratio cannot be maintained.

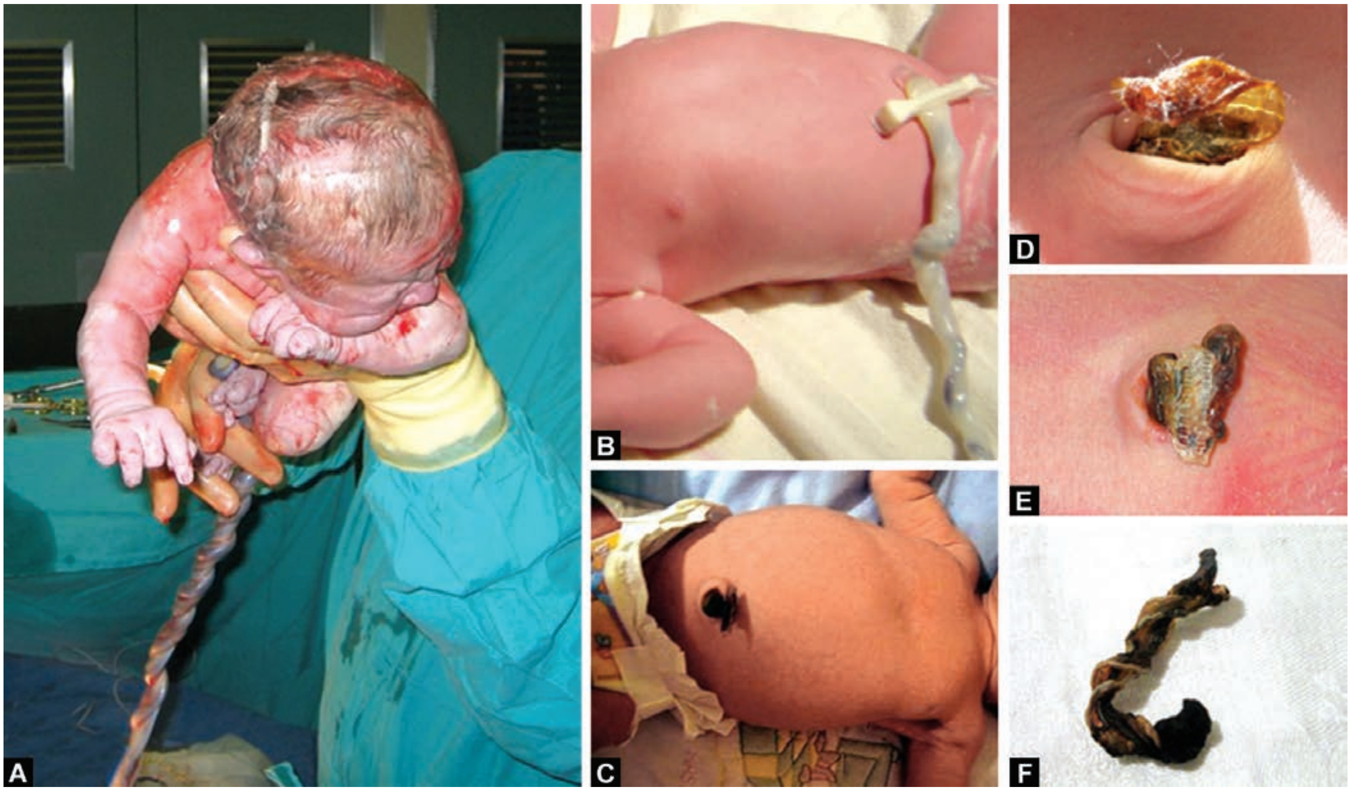
Hydrostatic Test (Floatation Test)

Bernard Knight in his classic *Forensic Pathology*, 2nd edition concludes 'Hydrostatic test is lightly studied black magic and is a complete waste of time' However, I differs with him pronounce that a salmon-pink spongy lungs that floats in water, is diagnostic of an infant who has breathed provided there is absence history of resuscitation and no putrefaction commenced, are most in keeping with lungs from." Here he shows aerated lungs floating in water in a case of alleged stillbirth without resuscitation. Byard refrains from giving his conclusions in this case, but he seems to have opined in favour of a live birth.

Principle: Air that has entered into lung tissue during respiration makes it lighter and floats in water. It is also a fact that specific gravity of the lung before respiration is 1040-1050, which becomes 0.940 to 0.950 which is less than that of water, after respiration, and makes the respired lung float.

Procedure

- Dissect out the fetal lungs
- Put both the lungs into a trough of water and observe.



Figs 27.3A to F: *Umbilical chord in different phases of healing:* (A) Just born newborn baby with umbilical cord attached just after a caesarean section, (B) Umbilical cord 3 minutes old age child with clamp applied, (C) Umbilical cord stump healing, (D) Umbilical cord stump of 7 days old baby, (E) Umbilical cord before falling, (F) Umbilical cord stump healed and detached from umbilicus

Table 27.5: Lung changes due to respiration

Features	*Respired*	*Unrespired*
I. Gross findings:		
Colour	Mottled pink	Bluish red or uniformly reddish brown
Volume	High/voluminous	Low/small
Position in thoracic cavity	Occupies fully the thoracic cavity, overlaps heart	Not full. Lies at the back of the cavity behind heart
Pleura	Taut and stretched	Loose and wrinkled
Diaphragm	At 6th rib level	At 3rd rib level
Margins/edges	Round	Sharp-liver like
Surfaces	Uneven	Smooth
Consistency	Spongy (crepitous)	Solid-liver like
Weight	1000 gm	About 500 gm
II. Cut section:		
Blood oozing	+ve	–ve

Inference

- If they sink—unrespired lung
- If they float—remove them form water, cut into small pieces and then squeeze or press firmly between sponges, again put into the water column
- If they sink—unrespired lung
- If they float—respired lung.

Explanation

The floatation observed in the test above for the second time is mainly because of the "residual air" that remains in the lungs, which cannot be squeesed out by pressing, if the child has breathed after birth.

Fallacies

A false-positive test of floatation of lung pieces may be observed in conditions such as:

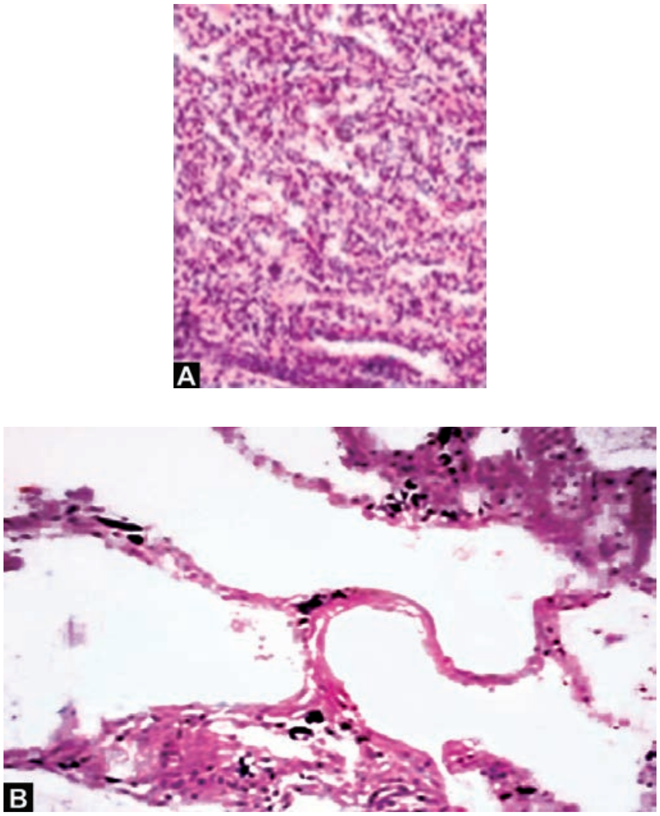
- Accumulation of putrefying gases
- Air pushed into lung by artificial respirators, etc.

A false-negative test of sinking of lung pieces can be observed in conditions such as:

- Atelectasis
- Pneumonic consolidation, etc.

Histology of Lung

Unrespired lung looks like section of parotid gland mainly because the alveoli with lining epithelium which is cubodial, while on



Figs 27.4A and B: *Microscopy of foetal lung:* (A) Before respiration (unrespired lung). Note the glandular appearance as alveoli are yet to be filled with air and lining epithelium look like cuboidal epithelium, (B) After respiration (respired lung). *Note:* Flattened epithelium with expanded alveoli filled with air

entry of air into it, the cells get flattened with dilatation-pavement epithelium (*see* Figs 27.4A and B).

Heart Changes

To understand this certain basics on foetal circulation may be essential and is discussed below (Figs 27.5 and 27.6) in brief:

When the baby is still in the mother's womb it does not need to breathe for itself as the mother, via the umbilical cord, is supplying all the oxygen that the baby needs. The circulation before birth is different from that after birth. It is designed so that the oxygen filled blood from the umbilical cord goes to the most important part of the body, for example the brain. Thus, very little blood needs to go to the lungs.

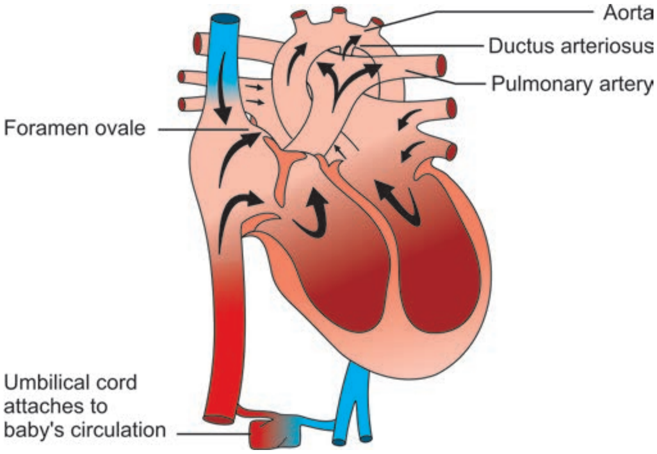

Aorta
Ductus arteriosus
Pulmonary artery
Foramen ovale
Umbilical cord attaches to baby's circulation

Fig. 27.5: Showing foetal heart and blood flow in it (*Internet source*: http://www.lhm.org.uk/Info/circulation-before-birth-35.aspx)

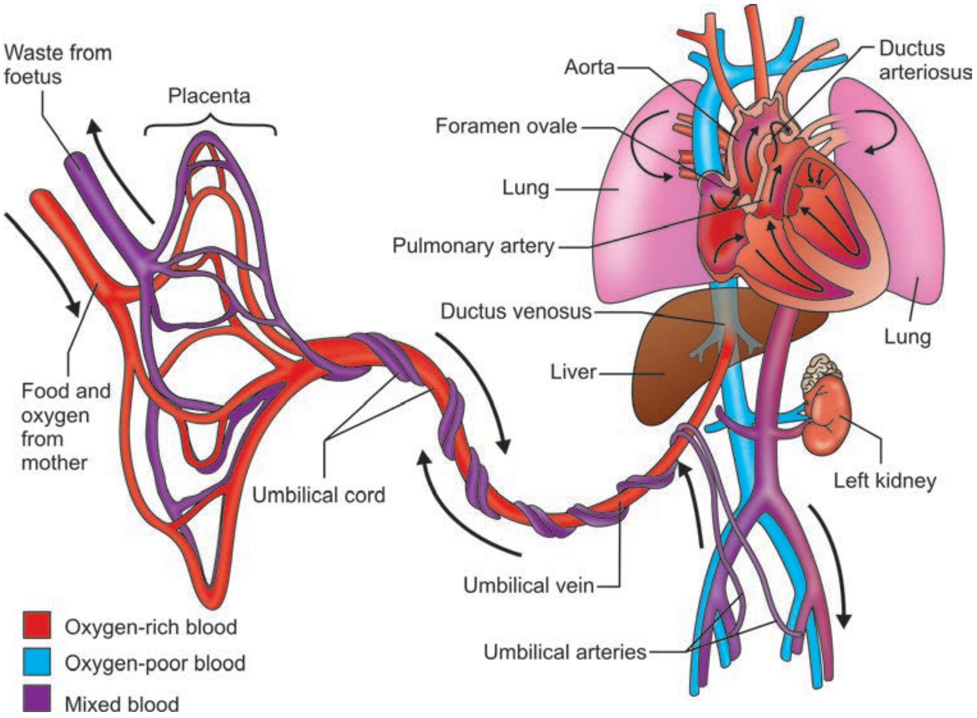

Waste from foetus
Placenta
Aorta
Ductus arteriosus
Foramen ovale
Lung
Pulmonary artery
Ductus venosus
Lung
Liver
Food and oxygen from mother
Left kidney
Umbilical cord
Umbilical vein
Umbilical arteries
🟥 Oxygen-rich blood
🟦 Oxygen-poor blood
🟪 Mixed blood

Fig. 27.6: Showing foetal heart and blood flow in it
(*Internet source*: http://www.childrenscentralcal.org/HealthE/PublishingImages/em_0181.gif)

There is a hole between the top collecting chambers (in the inter atrial septum of right and left atria) called the Foramen ovale: oxygen filled blood passes from the right collecting chamber to the left chamber through the hole and then on into the left pumping chamber (left ventricle) from where it is then pumped around the body.

There is also a connection called the ductus arteriosus which joins the lung artery (Pulmonary artery) and the body artery (Aorta). Blood passes from the right pumping chamber up the lung artery, some blood then passes to the lungs but most flows through the ductus arteriosus to the body artery and then around the body, again avoiding the lungs.

Thus, foramen ovale and ductus arteriosus act as bypass system of foetal circulation. When the baby is born and starts to breathe for itself, these bypass systems are no longer needed. Gradually over the first few days or weeks, after birth, the duct and the hole will close off and the baby's circulation will change to that in a normal heart.

Thus, changes in the heart suggestive of live birth are:
- Ductus arteriosus closure occurs on fourth day of birth
- Foramen ovale closure occurs by second month of birth. GIT changes suggestive of live birth are:
  - Presence of food, saliva, etc. in the stomach
  - Presence of air in the stomach with no changes of decomposition
  - Presence of meconium in intestine (Meconium is inspissated bile and mucus, usually voided out by 24 to 28 hours of birth. So, if meconium is absent in the intestine, child is live birth). Fallacy-if it may be voided off, if the case is of delivery of breech presentation.

Establishing Whether Dead Born

The dead born fetus may undergo a typical change, which is called maceration.

Macerated foetus: It is a state of aseptic autolysis occurring after the intrauterine death of a foetus (Fig. 27.7). On examination a macerated fetus shows following features:
- Emits a rancid odour
- Body is soft and pliable—it flattens when kept on a table
- Skin—sodden and coppery red/purplish (never greenish as with putrefaction) with blisters, and peeling cuticle
- Abdomen—distended
- Umbilical cord—thick, red smooth, soft.
- Joints—abnormally mobile or flexible
- Body cavity—presents reddish serum within
- Organs/viscera—soft and edematous, loose their morphology, but lungs and uterus remain unaffected for long period.
- Skull bones—loss of alignment and over-riding of bones of cranial vault and overlapping (seen on radiograph, called as *Spalding's sign*) due to shrinkage of brain after death seen within few days of death of foetus[16-17] (Fig. 27.8).

Time required: Maceration begins or sets in 5 to 7 days prior to expulsion, thus, it needs about 5 to 7 days time to form.[17]

Grades of Maceration (Table 27.6)

There are five-grade scales based primarily on the external characteristics of the dead born. That scale of severity of maceration is as follows:[16]
1. None
2. Slight—skin slippage, rare bullae, little (e.g. scrotum only or single spots of skin loss elsewhere) or no denudation.
3. Mild—focal denudation of multiple regions without other changes
4. Moderate—generalised skin maceration/ denudation but without significant compressive changes
5. Advanced—compression and/or mummification and/or internal liquefaction.

Based on these findings we can better estimate the relationship between maceration scale and time interval between death and delivery (Table 27.7).[16]

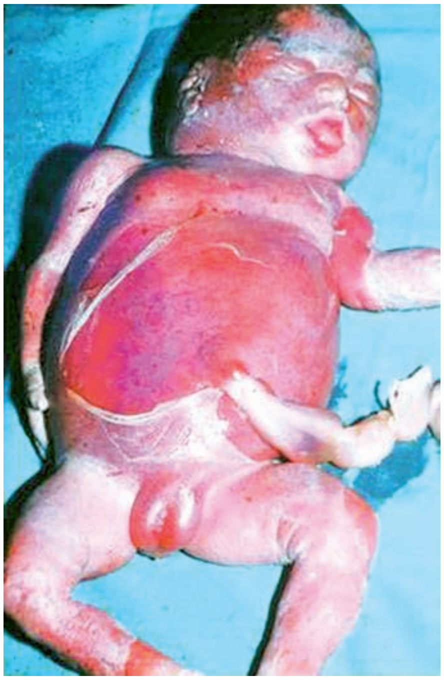


Fig. 27.7: *Dead born macerated foetus. Note* the soft pliable body, coppery red skin, thick umbilical cord, distended abdomen, etc

Table 27.6: Showing six of the measures applicable scale used in grading maceration[16]

Features	*Interval between death and delivery*
• Skin desquamation of > or =1 cm	> or = 6 hours
• Skin desquamation involving the face, back and/or abdomen	> or =12 hours
• Skin desquamation involving at least 5 per cent of body surface	> or = 18 hours
• Change of skin coloration to tan or brown	> or = 24 hours
• Generalised skin desquamation	> or = 24 hours
• Mummification	> or = 14 days

Time of Survival If Live Born

A rough estimation on how long a child survived after birth can be done by following data.
1. Caput succedaneum changes: It is a haematoma formed within the skin of presenting part of baby during delivery. It begins to disappear by 24 hours of birth and completely not seen by 7 days of birth. If the infant examined shows the presence of Caput, then the period of survival after birth can be given 7 days (Figs 27.9A and B).
2. Umbilical cord changes (refer above)

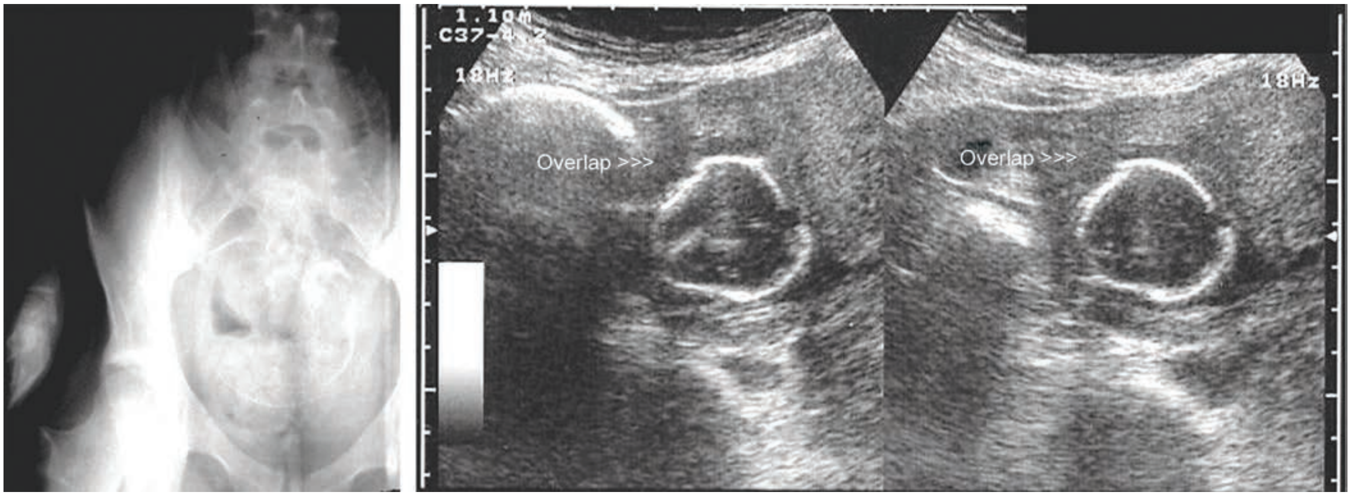

18Hz
Overlap >>>
Overlap >>>
18Hz

Fig. 27.8: *Spalding's sign*[16-17] X-ray (left) and ultrasound (right) of the foetal skull. *Note* overlapping of skull bones and ribs—IUD of foetus

3. Gastrointestinal changes (refer above)
4. Cardiovascular changes (refer above)
5. Changes in blood
    - Normoblasts will be present till 24 hours after birth.
    - Fetal haemoglobin will be detected in first six months after birth.

Cause of Death

Death of a newborn could be due to several reasons. Practice of perinatal autopsy in developed countries has revealed wide variety of causes and commonly they are shown in Flow chart 27.1.

Table 27.7: Time interval between death and delivery by maceration scale

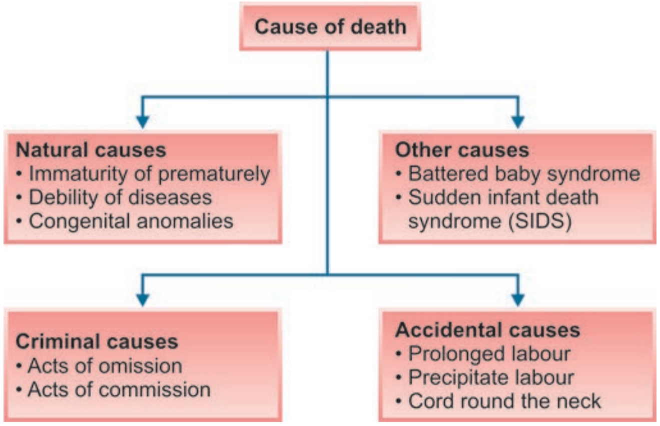
Maceration	*Interpretations of time interval between death and delivery*
1. None	Intrapartum death
2. Slight	Less than 12 hours
3. Mild	About 12-24 hours
4. Moderate	1-2 days
5. Advanced	More than 2 days

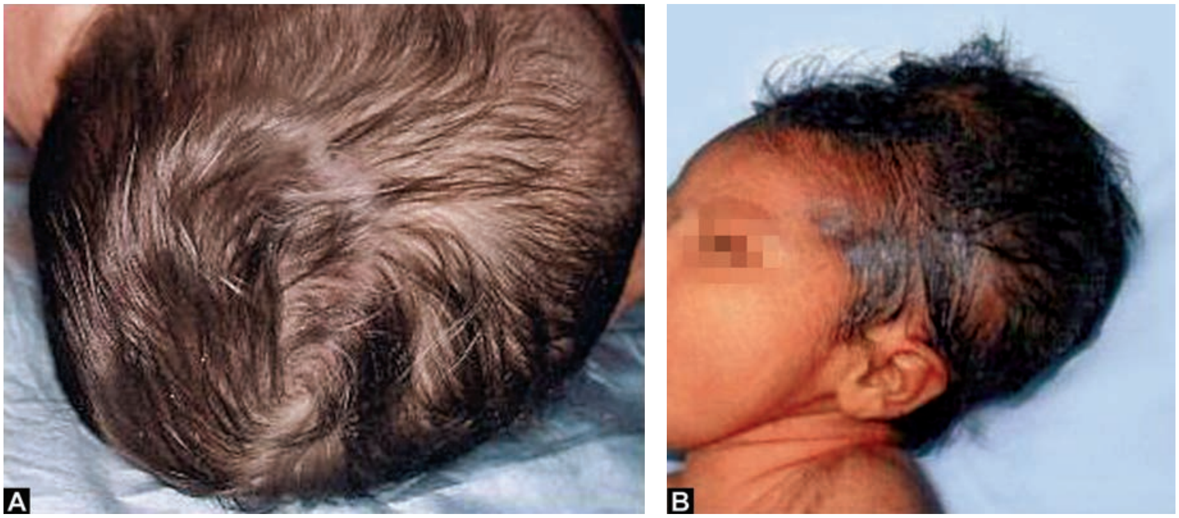
Natural causes such as immaturity or prematurity, debility or diseases, congenital anomalies being discussed elsewhere, others in the flow chart are considered here for brief discussion:

Acts of omission This includes, not doing certain things necessary for an infant, e.g.

- Avoid ligating umbilical cord before cutting
- Avoid feeding the infant, etc.

Flow chart 27.1: Causes of death in a newborn


Cause of death
Natural causes
• Immaturity of prematurely
• Debility of diseases
• Congenital anomalies
Other causes
• Battered baby syndrome
• Sudden infant death syndrome (SIDS)
Criminal causes
• Acts of omission
• Acts of commission
Accidental causes
• Prolonged labour
• Precipitate labour
• Cord round the neck



Figs 27.9A and B: Caput succedaneum due to vacuum suction applied on presenting part—head

Section 317 of IPC: Provides punishment of imprisonment of either description for a term which may extend to seven years or with fine or both for "Exposure and abandonment of child less than twelve years, by parent or person having care of it".

Acts of commission: This includes doing certain deliberate acts resulting in the death of the infant, e.g. smothering the infant to death, strangulation and head injury.

Prolonged labour: Here the fetus dies in uterus due to certain maternal causes such as contracted pelvis, cephalopelvic disproportion, etc.

Precipitate labour: Here the fetus dies at delivery. The victim is usually a newborn infant, alleged to have died in the toilet, bathroom, etc. Here the mother is always a grand-multi and in such woman, three stages of labour are condensed into one single stage, and the fetus is passed out suddenly with the onset of labour pain taking short time, cause of death in the infant is usually head injury.

Cord round the neck: During the delivery of a foetus the umbilical cord gets wound round the neck accidentally, resulting in death due to asphyxia, the cord acting like a ligature of hanging or strangulation.

FOETICIDE

Foeticide is a unique form of violence killing of a foetus, before being born. The term foeticide usually refer to total destruction of female foetus. Certainly, this is an atrocity against female and is actively being promoted by the sex determination clinics. This has assumed disproportionate growth in India recently by terminating birth of unwanted female child in northern India, while in the south, sex determination tests and abortions being considered as expensive, practice is to prefer to deliver the child and then kill it, if it is a girl.[1-3]

Economics of life has made the parents uncaring and heartless. Daughters are considered economic burdens to the family because of the high cost of weddings and dowries, while sons as providers of income, and are seen as type of insurance/security by their parents.

New prenatal sex-determination techniques, such as ultrasound, have led to an increase in the abortion of female foetuses rather than female infanticide. Female infanticide and abortion have increased in recent years also for other reasons such as women opt now for smaller families. In India the sex ratio is 93 women for every 100 men (i.e. F : M is 93:100), but in some regions this ratio is < 85 women per 100 men (i.e. F : M is <85:100). Research carried out at Mumbai hospitals revealed in 1995 that for every aborted male, there were 1,000 aborted females.[3]

Death is a catastrophe in whichever form, time, way or place it appears. The problem has assumed grave proportions and the sex ratio has been declining fast in India. From 972 females to 1000 males in 1901, it has descended to 933 females to 1000 males in 2001.[4]

However, enactment of the Pre-natal Diagnostic Techniques (PNDT) Act 1994 has though banned sex determination and female foeticide by vigorous legal and administrative provisions, the problem is yet to be eradicated completely.[3] Amendment of 2002, the nomenclature of the Act was re-amended as The Preconception and Prenatal Diagnostic Techniques (Prohibition of Sex Selection) Act, 1994.[3] This necessity emerged out of the fact that in the present setup, sperms can be processed in the laboratory to effect separation of X and Y chromosomes, thus ensuring the birth of a male child, i.e. sex selection take place even before conception. Hence, amendment in the nomenclature of the Act was carried out.[3]

Certain important conditions for regulation of various provisions (Chapter I, Chapter II and Chapters III) under the PNDT Act are furnished below:[3]

- Genetic Counselling Centres, Genetics Laboratories, Genetic Clinics, unless registered under the Act, cannot conduct or associate with, or help in, conducting activities relating to prenatal diagnostic techniques. They cannot employ or cause to be employed or take services of any person whether on honorary basis or on payment who does not possess the prescribed qualifications.
- Prenatal diagnostic procedures cannot be conducted unless
  - i. all side and after effects of such procedures have been explained to the concerned pregnant woman,
  - ii. age of the pregnant woman to be above thirty-five years and she has under gone two or more spontaneous abortions or foetal loss,
  - iii. pregnant woman had been exposed to potentially teratogenic agents such as drugs, radiation, infection or chemicals,
  - iv. pregnant woman or her spouse has a family history of mental retardation or physical deformities such as spasticity or any other genetic disease,
  - v. her written consent to undergo such procedures to be obtained in the language which she understands and a copy of her written consent to be given to the concerned pregnant woman and
  - vi. communication of sex of the foetus by words, signs or in any other manner to the concerned pregnant woman or her relatives or any other person to be prohibited.
- No Genetic Counselling Centres or Genetic Laboratories or Genetic Clinic or any person can conduct or cause to be conducted prenatal diagnostic techniques including ultrasonography for the purpose of determining the sex of the foetus. No person can cause or allow to be caused, selection of sex before or after conception.

Certain offences and penalties (Chapter VII) under the Act are furnished below:[3]

- Advertisement relating to pre-conception and pre-natal determination of a sex or sex selection is prohibited. Contravention is punishable with imprisonment up to three years and with fine up to Rs. 10,000.
- Any medical geneticist, gynaecologist, registered medical practitioner or any person who owns a Genetic Counselling Centre, a Genetic Laboratory or a Genetic Clinic or is employed therein and renders his professional or technical services there, whether on an honorary basis or otherwise and who contravenes any of the provisions of the Act or rules made there under shall be punishable with imprisonment up to three years and with fine up to Rs 10,000 and on any subsequent conviction, with imprisonment up to five years and with fine up to Rs 10,000. The name of the registered medical practitioner shall be reported to the State Medical Council concerned for taking necessary action.
- If any person seeks aid for sex selection or for conducting pre-natal diagnostic techniques on any pregnant woman, he shall be punishable with imprisonment up to three years and with fine up to Rs. 50,000 for the first offence and for any subsequent offence, with imprison up to five years and with fine up to Rs one lakh.

Statutes and Case Laws in Relation to Foetus in India

Death of a child inside the mother's womb is not homicide. However, this may amount to culpable homicide as foeticide causes death of a living child inside mother's womb, and is been brought forth, though the child may not have breathed or been not completely born (Section 299 of IPC). This explanation makes two things very clear: Firstly, it excludes human fetus from the category of a human being. Secondly, it makes infanticide dependent upon the foetus being sufficiently advanced/developed to have assumed the human shape/ existence and its emergence from the mother's womb. In a recent case at Maharashtra State Commission, in the case of Kanta Mohan Lal Kotehca Vs United India Insurance Co. Ltd, it ruled that claim in respect of the unborn child was maintainable.

The Tribune in 2003 reported an interesting case of doctor couple who were booked and sent to judicial remand following death of a patient at their nursing home while conducting abortion. The postmortem conducted by a board of doctors confirmed 16-20 weeks of gestation and abortion prior to her death. Violation of the provisions of the Act by the doctor couple, the registration certificate of the nursing home had earlier been suspended by the Appropriate Authority under the PNDT Act. Despite this, the doctor couple conducted sonography on the patient. The nursing home was also not registered under the MTP Act (1971) and hence, the couple had grossly violated other legal provisions.

Police registered a case against the doctor couple under Section 304A of IPC initially, was later on converted under Section 304 and Section 201 and the PNDT Act sub-sections 3, 4, 6, 22, 23 and 25 were added. During proceedings for bail, the counsel for the doctor couple argued that though the centre was not registered under the MTP Act, doctors could perform an abortion for saving the life of the patient as mentioned in the sub-section 2 of Section 3 of MTP Act. He further contended that Section 304 of IPC could not be imposed on the doctor couple there was no evidence to prove that they had a any specific intention to kill the patient.

Another point to be focused is, as alleged by prosecution, that the doctor couple had not maintained the necessary documents required under the Act. However, the bail granted to the doctor couple by the Magistrate was contested by the State in the Sessions Court by arguing that the lower court, in granting bail, had gone beyond its jurisdiction. The main contention of the public prosecutor for the cancellation of bail was that the Judicial Magistrate exceeded his jurisdiction by observing in his order granting bail that offence under Section 304 IPC was not made out. However, the counsel for the doctor couple furthered the argument by stressing that the prosecution had initially applied only Section 304A (which was a bailable offence) and later on replaced it by Section 304 and adding some other sections too, to make the case non-bailable. The bail granted by the Magistrate was set aside by Sessions Court laying down that 'observation of the Magistrate that offence under Section 304 was not made out at the stage when the investigation was still on was not correct.' The Judge ordered that the remand papers, etc. should be put before the Chief Judicial Magistrate for reconsideration of bail application, considering that offence under Section 304 IPC was there.

BATTERED BABY SYNDROME[18-21]

(Synonyms: Caffey Syndrome)

Definition

The battered baby syndrome is defined as a clinical condition in young children usually under three years of age, who have received nonaccidental violence or injury, on one or more occasions at the hands of an adult, a parent, guardian, or foster parent. In addition to physical injury, there may be deprivation of nutrition, care and affection in circumstances, which indicate that such deprivation is not accidental.

Causes

The victim is often an unwanted child, an illegitimate child, or a child whose father's paternity is doubted. The precipitating factor is usually a cry, which interferes with either a parent in sleep or the outing, or their television programme. Battering is result of sudden loss of temper under such and allied circumstances.

The type of persons involved in child battering has frequently a low IQ. Some have a history of family discord, long-standing emotional problems and financial stress, while others have a history of criminal background. Recent reports suggest that such parents had received similar treatment from their parents in their own childhood.

Injuries Sustained

Injuries are commonly multiple, although all are not necessarily severe (Figs 27.10A to D). They usually follow a pattern, with one or more severe localised bruises on the head quite inconsistent with a simple fall, or bruises on face, trunk and extremities consistent with grip marks. Tearing of the frenum of upper lip and of the alveolar margin of gums to stifle cries is commonly encountered.

Major injuries, which prove fatal, include head injuries, e.g. fractured skull and subdural haematoma or visceral injuries, e.g. ruptured liver and mesenteric hemorrhage. Clinical and radiological evidence may be obtained that injuries have occurred at different times.

Diagnosis of the Syndrome

The syndrome must be considered in any child:

- In whom the degree and type of injury is at variance with the history given
- When injuries are of different ages and in different stages of healing.
- When there is purposeful delay in seeking medical attention despite serious injury
- Who exhibits evidence of fracture of any bone, subdural hematoma, failure to thrive, soft tissue swelling or skin bruising, or
- Who dies suddenly.

  Figures 27.11 and 27.12 illustrate typical features of accidental and non-accidents injuries.

Medicolegal Importance

- The history may be completely misleading as to the circumstances surrounding death.
- The external and internal examination should be very thorough and supported by photographs, radiographs, microscopic sections of all pertinent lesions, and toxicological analysis.

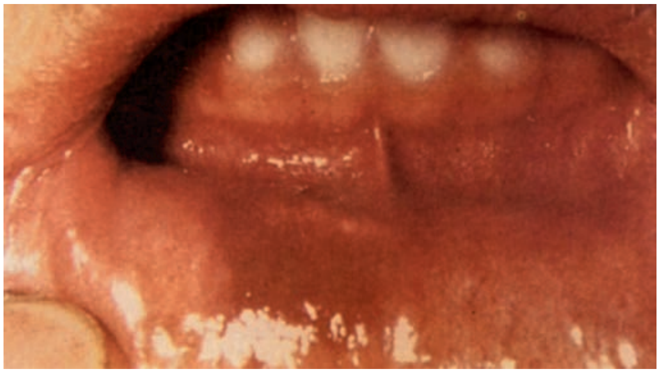


Fig. 27.10A: *Battered baby syndrome:* Contusion of inner aspect of lower lips, vestibule and labial frenum

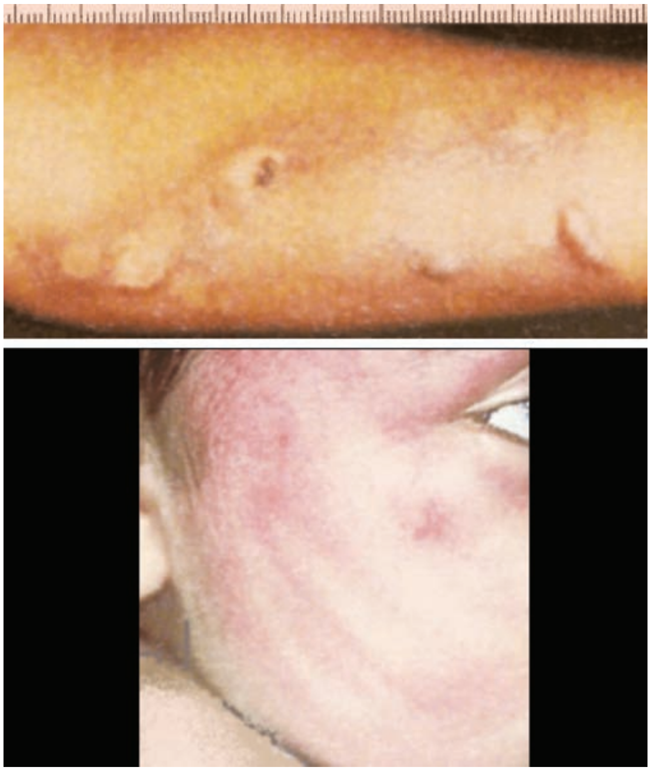


Fig. 27.10B: *Battered baby syndrome:* Multiple cigarette burn marks at different stages of healing on left forearm (above) and slapping mark on the face (below)

- In India as per the eastern culture babies are considered as gifts from god and cases of battered baby syndrome are rare. However, instances of ill treatment of young children (child abuse) who work as domestic servants are not uncommon.
- Charges of infanticide.

SUDDEN INFANT DEATH SYNDROME—SIDS[18-21]

(Synonyms: Cot Death, Crib Death)

Definition

Sudden infant death syndrome (SIDS) is a condition in which apparently healthy infants are found dead without any signs or symptoms that would have enabled such an event to be predicted, and on postmortem examination, there is insufficient pathology to explain their death satisfactorily.

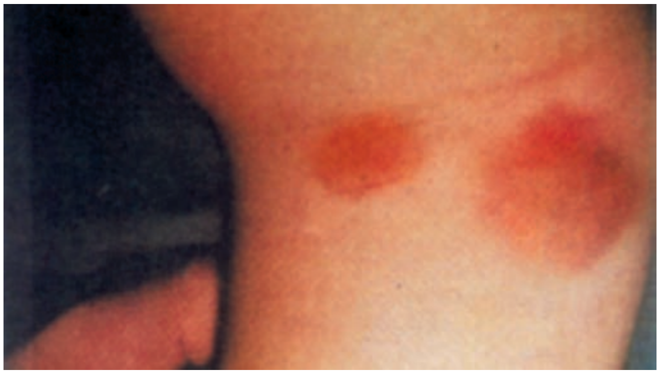


Fig. 27.10C: *Battered baby syndrome:* Grip marks on outer and upper aspect of the right thigh

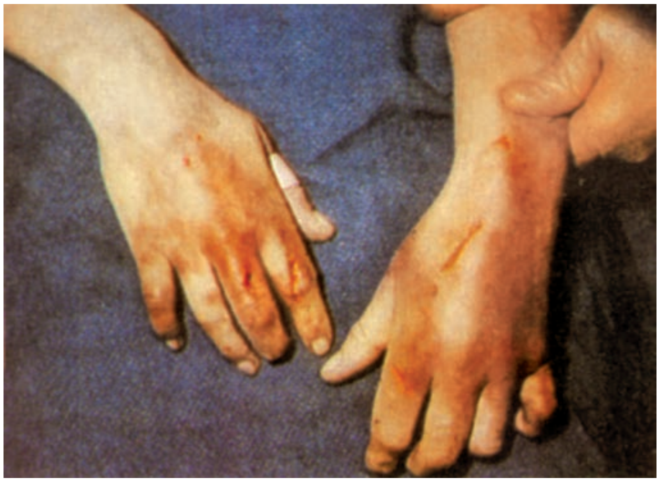


Fig. 27.10D: *Battered baby syndrome:* Beating on the hands for stealing

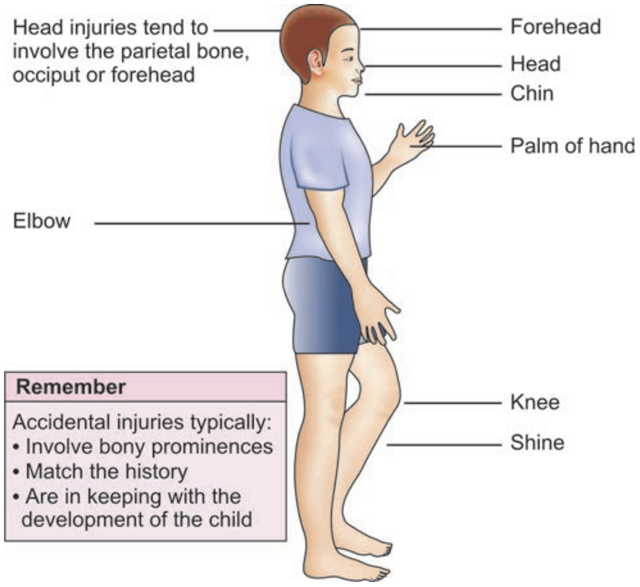
Incidence

The death rate is between 2 and 3 per 1000 live births. Death usually occurs between the ages of two weeks and six months, strikes boys somewhat more often than girls, is more common among low birth weight (LBW) babies and among lower income families, among children whose mothers smoke or are drug addicted and is commonly associated with seasonal upper respiratory diseases. About half of the victims have some symptoms of a cold during the week prior to death.

Causes

Aetiology on this is obscure. However, following are suspected to be the true causes:

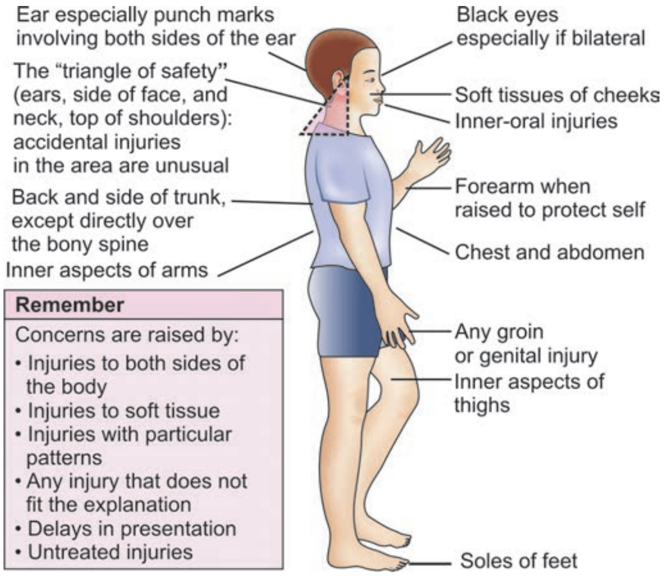
1. *Viral infection.*
2. *Milk allergy.*
3. *Autobeverage syndrome*— The enzymatic content in the stomach converts the milk into alcohol, resulting in alcohol poisoning.
4. Respiratory infection.
5. *Overlaying*— If the mother has a habit of feeding her baby lying on the cot, there is every possibility that mother may overlie the baby, breast of the mother may press the face of the infant, resulting in smothering and death. An important and widely accepted view is 'sleep apnea' leading to death.


Head injuries tend to involve the parietal bone, occiput or forehead
Forehead
Head
Chin
Palm of hand
Elbow
Knee
Shine

Remember

Accidental injuries typically:
- Involve bony prominences
- Match the history
- Are in keeping with the development of the child

Fig. 27.11: Typical features of accidental injuries


Ear especially punch marks involving both sides of the ear
The "triangle of safety" (ears, side of face, and neck, top of shoulders): accidental injuries in the area are unusual
Back and side of trunk, except directly over the bony spine
Inner aspects of arms
Black eyes especially if bilateral
Soft tissues of cheeks
Inner-oral injuries
Forearm when raised to protect self
Chest and abdomen
Any groin or genital injury
Inner aspects of thighs
Soles of feet

Remember

Concerns are raised by:
- Injuries to both sides of the body
- Injuries to soft tissue
- Injuries with particular patterns
- Any injury that does not fit the explanation
- Delays in presentation
- Untreated injuries

Fig. 27.12: Typical features of non-accidental injuries (Injuries which are suspicious)

6. *Shaken infant syndrome*— Violent shaking as a part of playing with the kid may produce intracranial haemorrhage due to hyperextension or hyperflexion.

Autopsy Findings

At autopsy, the trachea contains a small amount of edematous fluid, sometimes blood stained petechial hemorrhages are found on the visceral surface of pleura, pericardium and thymus, and often there is microscopic evidence of respiratory inflammation. There is usually evidence of a brief burst of spasmodic motor activity, bladder and bowels are empty and sometimes blanket fibres are found under the fingernails.

Medicolegal Importance

Charge of infanticide is confirmed.

CHILD SEXUAL ABUSE (CSA)

Rape of a child is a form of child sexual abuse. When committed by a parent or other close relatives such as grandparents, aunts and uncles, it is a form of incest and when committed by another child (usually older or stronger), it is a form of child-on-child sexual abuse. When a child is raped by a family member, especially a parent, it can result in serious and long-term psychological trauma.[22] When a child is raped by an adult who is not a family member but it a caregiver or in a position of authority over the child, such as school teachers, religious authorities, or therapists, to name a few, on whom the child is dependent, the effects can be similar to incestual rape.

Psychologists estimate that 40 million adults, 15 million of those being men (Adams 1991), in the United States were sexually abused in childhood often by parents, close relatives and other elders on whom they were dependent.[23] According to the National Centre for Victims of Crime 46 per cent of rape committed in the United States is perpetrated by a family member.[23,24]

Effects of child rape include depression,[24] post-traumatic stress disorder,[25] anxiety,[26] propensity to re-victimisation in adulthood[27] and physical injury to the child, among other problems.[28] Children, including but not limited to adolescents, raped by their parents and other close elders are often called 'secret survivors' by psychologists, as they often are unable or unwilling to tell anyone about these rapes due to implicit or explicit threats by the adult rapist, fear of abandonment by the rapist, and/or overwhelming shame. Since the signs of these rapes are usually invisible except to trained professionals, these children often suffer ongoing offenses in silence until independence from the adult rapist is attained. By that time, the statute of limitations is often long-expired, the adult victim's repressed memories are often considered inadmissible as evidence and the child-rapist is able to avoid punishment.

More than 67,000 cases of rape and sexual assaults against children were reported in 2000 in South Africa. Child welfare groups believe that the number of unreported incidents could be up to 10 times that number. A belief common to South Africa holds that sexual intercourse with a virgin will cure a man of HIV or AIDS. South Africa has one of the highest numbers of HIV-positive citizens in the world. According to official figures, one in eight South Africans is infected with the virus. Edith Kriel, a social worker who helps child victims in the Eastern Cape, said: "Child abusers are often relatives of their victims—even their fathers and providers".[29]

According to University of Durban-Westville anthropology lecturer and researcher Suzanne Leclerc-Madlala, the myth that sex with a virgin is a cure for AIDS is not confined to South Africa. "Fellow AIDS researchers in Zambia, Zimbabwe and Nigeria have told me that the myth also exists in these countries and that it is being blamed for the high rate of sexual abuse against young children".[29]

Incest between father and daughter is more common than reported all over the world. However, recently, in the last few years there is a remarkable increase in claims and admission of sexual abuse of children. Unfortunately in lower social class misbelieve prevails that venereal diseases can be cured by sexual intercourse with virgin and hence female child is common victim.

Child sexual abuse may be defined as the involvement of dependent, developmentally immature children and adolescents in sexual activities they do not truly comprehend, to which are unable to give informed consent: or which violate social taboos or family roles'.

The problem is huge. The diagnosis and management of CSA are multidisciplinary one, involving not only doctors, but social and sometimes law-enforcement agencies.

Examination of the CSA Case

Probably unless the doctor examining has good experience, opinions are not easy. It is the appreciation of the quite wide range of normal appearances of genitalia and anus that is a key-stone of a reliable opinion on the most serious of problems.

Digital examination of a prepubertal child is rarely indicated and may need to be performed under general anesthesia if thought essential.

Reflex Anal Dilation Test

It is a controversial test and has value as part of the over all examination, but should not be relied upon alone.[18-21]

Procedure: The child is placed in the left lateral position, rather than knee-elbow, and buttocks are gently separated. If the test is positive, the external sphincters relax within 30 seconds to allow the interior of the anal canal to be visible. This dilatation should persist for several seconds, quick reflex puckering is of no diagnostic value.

The possibility of sexually transmitted diseases being present must always be borne in mind. This has both diagnostic and therapeutic aspects for the examining doctor.

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Chapter

28

Forensic Psychiatry

INTRODUCTION

Forensic psychiatry is a subject dealing with application of knowledge of psychiatry in the administration of justice. Mental Health Act which was formulated in the year 1912, stands modified recently since 1987 and is of utmost importance for understanding the forensic psychiatry subject as discussed briefly below.[1-6]

The recognition of existence of mental disorders can be traced to ancient times.[1,5,8] Certain reliable facts that may be considered here are:

- Hippocrates *(466-375 BC)* taught that the brain is the seat of the mind
- The early *Roman law, the Justinian Code,* treated insanity as a special entity.

It has become an established branch of medicine, only for little over a century. The terminology of *noncomposmentis* or *unsoundness of mind* covers all disorders of the mind. The *Indian Penal Code* employs the term *unsoundness of mind* while referring to *insanity.* Mental disorders are mostly a specialist's province. The unique feature that distinguishes *Homo sapiens* from the other creatures that inhibit the planet earth is the degree of development of those higher nervous system functions that humans subsume under the construct of mind. The human mind has been fascinated by countless observations throughout recorded history. However, every medical practitioner is expected to be well conversant with the application of knowledge of *psychiatry* in *legal procedure*.

MENTAL HEALTH ACT, 1987[1-5]

The Mental Health Act (MHA) 1987 (22nd May, 1987), is an Act to consolidate and amend the law relating to the treatment and care of mentally ill persons, to make better provision with respect to their property and affairs and for matters connected therewith or incidental thereto. This was enacted all over India in every Indian state and its union territories, since 1st April 1993. This replaced the Indian Lunacy Act, 1912, which had earlier replaced the Indian Lunatic Asylum Act of 1958.

It is considered necessary:

- To regulate admission to psychiatric hospitals or psychiatric nursing homes of mentally ill-persons who do not have sufficient understanding to seek treatment on a voluntary basis, and to protect the rights of such persons while being detained;
- To protect society from the presence of mentally ill persons who have become or might become a danger or nuisance to others;
- To protect citizens from being detained in psychiatric hospitals or psychiatric nursing homes without sufficient cause;
- To regulate responsibility for maintenance charges of mentally ill persons who are admitted to psychiatric hospitals or psychiatric nursing homes;
- To provide facilities for establishing guardianship or custody of mentally ill persons who are incapable of managing their own affairs;
- To provide for the establishment of Central Authority and State Authorities for Mental Health Services;
- To regulate the powers of the Government for establishing, licensing and controlling psychiatric hospitals and psychiatric nursing homes for mentally ill persons;
- To provide for legal aid to the mentally ill persons at State expense in certain cases.

The main object of the bill is to implement the aforesaid proposals. Mental Health Act is having 10 chapters comprising of 98 sections.

Chapter I

Deals with various terminologies and defines them. It also provides certain newer terminologies, which replace the older ones and are enumerated in Table 28.1. Thus, the Act has defined the terminologies of medical officer, medical officer in-charge, medical practitioner, mentally ill person, psychiatric hospital or nursing home, psychiatrist, reception order, relative, etc. as:

Medical officer: refers to a Gazetted Medical Officer in government service appointed by State Government to be a Medical Officer for purpose of this Act.

Medical officer in-charge: is a Medical Officer who for the time being is in-charge of a psychiatric hospital or nursing home.

Medical practitioner: refers to a person with recognised medical qualification under the provisions of the Act.

Mentally ill person: is a person suffering from mental disorders other than mental retardation, needing treatment.

Mentally ill prisoner: is a mentally ill person, ordered for detention in a psychiatric hospital, jail or other safe custody.

Psychiatric hospital or nursing home: is a hospital for the mentally ill persons, maintained by the Government or private party with facilities for outpatient treatment and registered with appropriate Licensing authority. Admitting a mentally ill to a general nursing home is an offence. Such patient is to be admitted to psychiatric hospital.

Psychiatrist: refers to a medical practitioner possessing a postgraduate degree or diploma in psychiatry recognised by IMC declared by State Government for the purpose of this Act.

Table 28.1: Showing change in terminologies according to the Mental Health Act, 1987

Outmoded terms	*New terms*
Asylum	Psychiatric hospital or nursing home
Lunatic	Mentally ill person
Criminal lunatic	Mentally ill prisoner

Reception order: refers to an order for admission and detention of a mentally ill person in a psychiatric hospital or nursing home.

Relative: includes any person related to a mentally ill person by blood, marriage or adoption.

Chapter II

Provides the procedures for establishment of mental Health Authorities at Centre and State.

Chapter III

Lays down the guidelines for establishment and maintenance of psychiatric hospitals and nursing homes. Accordingly this constitutes a *Licensing Authority* who will process applications for licenses and arranges for inspection of such psychiatric hospitals or nursing homes regularly, renewing the license every five years.

Chapter IV

Provides the procedures of admission and detention of a mentally ill patient in a psychiatric hospital or nursing home. In other words it deals with methods of *restraint of the insane/ mentally ill* person (refer below).

Chapter V

Provides formalities for inspection, discharge, leave of absence and removal of mentally ill persons.

Chapter VI

Provides methods of judicial inquisition regarding alleged mentally ill persons possessing property, and how such property be managed. If the court feels that the mentally ill is incapable of looking after himself or his property, an order can be issued appointing a guardian. However, if the court is convinced that the person can look after himself, but is unable to manage the property by him, a manager can be appointed.

Chapter VII

Provides procedures of protection of human rights of the mentally ill persons.

Chapter IX

Provides the penalties for infringement of guidelines.

Chapter X

Deals with miscellaneous matters under the Mental Health Act.

CURRENT CONCEPT

Current concept recommends the following:

- A mentally ill person should be treated like any other sick person without any stigma attached to such illness
- The law assumes that a person who is mentally ill is not responsible for his or her actions and therefore, if such a person commits a crime, he or she is not to be punished for it.
- The law presumes that every person is mentally sound, unless he or she is proved mentally disordered.

Legal term mental illness as used in Mental Health Act, 1987, is not defined, but includes psychoses, neurotic disorders and various organic disorders.

SYMPTOMS OF MENTAL ILLNESS

Considering the psychiatry as an independent subject, it is better to get acquainted with the various common psychiatric terminologies, which depicts the symptoms of mental illness. Knowledge about each of these is essential in order to understand their medicolegal significances also. *Glossary* below highlights these terminologies in an alphabetical order.$^{1-8}$

Affect

Affect means the outward manifestation of person's feelings, emotions, tone or mood.

Abreaction

Abreaction is a process of bringing to conscious awareness, previously suppressed unconscious conflicts and emotions. Such a release (catharsis) can have therapeutic value.

Aphasia

The loss of ability to express meaning by the use of speech or writing (motor aphasia), or to understand spoken or written languages (sensory/auditory aphasia).

Cognition

This refers to higher mental functions, e.g. memory, intelligence, concentration, orientation, etc.

Confabulation

Confabulation means purely imaginary events or fabrications that fill the gap of pathological loss of memory. In other words, it comprises of false memory that the patient believes to be true.

Delirium

Definition: Delirium is defined as an acute confusional state.

Causes: Drugs or alcohol intoxication or withdrawal head injury, high fever, stress, etc.

Clinical features: Clinically the patient may present with clouding of consciousness, disorientation, inco-ordination, and getting abnormal experiences such as hallucinations, delusions, illusions, etc. Impulsive acts such as suicide, homicide, etc.

Medicolegal Importance

- It may last for a few hours, days or weeks and may end with varying degree of recovery
- As per Section 84 of IPC—A person is not punishable, if he or she commits any offence in delirium.

Delusion (Disorder of Thought)

Definition

Delusion is defined as a false, but firm belief in something that is not a fact.

Classification

Hypochondriacal delusion: Person feels that something is wrong in his or her body, though he or she is healthy.

Delusion of poverty: Person thinks he or she is poor/pauper, though he or she is rich.

Nihilistic delusion: Person declares that he or she does not exist and the world also has no existence, etc.

Delusion of grandeur: Person imagines that he is rich/and famous, wherein he is actually poor/and inconsequential.

Delusion of persecution (paranoid delusion): Person thinks that his or her nearest and dearest relatives are trying to poison or kill him or her.

Delusion of reference: Person believes that people, things or events happening around him or her are referred to him or her in a special or indirect way.

Delusion of influence (control): Person feels that he or she is controlled by an outside power, agency, radio, hypnotised telepathy, etc.

Delusion of infidelity: Person imagines that his/her spouse is unfaithful.

Delusion of self-accusation: Person keeps on blaming himself/herself on trivial incidents that happened in the past.

Erotomania (Clerambault-Kandinsky Complex): Common among the females, in which she is convinced that a particular individual, especially her superior officer or her employer, etc is in love with her.

Pseudologia phantastica: A variation of this is the *Munchausen's syndrome*, in which the person is convinced that he/she is seriously ill, and visits doctor-to-doctor, hospital-to-hospital in a vain attempt to diagnose the non-existing illness.

Bizarre delusion: is an outrageous delusion, which can take various forms.

Medicolegal Importance

It is not an isolated disorder but only indicates a deep-seated mental disorder such as schizoma. For this reason the patient cannot be held fully responsible for antisocial acts. It can affect the conduct and action of the patients, which may lead to the commission of suicide or homicide and suicide or other crimes.

DÉJÀ VU

A sense of familiarity with unfamiliar surroundings.

Disorientation

Disorientation is an impairment of the understanding of temporal, spatial or personal relationship, e.g. data given by such patients about time, place, people, etc., will be totally wrong.

Fugue

Definition: Fugue is defined as a state of disturbed consciousness with which a patient performs some acts. Though he or she looks apparently normal while doing the act, on recovery he has no recollection of events.

Causes: Dissociative disorders (hysteria), epilepsy, etc.

Medicolegal importance: It may lead to some unpleasant situations.

Hallucination (Disorder of Perception)

Definition: Hallucination is defined as a false perception without sensory stimulus.

Clinical features: Clinically the patient will see, hear, smell, taste and touch things, which are not there in reality.

Causes

- Altered state of consciousness (organic psychosis)
- Schizophrenia, epilepsy, drugs,
- Depressive disorders, etc.

Classification/Types

- **Visual hallucinations**: (more common) Person imagines that a lion or a tiger, etc attack him or her when none of them exists in front of him or her.
- **Auditory hallucinations**: Person hears voices or imagines that another person is speaking to him or her when no one is present.
- **Olfactory hallucinations**: Person smells pleasant or unpleasant odors when nothing exists in reality.
- **Gustatory hallucinations**: Person feels a good or bad taste in mouth, though no food is actually served.
- **Tactile hallucinations**: Person imagines that insects are crawling under his or her skin or bed when actually there are none.
- **Trichotillomania**: Person has an irresistible urge to pluck his/her own hairs.

Medicolegal importance: Hallucinating patients may commit some violent acts.

Illusion (Disturbance of Perception)

Definition: Illusion is defined as a false interpretation of an external object or stimulus, which has a real existence of its own, e.g. mistaking a stick for a snake.

Causes: Aging, the bereaved, delirious state.

Medicolegal importance: It is not indicative of insanity.

Insight

It is awareness of ones own mental condition, characterised by significant basic changes in the future behavior and personality.

Intelligence Quotient (IQ)

Intelligence quotient means the intellectual capacity of an individual in relation to his or her chronological age. It is expressed as a percentage.

Average or normal IQ for an adult of maximum lower age limit of 16 years is 90 to 110 per cent.

Lucid Interval

Definition: Lucid interval is defined as a period in the course of mental illness during which there is a complete cessation of symptoms of insanity, and the person is considered perfectly normal mentally.

Causes: Commonly seen in depressive mania and head injury (extradural hemorrhage).

Medicolegal Importance

During this period an insane person can:

- Make a will, which becomes valid
- Give evidence in court, validly.

In criminal cases, it is difficult to decide if some mental aberration was there or not at the time of the commission of a crime. Thus, it is advisable to regard the person as insane.

Mood

It is the pervasive emotion or feeling, which is sustained.

Neurosis

It is an emotional disorder in which the patient does not lose touch with reality.

Obsession (Disorder of Posession of Thought)

Person will have a symptom of a single idea, thought, or emotion entertained constantly and continuously, which persists inspite of recognising it as irrational and all efforts being made to drive it from the mind.

Panic

It is an acute, intense, overwhelming episode of anxiety associated with feelings of impeding doom.

Phobia

It is an excessive or irrational fear of an object, situation, or activity.

Psychopath (Sociopath)

Psychopath is a personality disorder, wherein the person is neither mentally ill nor defective, but does not conform to be normal due to failure to adopt normal ethical standards of behavior in the society. Thus the person is normal, but has deep-rooted abnormalities of personality, which he is unable to confirm to conventional standards of behavior. Often he has criminal behaviour, without any guilt feeling or remorse.

Medicolegal Importance

Persons may:
- Be abnormally aggressive, asocial, antisocial, submissive, demanding, mistrustful and criminal.
- Give a childhood history of similar behavior.
- Have normal IQ or even high IQ and be a genius.
- Be charming and impress people externally but deceive for long-time indulging in fraud, theft, deception, assault, etc.
- Show—temperamental outbursts/verbal and physical attacks without provocation, etc. with which only he or she feels better. This is called "*short circuit reactions*".

Psychosis

Psychosis is a mental disorder, which is severe and characterised by withdrawal from reality and living as if in another world, a world of fantasy with delusions and hallucinations. There is deterioration in personality and progressive loss of contact with reality.

Clinical features: Patient accepts all symptoms as real, reconstructs an environment of his or her own with which he or she tries to recreate the world and claims to hear strange voices, etc, and attracts, followers or disciples.

Classification

Psychosis could be of two types:
- *Organic psychosis:* has physical causes and could be acute as with high fever, delirium, etc. or chronic as with dementia.
- *Functional psychosis:* as with pathological variation of mood.

Examples

The Psychotic Killer – Such person is incapable of knowing the nature of his act or his judgment is faulty due to delusion and hallucinations. Murders are common in depression. Schizophrenics commit murders due to delusions of persecution. Morbid jealousy associated with alcoholism may lead to murder of spouse due to delusion of infidelity. Rarely maniacs and hypomaniacs commit murder due to delusions.

The Psychopathic Killer – The killing may be unintentional due to loss of control. Over-controlled murderer is one who has high-level control over his aggression, but commits murder due to an explosive response. After the aggressive act, he returns to his rigidly controlled behaviour. In some, normal emotional response may be almost absent.

Stupor

Stupor is a state of akinesis and mutism, with complete suppression of speech, movement and action with no disturbance of consciousness. Commonly seen in:
- Schizophrenia
- Depression
- Hysteria, epilepsy, etc.

Trance

Trance is a state of altered consciousness often with absence of voluntary movement or automation as in hypnotism/epilepsy.

Twilight State (Psychomotor Automatism)

Twilight state is a state of diminished awareness of acts of relatively short duration; the act performed during the state leaves little or no subsequent memory. Commonly seen in hysteria, epilepsy, etc.

Undue Influence

Undue influence is defined as physical or mental pressure of such degree that a person is deprived of his or her privilege to exercise his free will, e.g. a son refusing to give his father a painkiller to relieve the pain of surgical amputation unless he signs the will bequeathing all his property to his name.

DISORDERS OF THE MIND AND BEHAVIOUR

Disorders of the mind and behavior are of several types. Aetiology and classification of the same are discussed briefly.

Aetiology

Various causative factors which have specific relationship with disorders of mind and behavior are enumerated as follows:
- **Heredity**: For example, huntington chorea, amaurotic family idiocy, etc.
- **Environmental factors**: For example, faulty parental attitude and lack of mental hygiene.
- **Psychogenic**: For example, unsuccessfully repressed mental conflicts.
- **Precipitations**: For example, financial and business, frustrations and disappointments in sexual affairs, death of a close relative, etc.
- **Organic**: For example, head injury, atherosclerosis, senile degeneration, myxoedema, pernicious anemia, etc.
- **Unknown**: No cause or exact etiological factors can be identified for several mental disorders.

Classification

World Health Organisation (WHO) in its International classification of diseases, injuries and causes of death 10th edition (ICD-10), chapter F, provides a detailed classification of psychotic disorders. International Classification of Disease, 10th revision ICD – 10, Chapter F. The variety of disorders codified from F00 – F99 in the list, highlight the recent developments in the psychiatry specialty.[9]
- F00 Alzheimer's disease (G30)
- F01 Vascular dementia
- F03 Unspecified dementia
- F04 Organic amnesic syndrome, not induced by alcohol and other psychoactive substances
- F05 Delirium, not induced by alcohol and other psychoactive substances

- F06 Other mental disorders due to brain damage and dysfunction and to physical disease
- F07 Personality and behavioral disorders due to brain disease, damage, and dysfunction
- F09 Unspecified organic or symptomatic mental disorder
- F10 Mental and behavioural disorders due to use of alcohol
- F11 Mental and behavioural disorders due to use of opioids
- F12 Mental and behavioural disorders due to use of cannabinoids
- F13 Mental and behavioural disorders due to use of sedatives or hypnotics
- F14 Mental and behavioural disorders due to use of cocaine
- F15 Mental and behavioural disorders due to use of other stimulants, including caffeine
- F16 Mental and behavioural disorders due to use of hallucinogens
- F17 Mental and behavioural disorders due to use of tobacco
- F18 Mental and behavioural disorders due to use of volatile solvents
- F19 Mental and behavioural disorders due to multiple-drug use and use of other psychoactive substances
- F20 Schizophrenia
- F20.0 Paranoid schizophrenia
- F20.1 Hebephrenic schizophrenia
- F20.2 Catatonic schizophrenia
- F20.3 Undifferentiated schizophrenia
- F20.4 Postschizophrenic depression
- F20.5 Residual schizophrenia
- F20.6 Simple schizophrenia
- F20.8 Other schizophrenia
- F20.9 Schizophrenia, unspecified
- F21 Schizotypal disorder
- F24 Induced delusional disorder
- F25 Schizoaffective disorders
- F28 Other nonorganic psychotic disorders
- F30 Manic episode
- F31 Bipolar affective disorder
- F32 Depressive episode
- F33 Recurrent depressive disorder
- F34 Persistent mood (affective) disorders
- F38 Other mood (affective) disorders
- F40 Phobic anxiety disorders
- F41 Other anxiety disorders
- F42 Obsessive-compulsive disorder
- F43 Reaction to severe stress, and adjustment disorders
- F44 Dissociative (conversion) disorders
- F45 Somatoform disorders
- F48 Other neurotic disorders
- F50 Eating disorders
- F51 Nonorganic sleep disorders
- F52 Sexual dysfunction, not caused by organic disorder or disease
- F53 Mental and behavioral disorders associated with the puerperium, not elsewhere classified
- F54 Psychological and behavioral factors associated with disorders or diseases classified elsewhere
- F55 Abuse of non-dependence-producing substances
- F59 Unspecified behavioral syndromes associated with physiological disturbances and physical factors
- F60 Specific personality disorders
- F61 Mixed and other personality disorders
- F62 Enduring personality changes, not attributable to brain damage and disease
- F63 Habit and impulse disorders
- F64 Gender identity disorders
- F65 Disorders of sexual preference
- F66 Psychological and behavioral disorders associated with sexual development and orientation
- F68 Other disorders of adult personality and behavior
- F69 Unspecified disorder of adult personality and behavior
- F70 Mild mental retardation
- F71 Moderate mental retardation
- F72 Severe mental retardation
- F73 Profound mental retardation
- F78 Other mental retardation
- F79 Unspecified mental retardation
- F80 Specific developmental disorders of speech and language
- F81 Specific developmental disorders of scholastic skills
- F82 Specific developmental disorders of motor function
- F83 Mixed specific developmental disorders
- F84 Pervasive developmental disorders
- F88 Other disorders of psychological development
- F89 Unspecified disorders of psychological development
- F90 Hyperkinetic disorders
- F91 Conduct disorders
- F92 Mixed disorders of conduct and emotions
- F93 Emotional disorders with onset specific to childhood
- F94 Disorders of social functioning with onset specific to childhood and adolescence
- F95 Tic disorders
- F98 Other behavioral and emotional disorders with onset usually occurring in childhood and adolescence
- F99 Mental disorder, not otherwise specified

Most Commonly Accepted Classification

The classification of psychiatric disorders presented below is the one, which is mostly accepted all over the world.[9]

I. Psychosis
  A. Organic psychoses
    - Dementia – Senile and presenile
    - Drug-induced psychosis
    - Confusional state psychosis and
    - Psychosis following epilepsy, pregnancy, childbirth, trauma, and general disease.
  B. Functional psychoses
    - Schizophrenia – Simple, Hebephrenic, catatonic, paranoid and other uneffective.
    - Paranoid status
    - Affective disorders – Involuntary, melancholia, mania, depression, paranoid status, other atypical forms
II. Neurotic disorders
III. Personality disorders
  - Sociopath
IV. Sexual deviations
V. Mental retardation

Each one in the second classification is discussed in detail below.

DEMENTIA

Dementia is a type of organic psychosis, wherein the mind, after reaching a certain stage of development, begins to deteriorate. Clinically onset is insidious/ progresses slowly/occurs Pari-Passu. Patient presents with decline of intellectual functions of memory, comprehension and reasoning ability.

CLASSIFICATION

Dementia could be of three types—organic, senile and dementia paralytica.

Organic Dementia

Causes – Localised or diffuse brain lesion

Clinical picture – Restless/irritable with delirium/ excitement/ depression, leading to loss of memory, confusion as to time and place and mistake as to identify and childishness.

Senile Dementia

Causes – Old age and cerebral arteriosclerosis

Types – Alzhemers dementia, vascular dementia (multi infarct dementia), dementia due to Parkinsonism and Huntington's disease.

Clinical picture – Progressive mental deterioration with loss in memory, childishness, perverted behaviour, and delusions of family neglect.

Other Types of Dementia

These include – Crenty field Jacob's disease, Pick's disease, dementia due to head injury, anaemia, hyperthyroidism and infective (Syphilitic and HIV).

Drug-induced Psychoses

Drugs that can produce psychoses are:

- Alcohol
- Heroin
- Morphine
- Cannabis indicia
- Cocaine
- D-lysergic acid diethyl amide (LSD).

Alcoholic Psychoses

Mental disorders produced by alcohol are included under this and they are:

- Delirium tremens and acute confusions
- *Korsakoff's psychoses (confabulation)*
- Alcohol dementia
- Sexual jealousy and crime

Heroin/Morphine Psychoses

Both can produce progressive mental deterioration leading to:

- Loss of interest in environment
- Lowered intellectual efficiency
- No self-respect and trust
- May commit any crime to get the drug.

Cannabis Psychosis

In cannabis psychosis insanity can develop called hashish psychosis characterised by:

- Addiction
- Hallucination of sensuous type
- Delusion of grandeur/persecution
- Sexual jealousy and crime
- *Run-amok*: The person will kill first his enemy and then everyone else on his way and finally commandts suicide.

Cocaine Psychoses

These can be characterised by:

- Delusion of persecution
- Hallucinations of tactile type (cocaine bugs) and visual type.
- On prolonged use it can lead to both mental as well as physical deterioration.

LSD Psychoses

In LSD psychoses, a condition called bad trip can develop which is characterised by:

- Acute anxiety
- Depersonalisation
- Psychotic episode persists for months
- On prolonged use it can lead to permanent damage of brain cells.

Confusional State Psychoses

The patient presents with a state of confusion and various causes leading to this disorder are excess of physical/mental fatigue, acute infectious diseases, epilepsy, childbirth and other stresses of life, and trauma (head injury).

Clinical Manifestations

The clinical manifestations are characterised by:

- Restlessness, insomnia
- Confused ideas and mistaken identity
- Apprehensive and uncertain state
- Auditory/visual hallucinations

However, here recovery is a rule, and it may be also seen with acute delirium and stupor.

Psychosis Following Epilepsy

Epileptic automatism is often used as the defense in serious crimes when no other defense is available. An EEG has to be produced as an evidence of epilepsy. This has made epilepsy an important medicolegal issue. To know more about this, let us try to understand more about epilepsy here.

Epileptic convulsion characteristics: It includes several stages and they are as follows:

- *Pre-epileptic confusional state:* It may commence a few days before onset of the fit development of convulsions.
  - Mood irritability
  - Clouding of consciousness
  - Delusions and hallucinations under which, he/she may perform a crime. Normal state is regained after the cessation of convulsion.
- *Masked or psychomotor epilepsy:* Here the patient does not show any convulsions. He or she undergoes a mental disturbance, which replaces the convulsion completely, and these mental disturbances may bring about certain outrageous acts such as murder of a person who is usually a stranger. However, he or she also presents the facts that there will be:
  - No motive
  - No preparation (No preparedness)
  - No accomplices
  - No attempt to hide/conceal after crime
  - No attempt to escape after crime.
- *Post-epileptic automatism:* It commences after convulsions, for instance in petitmal epilepsy, patient will have lapse of consciousness, and performs acts without volition, which cannot be recollected after gaining consciousness, e.g. picking up things in shops and being arrested for theft.

SCHIZOPHRENIA (SCHIZOMA)

Schizophrenia, a type of functional psychosis, is said to be disorder of thought and disintegration of emotional stability (split between the thought and emotion-split personality), e.g. schizophrenic describing with interest and pleasure how he attacked and killed his victim.

Clinical Manifestations

Clinical manifestations can be described under two phases, namely an early phase and a late phase.

- *Early phase:* Usually presents with one of the following predominating at a given stage.
  - *Disorder of thought:* Misinterpretations of reality due to hallucination, illusions, delusions, etc., making him dwell in his own world.
  - *Disintegration of emotional stability:* change in behavior, e.g. withdrawn, depressive/violent, etc.
- *Late phase:* As the disease progresses, it makes the person withdrawn from environment with:
  - Lack of drive and ambitions
  - Gives up all hobbies
  - Loss of interest in friends
  - Stands indifferent to his or her surroundings, etc.

Classification

Schizophrenia usually presents in four subtypes and they are noticed as:

- *Schizophrenia simplex:* In schizophrenia simplex, the patient will show all clinical findings described above, but the symptoms that help in diagnosing the type are by observing his/her reactions to happenings of great importance as if they are not concerned with him/her.
- *Hebephrenic schizophrenia:* In hebephrenic schizophrenia the patient is very much disorganised (thought disintegration) by hallucinations, illusions and delusions, etc., that he or she may become impulsive and commit crimes. These impulses, though not common in early stages of the disease, might develop at any time. It may not be present in consciousness at the time of committing the crime.

  Case example: A patient heard strange voices instructing him to kill his own mother with a razor, but was afraid of doing it then. However, the next day, although no voices were heard, he went downstairs, got a razor and come and cut his mother's throat silently from behind and killed her.
- **Paranoid schizophrenia**: In this type, the patient will retain much of the original personality but will suffer from *distortion of thought* with *persecutory or grandiose delusions* and *hallucinations* to such an extent that he/she will pose a distorted view of the world around. *Othello Syndrome* is a dangerous state of *morbid jealousy*. It is infact a perfect example for paranoid schizophrenia. The patient will suffer from delusion of infidelity about his wife or mistress and assault, murder her or make an attempt to kill her.
- **Catatonic schizophrenia:** In this type, patients will have the mood disorders and characterised by rigidity stupor, agitation, bizarre posturing and repetitive imitation of movements or speech of other people. They are at risk of malnutrition, exhaustion and self injury.
- **Undifferentiated schizophrenia:** In this type, patient will have characteristic positive and negative symptoms of schizoma but do not meet the specific criteria of other subtypes.

Clinical Manifestations

Patient can present with any of the following:

- Delusion of reference
- Delusion of persecution
- Delusion of grandeur
- Paranoid delusions

All these may compel him to commit a crime. Since much of the original personality is preserved the crime he commits may not be the result of a sudden impulse, instead, is usually preceded by much complaining and planning and it may look, as if the legal test of insanity or Certificate of mental illness is not necessary.

Paranoid Status

Paranoid state is a type of functional psychosis. The patient presents purely with a variety of delusions with or without hallucinations, but there is no disturbance of mood and thinking, personality being preserved well.

Clinical Manifestations

This condition usually develops at middle age or even later and could be of two types:

- *Paranoia:* Age of onset – 25 to 40 years. This type is common in males. This is a rare illness of mind, wherein the patient develops gradual delusions of persecution of systematised nature, having a grave criminal association.
- *Paraphrenia:* Age of onset – 45 years or so. This is a rare type of mental illness, wherein the patient develops systematised delusions, ideas of reference and vivid hallucinations of auditory type, commonly.

Medicolegal Importance

- May create problems with neighbors due to delusion of persecution – calling police
- May suspect court or police in giving decisions of a case and making public announcements stating so, etc.

DIAGNOSIS OF MENTAL ILLNESS AND CERTIFICATION[1-9]

Diagnosing a frank case of mental illness is not at all difficult. Cases, which pose problems to a doctor or law in diagnosing, are those, which are in the early stage of mental illness. This indicates the need for a proper examination of every case of suspected mental illness and the scheme of examination includes the following:

Preliminary Particulars

Record the name, age, sex, marital status, education, occupation, income, address, religion, socioeconomic background, etc of the patient. Record all particulars of the accompanying person especially name, age, sex, and address, whether staying with the patient or not, etc. Also note down the statement of the accompanying person in addition to patient's statement. Note two identification marks such as moles, birthmarks, etc.

Presenting Complaints

Record the presenting complaints with particular reference to onset of present illness, duration, course, precipitating, aggravating, maintaining or relieving factors, etc.

History of Present Illness

Note down details on when was the last time patient appeared normal, evolution of symptoms in a chronological order. Record about details on any suicidal intentions/attempts, insomnia or disturbances of sleep, appetite, sexual functions, etc.

Past History

Note down any similar or other major/ minor illness and treatment received in the past. Elicit about alcoholism or other drug abuse in the past.

Family History

History of chorea, epilepsy or frank mental illness, etc, may be found among the parents or siblings of the patients, as most of the mental illnesses are hereditary in origin, seen in members of the same family.

Personal History

Elicit proper childhood history, play history, friends, puberty, menstrual and obstetrics history if female, history of any head injury, drug addiction, certain problems, which are unbearable such as domestic difficulties, emotional shock, frustration in life, love, sex, etc., may be elicited as precipitating factors in the onset of mental illness. Record also about the premorbid personality details such as the interpersonal relationships, attitude towards self and others, attitude towards work, religious beliefs and moral attitudes, mood particulars, habits, fantasy life, leisure activities, etc.

Physical Examination

Patient can present with deformities in the head or body, careless dressing style, abnormal walking manners, furred tongue, dry skin, moist palms and soles, rapid pulse, abnormally high body temperature, etc.

Examination of Mental Status/Conditions

Eliciting certain tests might prove the illness. Following are recommended tests.

- Memory test—ask him/her for the day, date, time, name and names of his relatives, etc. He or she will not be in a position to answer properly
- Power of reasoning and sound judgment—simple mathematical sums may be asked, which he or she will not be able to solve
- Handwriting—will not be clean and clear but shabby
- Speech—look for rate, quantity, volume, tone, flow and rhythm.
- Conduct before, during and after the incidents—the patient may not be able to show any response to any incidents taking place around or may be reacting in his or her own way unconnected to the incident or happening.
- Assessment of general appearance and behavior—general appearance can be assessed by the type of physique, build, height, weight, dress, hygiene, gait, posture, etc. Whether the patient is co-operative, hostile, evasive, combative, hostile, excited, shows involuntary movements, restlessness, catatonic signs, nature of eye contact, ability to establish rapport, whether hallucinating i.e. talking to himself, making odd gestures, smiling or crying with no proper reasons, etc can assess the behavior of the patient.
- Cognition assessment – is done by looking for consciousness, orientation, attention, concentration, abstract thinking, etc.
- Insight assessment – assess the degree of awareness and understanding of patient regarding his illness.
- Assessment on ability to judge – done by assessing the ability to understand the situations correctly and act appropriately.

Investigations

- Following tests are useful
- Complete medical toxicological screening tests
- Drug levels
- Electro-physiological tests
- Brain imaging tests
- Neuroendocrine tests
- Genetic tests
- Sexual disorder investigations, etc.

Diagnostic Formulation

After complete psychiatric assessment, diagnosis and differential diagnostic assessment is done along with a proper treatment plan.

Certification[1-6, 23]

A certification of a mentally ill by a doctor on single examination is not correct. Recommendations in issuing certificate for mental illnesses are as follows:

- Conduct three consecutive examinations on three occasions
- Describe the actual clinical picture in the certificate
- Give clear-cut reasons of diagnosis made
- Rule out the possibilities of *feigned insanity.*

Feigned Insanity

Feigned insanity is defined as a condition wherein a person is pretending to be insane.

Purpose

- To escape capital punishment by a criminal (criminal death sentences)
- To avoid business transactions or deeds
- To quit service in military jobs.

Medicolegal Importance

- It is a most responsible duty of the doctor to detect and report feigned insanity
- Observation is a must for a minimum of 10 days (may be extended with permission of magistrate).

Distinguishing features of feigned insanity Several features suggestive of feigned insanity are presented below and Table 28.2 presents the differences between true and feigned insanity.

- Onset sudden (with some motive)
- Acts as insane only when observed
- Symptoms are not of one type of insanity
- Pretending insanity can lead to exhaustion
- A malingerer as a rule is not dirty or filthy in habits. Usually keeps clean, eats well, etc.
- A malingerer resents frequent examinations
- It is impossible to feign insomnia for a long time
- Feigned *deafness* and *mutism.*

RESTRAINT OF MENTALLY ILL

Restraint of the mentally ill or insane is defined as keeping a dangerous insane person under lawful restraint in a mental hospital. Mental Health Act, 1987 amended the law relating to the treatment and care of mentally ill person, to make better provision with respect to the property and affairs held by him/her. Basically there are *two types* of restraints and they are *immediate restraint* and *admission to a psychiatric hospital.*[1-6]

Immediate Restraint

In immediate restraint the patient needs to be restrained immediately. The following can be the indications to this:

- When a person develops profound mental incapacity turning gravely dangerous to himself or others
- Delirium due to disease
- Delirium tremens.

Table 28.2: Differences between true and false insanity

Feature	*True insanity*	*Feigned insanity*
Onset	Gradual and insidious	Abrupt and dramatic
Motive	Absent	Present (Commission of offence)
Precipitating Factor	May be present-(stress, financial loss, bereavement)	Absent
Symptomatology	Conforms to a particular type of psychiatric disorder	Usually does not conform to any known type, and often exaggerated. No symptoms when not under observation
Facial expression	Listless, vacant and fixed	Frequent changes, exaggerated grimaces
Insomnia	Present often	Cannot pretend insomnia for more than a night or so
Personal hygiene	Do not pay attention. May be filthy	Not so
Exertion	Can withstand prolonged fatigue, hunger	Cannot withstand prolonged fatigue, hunger for more than 1-2 days
Frequent examination	Will not mind detected of feigning	Resents, as he is afraid of getting

Methods: Safely locking in a room, with the consent of the guardians or others. Consent is not necessary if there is no time to obtain it. However, he or she should release the person whenever he or she becomes no more dangerous.

ADMISSION TO PSYCHIATRIC HOSPITAL

Methods Admission to psychiatric hospital includes several accepted methods as approved by law. They are as follows:

- Voluntary or direct restraint
- Reception order on petition
- Reception order other than on petition
- Reception after judicial inquisition
- Reception of mentally ill criminal
- Reception of the escaped mentally ill.

Voluntary or Direct Restraint

Here the mentally unsound person submits a written application directly to the officer in-charge of the hospital for admission and treatment. However, the application must be reviewed and applicant must get the consent of the two appointed visitors of the hospital, who should confirm that the applicant really needs care and treatment as in-patient of the hospital.

Reception Order on Petition

Here the insane is admitted to the mental hospital only if the following formalities are fulfilled:

- **Petition**: It is an application to the magistrate in a prescribed form by a relative or a friend, who is taking care of the patient at least for a period of 14 days prior to date of petition writing.
- **Other requirements**: Along with the petition there are certain other requirements, which need to be submitted for a favourable action, and they are as follows.
  - A medical certificate from registered Medical practitioner stating that patient needs mental hospitalisation for treatment
  - A certificate from a gazetted medical officer who has examined the patient within 7 days prior to issuing the certificate specifying the need for hospitalisation
  - A doctor's certificate mentioning physical fitness of the patient to travel
- **Magistrate's order of reception**: Magistrate shall issue the order of reception and if there is a need he or she may personally come and examine the patient. This order holds good for 30 days.

Reception Order other than on Petition

Following are the indications and procedures.

- Wandering and dangerous lunatic
  - Police officer is authorised to arrest such patient and then produce before a magistrate.
  - Magistrate can issue reception order directly or in case of doubt he or she may send the patient for medical examination and then, issue reception order only if the patient is certified medically as mentally ill and dangerous.
- **Lunatic not cared for properly:** Police officer can produce a mentally ill person who is not properly cared for or cruelly treated by the relatives to the magistrate and a reception order can be sanctioned.

Reception after Judicial Inquisition

If a person possessing huge property turns mentally ill, the high court or district court may pass an order of inquisition and arrange for:

- Reception of the patient to a mental hospital
- Proper care of his or her property
- Arrangement for recovering necessary fees from the profits or income from property of the patient under court care.

Reception of Mentally Ill Criminal

It is one who is mentally ill and has committed a crime or become mentally ill after being imprisoned. For such a patient's reception presiding officer of the court issues order.

Reception of the Escaped Mentally Ill

Such a patient can be readmitted to mental hospital by a police officer or any officer or servant of the hospital.

Discharge of Mentally Ill from Psychiatric Hospital

Discharging the patient from a mental hospital is based on certain factors and they are:

- Recovery or cure
- Request of person who brought petition to discharge
- The order of an authority, on assuring proper care by the relatives
- A judicial requisition confirmed to be sane
- Intending for discharge by the voluntary or direct boarder.

RESPONSIBILITIES OF AN INSANE

Responsibilities legally mean liability of a sane individual for his actions or omissions, and punishment consequently by law.[1-9] Responsibilities of an insane person are dealt in two heading:

- Civil responsibilities
- Criminal responsibilities

Civil Responsibilities

Civil responsibilities are dealt in relation to:

- *Management of property*—Under Sections 50,51, 53,54 of Mental Health Act, 1987, the court can look after the property under guardianship by appointing a manager. However, this is done only when a friend or relative approaches the court and a judicial inquisition held and person is proved beyond doubt to be mentally unsound.
- *Contracts*—Under Section 12 of Indian Contract Act (ix) of 1872, any contract with a mentally ill is invalid. However, it is valid, if done during lucid interval. If the insanity develops later after the contract will not necessarily invalidate the contract. Also, if at the time of signing, the fact that one of the signatories to the contract was insane but was not known to the other party, then the court can declare the contract invalid.
- *Marriage and divorce*—Under Divorce Act of 1869, marriage is *null and void* if the person is mentally ill at the time of marriage. Here the marriage is legally considered to have never taken place, even if the consummation had been accomplished after the marriage. Nullity will not be sanctioned if one of the spouses turns mentally ill after the marriage. However, it may constitute sufficient ground for *divorce*.
- *Competency as a witness*—Under Section 118 of Indian Evidence Act, there is no competency for a mentally ill to be a witness unless he is in lucid interval. However, considering the person to be in lucid interval is the discretion of the judge.
- *Validity of consent under Section 90 of IPC*, the consent given by mentally ill is invalid for all purposes.
- *Testamentary capacity*—Under Indian Evidence Act, the will is usually contested and cannot be valid. Here a doctor has to prove that the testator is mentally sound (compos mentis).

Criminal Responsibilities

It can be dealt by knowing the following:

- Definition - In law Criminal responsibility is defined as *criminal liabilities* due for punishment.
  - Law presumes that every person is sane and accountable for his or her actions until the contrary is proved.
  - Law also assumes that every person who is proved to be insane is not responsible for his actions.
- Section 84, IPC—According to this, nothing is an offense which is done by a person, who at the time of doing it, by any reason, is suffering from unsoundness of mind and is incapable of knowing:
  - The nature of the act done or
  - What is done is either wrong or contrary to law. (McNaghten's Rule of English courts is an equivalent to Section—84, IPC).
- Legal test—Defined as the test for insanity, which precludes responsibility for the commission of a crime.
  - Requirements /The test should provide evidence of:
    - Presence of mental illness or defect
    - Presence of the same at the time of committing the crime
    - Disease making the person unable to assess his or her acts as wrong or contrary to law.
  - Medicolegal importance
    - All the three requirements are to be offered during trial in court as defense to the criminal charges.
    - It may prevent a criminal trying to escape from the prime punishment.
- Insanity and murder—It includes murder by a mentally ill who is not punishable by court (Section 84, IPC). Some examples are:
  - Psychotic murderer (schizophrenic committing a murder)
  - Sexual killer
  - Psychopathic killer (hit man-killer)
  - Jealous killer (Othello syndrome)
  - Alcoholic killer (infidelity)
- Insanity and other pleas—These include pleas concerned with
  - *Pleas on somnambulism:* It is a dissociated consciousness and crime committed during this is usually not willful or pre-planned. Hence not punishable
  - *Hypnosis and crime:* A hypnotised person cannot be tricked to do immoral or dishonest acts.
  - *Delirium (Hallucination and delusion):* A delirious person committing a crime is not punishable
  - *Drunkenness and criminal responsibility:* IPC Section 85 states that a person is not held responsible for any acts amounting to crime, under the influence of alcohol or such other intoxicative drugs, provided he was made to consume it without his knowledge
  - *Impulse:* Irresistible forces compelling to do certain conscious acts without motive or forethought (kleptomania, pyromania, etc) are not punishable if the impulsive disorder is due to some organic mental illnesses.

HISTORICAL ASPECTS OF CONCEPT OF CRIMINAL RESPONSIBILITY [10-19]

Being ruled by the Britishers for a long period, the law in India evolved from England and Wales. It has developed here for over many centuries and with it the concept of 'criminal responsibility'.

In order to be guilty of an offence, it is necessary for the prosecution to prove all the required elements of the charge to the required standard of proof. This standard is proof '*beyond all reasonable doubt*' [10], which means that the evidence is so strong against the defendant as to leave only the remotest of possibilities that he is not guilty.[11]

The prosecution must prove not only that the accused committed the act described (*the actus Reus*), but also had the required '*state of mind*' or '*mens rea*' (literally '*guilty mind*') for the offence in question.

Commentators have explained that the whole concept of the criminal responsibility of an individual is thus reflected by the doctrine of '*mens rea*', as the basis of liability for a crime rests on what the individual 'intended' to do. Crimes set out in statute law may prescribe the required 'mens rea' for a particular crime, in words such as '*intended*', '*recklessness*', '*knowingly*', or '*maliciously*', etc, although there has been a recent trend for offences of '*strict liability*', which require no '*guilty mind*' to apply to (mainly) administrative '*crimes*'.

The concept of '*responsibility*' is fundamental to our view of man as a '*free, intentional being*', and is said to form the basis of criminal codes and systems of punishment in most societies. One could argue that a person is responsible for something if he can answer questions about that thing, and this 'knowledge or control' is a necessary condition of most ordinary conceptions of responsibility – in terms of 'moral responsibility', one could not expect a person to be morally responsible for something that he has absolutely no control over.

However, not everybody is assumed to have the same level of control – for example, children and the mentally abnormal have historically been deemed to have less control over their actions than 'normal' adults, and certain acts may be excused on the grounds that they were the result of a '*mistake'* or '*accident'* or in response to '*provocation',* etc.

Researchers into the development of the concept of '*mens rea*', state that it is apparent from the writings of Henri de Bracton (legal scribe to Henry II) in the thirteenth century, and has survived almost unquestioned until the twentieth century. It was equated with '*moral guilt*', but it has been argued by Lady Wootton that 'mens rea' is becoming increasingly irrelevant to the concept of responsibility, which in itself has been 'withered away'.

Criminal responsibility or '*the ultimate issue before a court*' is a legal question, and as such must be determined by the court. According to one commentator writing in 1883, the meaning of responsibility is '*liability to punishment*', and if the criminal law does not determine who are to be punished, '*it does nothing*'.[16]

The emergence of medical witnesses (at first any medical witness, including physicians, surgeons and apothecaries), and in particular psychiatric witnesses began to challenge this assumption when dealing with the responsibility of mentally abnormal persons, albeit with varying degrees of success (psychiatric testimony of delusions was ignored in Fooks 1863), where the judge directed the jury, '*You are not to be deprived of the exercise of your common sense because a gentleman comes from London and tells you scientific sense'*

The Report of the Royal Commission on Capital Punishment (Cmnd 8932 1953 para 281),[22] however, took another view, 'A just and adequate doctrine of criminal responsibility cannot be founded on legal principles alone. Responsibility is a moral question; and there is no issue on which it is more important that the criminal law should be in close accord with the moral standards of the community. There can be no pre-established harmony between the criteria of moral and of criminal responsibility, but they ought to be made to approximate as nearly as possible'.

The question of whether a person should be held criminally responsible or not has been addressed by commentators, where they draw attention to the fact that modern standards of insanity (i.e. the Mc'Naghten Rules or test) are ambiguous in their meaning—do they require a person to know that their action was legally or morally wrong? They proposed three alternative meanings of '*wrongfulness'*:[14]

- The illegality standard, where the accused who lacks the capacity to know or appreciate that their acts violated the law is not held responsible for those actions,
- The subjective moral standard, where the person is not held responsible if they suffer from a disease of the mind that results in their belief that they were morally justified to carry out their actions, and
- The objective moral standard, where the person lacks the capacity to understand that society considers their actions to be morally wrong.

Thomas Aquinas said that it was impossible to make any kind of statement about a man's mind, and this was developed by Lady Wootton, applying this principle to attempts at proclaiming a man's criminal responsibility based on those statements.[12] The question of a person's capacity to resist temptation and of a person's responsibility is beyond mere mortals; they lie buried in his consciousness into which no human being can enter'. Other philosophical arguments against our ability to determine a person's responsibility include those based on Descartes (via John Locke) and Jeremy Bentham, i.e. that our actions are completely separate from our thoughts ('Cartesian dualism'). These can be countered by 'monist theories'.

Lady Wootton would also argue that there is a logical flaw in the concept of responsibility itself, in that there are no logical criteria upon which those who are responsible can be distinguished from those who are not responsible. She, and others, have attempted to support their argument by stating that even science cannot provide any answers to the questions about one's responsibility. However, this criticism is pointing out the fact that juries take a common sense approach to the question of a man's intentions, and his criminal responsibility, and they do so on a daily basis, without too many problems.

The philosophers then have a role in the criticism of law, but those required to implement it – the public in the form of the jury, do not rely on esoterical arguments about the moral philosophy of their role – they perform it utilising their normal human insight into man's behavior.

The Historical Treatment of the Mentally Abnormal Offender[18-21]

Very little is known about the treatment of the insane in early Indian criminal history. However, under Roman law the insane offender was treated with leniency, on the basis that the madness itself was sufficient punishment (*satis furore ipso punitur*). This was a morally acceptable solution to the problem of the mentally abnormal offender, based on the Greek moral philosophy (*particularly Aristotle*) and Hebrew law. Indeed, one of the earliest sources of the *'right-wrong test'* is found in the Book of Genesis. Bracton wrote in the thirteenth century, that the *'will to harm'* must be present for a crime, and without such a will, the madman was excused of responsibility.

Sir Edward Coke (1552-1634) argued in Beverley (1603) that the madman 'did not know what he did', and could not, therefore, have the necessary criminal intent.[18] This set the scene for 'insanity' to be judged in purely '*cognitive terms*' – did they know that what they were doing was wrong? (*The 'right-wrong' test*).

Only somebody who was 'totally insane', as opposed to 'partially insane' would benefit from this excuse of criminal responsibility, as stated by Sir Matthew Hale in the *History of the Pleas of the Crown (1736)*, and the 'understanding' of a madman was compared to that of a child, '…they have not the understanding, and act not as reasonable creatures, but their actions are in effect in the condition of brutes'.[20]

Hale's work had a great deal of influence on lawyers of the 18th and 19th Centuries, but there were to be a series of cause célebrès that had the effect of clarifying the common law position with regard to the responsibility of the 'madman'.[13]

R – v - Arnold (1724)[24]

Arnold suffered from delusions that Lord Onslow had bewitched him, and sent into his '*chamber devils and imps*', that had '*invaded his bosom such that he could not sleep*'. Onslow was considered by Arnold to be the cause of '*all troubles in the Nation*'. Despite these delusions, Arnold's defense was unsuccessful, probably because he did not display a 'total deprivation of reason', and he was hanged.

In the summing up by the Judge, not only was the madman compared to an infant, or a 'wild beast' (the '*wild-beast test*'), but an additional criterion for judging criminal responsibility appeared – that of being able to distinguish between 'good and evil'. This criterion was eventually developed by judges to become an independent criterion of insanity. Juries were now in a position to return a 'special verdict' of not guilty by reason of insanity, even where an offender had some sense of what he had done, but did not know that it was wrong.

R - v - Earl Ferrers (1760)[25]

Ferrers had shot the receiver of his estate, Johnson; after becoming convinced that he was 'conspiring with his enemies'. However, the shot was not immediately fatal, and Ferrers summoned medical assistance for him. At his trial for murder, he tried to claim that he acted on an 'irresistible impulse', but was unsuccessful. Only a 'total, permanent or temporary want of reason would acquit a prisoner'.

R – v - Hadfield (1800)[26]

James Hadfield (see below) suffered from a delusion that called for him to sacrifice his life for the salvation of the world. However, he did not wish to commit suicide (a crime of 'self murder' and a moral crime), and decided to commit regicide, and be executed.

On the 15th May 1800, Hadfield fired at George III at the Theatre Royal, Drury Lane, London. (Keeton 1961 p.17). He was tried for treason, and this charge allowed him to seek the services of Counsel (unlike Arnold and Ferrers before him) to present his case, examine and cross examine witnesses, and to sum up on his behalf. This level of legal representation was not allowed in trials other than treason until the Prisoner's Counsel Act 1836 was passed. His lawyer, the famous Erskine, discredited the traditional tests of insanity, complaining that under Coke's view of '*total deprivation*' of memory or understanding, it would be almost impossible for anybody to be found '*insane*'. Erskine raised the possibility that a person could '*know what he was about*', but not be able to resist a '*delusion*'. Hadfield was acquitted, but it is generally thought amongst historians that this was due to the oratory brilliance of Erskine, rather than a correct interpretation of the law regarding insanity at that time.

Another possibility is that the jury sympathised with Hadfield in that medical testimony (from Dr. John Monro, physician to Bethlem Hospital, and the first example of forensic psychiatric witnessing) had been heard that the probable source of his delusions was his horrendous war wounds, received at the hands of the French.

R – v - Bellingham (1812)[27]

Bellingham was a paranoid individual, who blamed the Government, and in particular the Prime Minister Sir Spencer Perceval, for his business problems. He shot and killed the popular Perceval. After the court accepted a strict interpretation of the common law on insanity, he was convicted and hanged within a matter of days of the shooting.

R - v - M'Naghten (1843)[28]

Daniel M'Naghten (see below) was a young schizophrenic / eccentric, was under the belief that the police, the Church of Rome, the Tories and the Prime Minister Sir Robert Peel, were persecuting him.

On the 20th January 1843 he shot and killed the private secretary to the Prime Minister, Sir Robert Peel, mistaking Drummond for Peel. The Prosecution Solicitor General, Sir William Webb-Follelett stated that although it was difficult to understand the motives behind some crimes, and in particular political assassinations, this fact did not excuse the assassin. However, M'Naghten was acquitted on the basis that he labored under a delusion, which prevented him from controlling his actions. Although he was removed to Bethlem Hospital, and then Broadmoor (and not simply released), the result of this trial gave rise to much public outrage, summed up by the Times (6/3/1843) who asked the question - where did sanity end and insanity begin?

Public disquiet was reflected by the Lord Chancellor, who initiated a debate in the House of Lords, which in turn led to several questions being put to the judges of the *M'Naghten case*, seeking to clarify the common law of insanity in England at that time.

The judges answers now form what is known as the '*M'Naghten Rules*', and form the basis of the '*test*' of insanity that the jury should be instructed to apply in each case of alleged insanity. The rules still provides the test of insanity in this country. Jurors are thus instructed that:

- Every man is presumed to be sane and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved to their satisfaction
- To establish an insanity defence, it must be clearly proved that, at the time of the committing of the act, the accused was laboring under '*such a defect of reason, from a disease of the mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing what was wrong*'. This is a purely cognitive test (subsequently reinforced by case law - Codere[29] and Windle,[30] and makes no mention of '*partial insanity*' or *delusions*. This has been the subject of many critics, and other jurisdictions including Queensland, Tazmania and the USA (in their Model Penal Code) have attempted to improve the test, by adding their own '*third limb*' – that of '*irresistible impulse*', or '*a lack of ability to control his actions*', etc. However, in the light of 19th Century psychiatric knowledge, some commentators are of the view that a cognitive test is the only logical and reliable criterion that could have been chosen for 'insanity'. Committees set up to examine insanity and crime, or capital punishment unanimously called for such a change to the test in England (including the Atkin Committee on Insanity and Crime –1923, the Select Committee on Capital Punishment – 1930 and the Royal Commission on Capital Punishment – the Gower Commission – 1953), but their recommendations in this regard were rejected - the Judiciary held a deep scepticism when it came to '*partial insanity*' and '*irresistible impulses*'. Indeed, Baroness Wootton argued in '*Social Science and Social Pathology (1959)* that an extension of the rules would

> be catastrophic – we would be left with 'no logically secure resting-place short of total abandonment of (criminal) responsibility'.[19-21]

After satisfying the rules, the accused was not at liberty to go on his way – he would be kept in custody 'until His Majesty's pleasure be known'. This was a direct result of the *Criminal Lunatics Act 1800*, enacted following Hadfield's acquittal [c], which did away with the simple acquittal or 'special verdict' of acquittal on the grounds of insanity. Although the new verdict appeared to be absolving the mentally abnormal offender of criminal responsibility, on the basis that they were as '*morally innocent as a man who had done harm by accident or self-defence*', it recognised that the dangers of treating him as innocent (i.e. not punishing him) were too great. Despite Queen Victoria's attempt at changing the '*insane'* verdict to '*guilty but insane*', under the provisions of the Trial of Lunatics Act 1883 (the monarch was the subject of many attempts at her life by madmen, who were subsequently acquitted), case law confirmed that this 'special verdict' of insanity was indeed an acquittal, but that it was a '*qualified'* rather than '*absolute*' acquittal.[31,32] The last word on the matter was made by the *Criminal Procedure (Insanity) Act 1964* which re-affirmed the common law position with respect to the effect of the 'special verdict'.

Diminished Responsibility

Diminished responsibility is a concept that had existed in Scottish law prior to its introduction in England and Wales by virtue of *Section 2* of the *Homicide Act 1957*. Under Scottish law, partial insanity had a mitigating effect on the punishment meted out as early as the 17th Century. However, it was not a 'defence' in its own right – merely a tool for mitigating the act.

The effect of the statutory defence was to alter the category of crime committed—from murder to manslaughter.

'Where a person kills, or is party to the killing of another, he shall not be convicted of murder but shall be convicted of manslaughter, if he was suffering from such abnormality of mind (whether arising from a condition of arrested or retarded development of mind or any inherent causes or induced by disease or injury) as substantially impaired his mental responsibility for his acts and omissions in doing or being a party to the killing'. (Section 2(1) Homicide Act 1957).

The wording of the defence drew on that seen in the Mental Deficiency Acts of 1913 and 1927, and gave rise to a very broad interpretation of 'abnormality of mind' and 'mental responsibility for his acts' in resulting case law.[33] If the defence was successful, the court were able to exercise a wide range of punishment options, ranging from '*life imprisonment'*, to commitment to a mental hospital, to absolute discharge. A consideration of the ability of the defendant to exercise will power and resist impulses (as well as cognitive aspects familiar to the existing common law of insanity) now contributed to a wider test of criminal responsibility. However, it did not give rise to the expected flood of convictions for manslaughter instead of murder, by offenders who would not previously have been considered 'insane', and some researchers explain this phenomenon by considering the proportions of offenders being found insane at various stages of the criminal law process (i.e. unfit to plead, acquitted on the grounds of insanity, etc) before and after the introduction of the new defence. They found that the overall proportion of those found to be 'mentally abnormal' did not change, but now that the defence of diminished responsibility was open to them (and now that capital punishment was carried out in only restricted circumstances (and suspended completely in 1965), the offender was less likely to plead insanity, and risk a life-times' incarceration, but could now plead guilty to manslaughter. What the introduction of this new defence did do was to bring the legal concept of 'insanity' and mental illness into the 20th Century, and more closely in line with contemporary thought.[19-21]

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Chapter

29

Forensic Radiology

Forensic radiology comprises the performance, interpretation, and reporting of those radiologic examinations and procedures that concern the courts and/or the law.

INTRODUCTION

Forensic radiology is an area of expertise in medical imaging utilising radiological techniques to aid physicians, pathologists and medicolegal experts in the matters associated to the law[1]. The forensic use of radiography has a long history indeed within months of the discovery of X-rays by Wilhelm Roentgen (Fig. 29.1) in November 1895[2]; a bullet lodged in the leg of a gunshot victim was located using X-rays and the obtained images used in the subsequent successful prosecution case for attempted murder.[1,2] Pathologists regularly use radiographic images during the course of autopsy to assist them in identification of foreign bodies or determination of death. Over the last 20 years, radiological techniques have become more sophisticated with the introduction of CT, MRI and ultrasound. These newer procedures are now being applied in the forensic environment requiring the active participation of medical imaging specialists (Radiologists).

SCOPE OF FORENSIC RADIOLOGY

The scope of applications of forensic radiology includes determination of identity, evaluation of injury and death, use in criminal and civil litigation, in administrative proceedings such as workman's compensation hearings, in medical education, and in research. Radiology plays an important role in forensic sciences. It is a visible and permanent record. Because it is permanent it is available for reinterpretation and revaluation at any given time. Identification data such as name, place and time of exposure are extremely significant and should be exposed as much as possible on the radiograph. Radiologic data are specialised in character, and so they require the skilled interpretation of a radiologist while giving evidence. A radiologic data should be presented in scientific terminology first and then the clarification done with non-medical terms. When presenting a radiograph in the court it is important to first establish how and under what circumstances the radiographs were made, where were they preserved and who had the possession of it. It is important to establish that the films neither ever left the possession of the radiologist nor could they have been replaced by replica. Radiograph is valuable in many occasions in Forensic Medicine. Here is an account of where radiography is important in different circumstances.

Fig. 29.1: Wilhelm Roentgen

Identification

The importance of radiology in identification of body was realised by Gray[3] when he examined mummies in the museums of Great Britain and other European countries. He found out that radiographic examination of these mummies was helpful in estimation of age and determination of sex.

X-ray examination of pelvis, skull, frontal bones and mandible may give definitive information about sex.[4] The pubic bone adjacent the pubic symphysis in pelvis will be rectangular in females and triangulate in males. Male vertebral bodies tend to be larger. Long bones of the females are thinner and smoother. Females have smaller supraorbital ridges, mastoid processes, and mandibular condyles. It has been demonstrated that in 88 per cent cases sex can be determined accurately from a radiologic study of skull. However dimorphism should be kept in mind. Radiological study of costal cartilages calcification pattern (Fig. 29.2) is recent advance in sex determination.[5,6]

Also, age can be determined from a radiograph.[7] Orthophantomogram (OPG) is an ideal evidence in determining age depending on tooth eruption (Figs 29.3A and B). It might also reveal tooth restoration particularly which can directly help establishing identity with great accuracy. Measuring diaphysial length and demonstrating ossification centres can estimate fetal length and age. Appearance of ossification centres and its fusion is a well established and accepted method of age determination approved by the court (Figs 29.4A to D). In infancy and childhood, the ossification centres present in the wrist radiograph are helpful. In adolescent, demonstration of epiphysial union

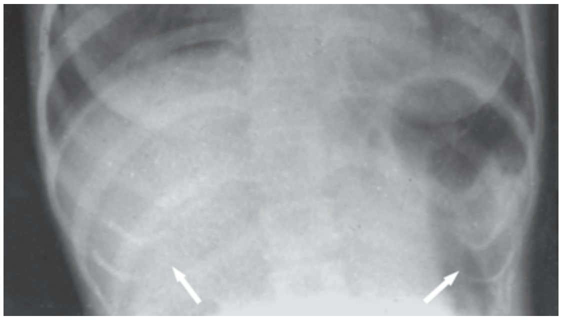


Fig. 29.2: Costal cartilage calcification pattern: Female type (arrows)

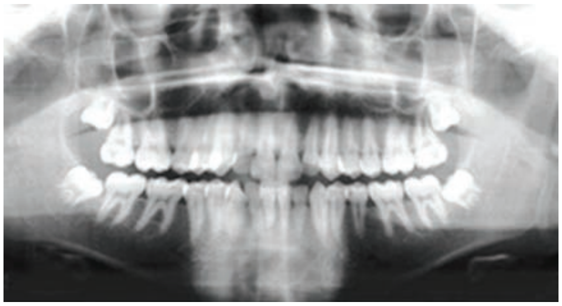


Fig. 29.3A: Orthophantomogram (OPG) of age <18 years

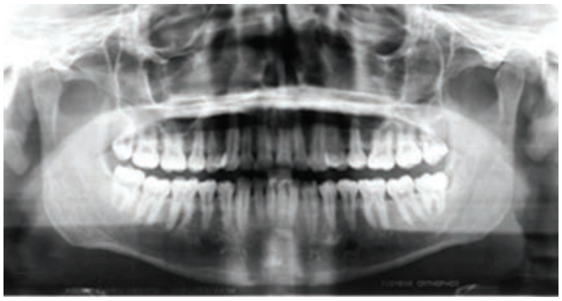


Fig. 29.3B: Orthophantomogram (OPG) of age >18 years

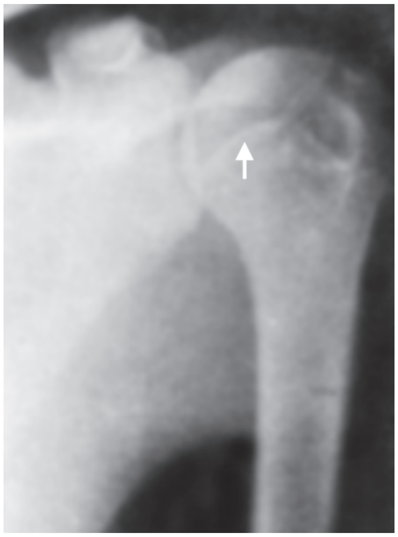


Fig. 29.4A: *X-ray shoulder joint:* Age just 18 years: as all ossification centres for upper end appeared and fused. *Epiphyseal scar* formed (arrow)

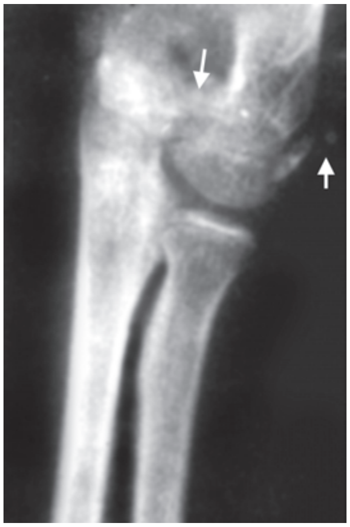


Fig. 29.4B: *X-ray elbow joint:* Age >11 years: as lat epicondyle centre appeared (arrow). <16 years as composite epiphysis of lower end humerus not formed

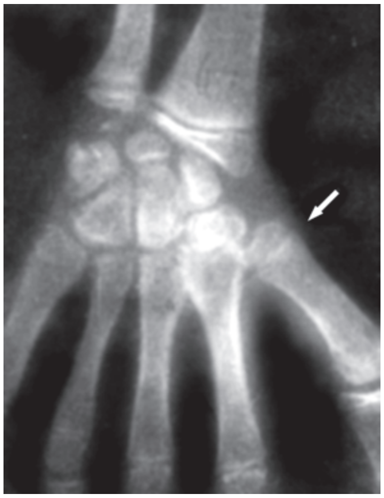


Fig. 29.4C: *X-ray wrist joint:* Age 10-17 years: as pisiform appeared and base of first metacarpal not fused

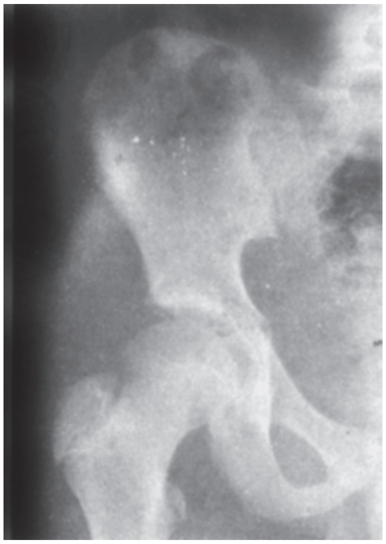


Fig. 29.4D: *X-ray hip joint:* Age >12 years as centre for lesser trochanter appeared; and <14 years as centre for iliac crest not appeared

at the wrist, base of the skull etc. helps to evaluate the age. X-ray can demonstrate calcification of laryngeal and costal cartilages in 5th decade of life. Calcification in trachea can be demonstrated.[7]

Radiology of wrist can also help in determination of age, from internal structure of the cancellous bone and cortical thickness of the head of the humerus.[5] Radiographic identification by studying frontal sinuses (Fig. 29.5) has been found very useful in mutilated bodies or burnt bodies.[6] Poole in 1931 pointed out that no two persons (not even identical twins) have the same profile of these air spaces. They appear in second years of life and increase in size for first 2 decades. For sinuses to be used, an antemortem anteroposterior skull radiograph must be available. The cadaver skull or head must be X-rayed in exactly the same orientation and degree of enlargement. Now superimposition technique of skull can also be employed to find the identity.[7]

Other radiological methods of comparing identity include matching of hand and wrist films, matching the profile and structure of first rib and clavicle. However, in general, it may be said that, wherever antemortem X-rays are available, especially of skull, thorax, hip or shoulder, then radiological comparisons of the dead material can be done and this can almost exclude an identity and thus on several circumstances identity is confirmed.

Anatomical matching can be done in a body if radiological evidence is present. *Superimposing* AP and lateral view of the skull can do it. Discrete abnormalities such as healing fractures, metal prostheses, bone disease or congenital defects in a radiograph may immediately indicate that the bone is unique and is helpful in establishing accurate identity of the deceased. Radiography has a special place in forensic odontology. When the radiograph is taken for frontal sinus visualisation the radiograph may show unexpected foreign bodies having unique dental importance, such as broken drill, broken roots or congenital abnormalities which may be matchable with clinical films or a note in the ante-mortem records. X-ray can disclose very specific morphological patterns of the individual, dental restorations, information on root canal treatment, buried root tips and jaw bone pattern, dental peculiarities such as gaps between teeth, a broken tooth, gold crowns, etc. may enable identifications easy when it matches with the antemortem dental radiographs of the deceased are well preserved by the dental practitioner. Forensic dentistry plays a major role in victim identification. DNA and dental identification of human remains depends on sufficient availability of ante mortem information, existence of sufficient postmortem material and a comparison or match between ante and postmortem details. Forensic odontology is a specialty with a specific training, and cannot simply be carried out by dentists without such training.[8]

Radiology and Trauma

Radiologic investigations often make significant contributions in the analysis of diagnosing trauma. While the gold standard of this investigation is a forensic pathology examination, numerous reports stress the important role of computed tomography in the postmortem evaluation of trauma victims[9] (Figs 29.6A and B). Bones may be injured, with fractures and haematoma can be detected. Joints may be injured with resultant *subluxation* or *dislocation*. Loose bodies or air may appear in these cases in the joint. Neurotropic joint abnormalities due to *diabetes* or *Tabes dorsalis* can be detected radiologically and help in establishing identity.

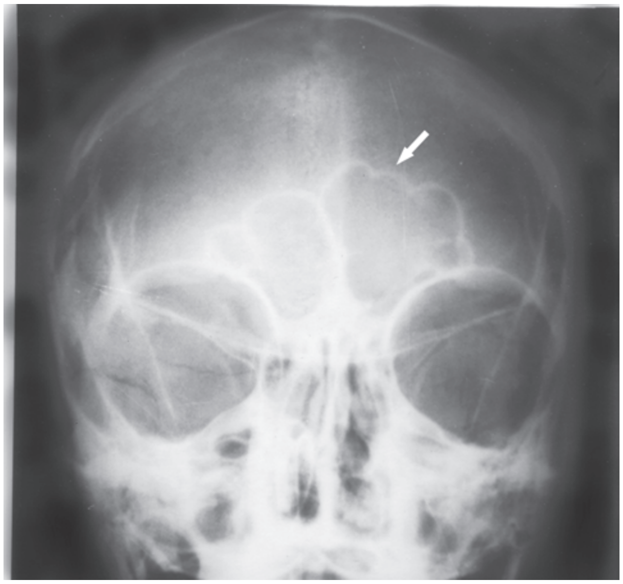


Fig. 29.5: *Frontal air sinus. Note* the unique profile that help establishing identity

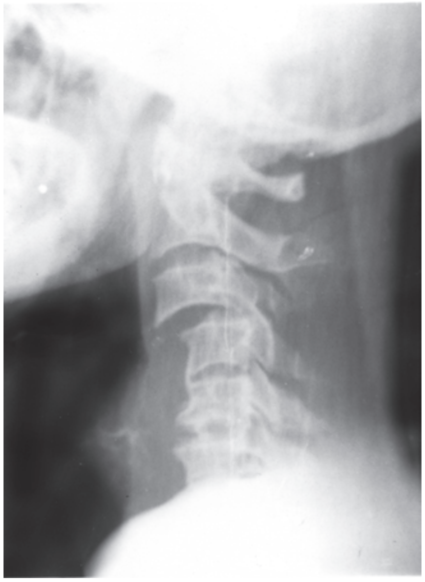


Fig. 29.6A: Fracture dislocation of 3rd and 4th cervical vertebrae may help in deciding cause of death

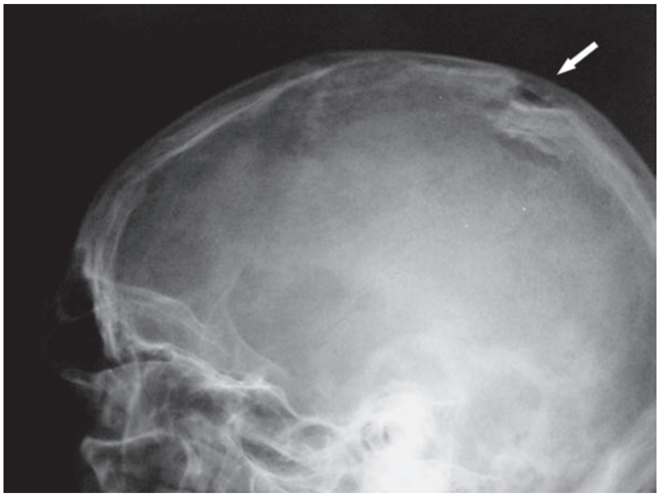


Fig. 29.6B: Depressed fracture skull (arrow)

Soft-tissue trauma may be manifested as swelling, gas within the soft tissue and at a later date by calcification. Laceration, denudations and defects may be detected on X-ray. Foreign bodies whose density is significantly different from that of surrounding soft tissues may be visualised radiologically.

The solid and hollow viscera, which are injured, can be detected on X-ray. Abnormal air accumulation in abdomen indicates a visceral injury. The presence of a *pneumothorax* demonstrated on X-ray chest indicates a lung injury. In suspected air embolism and barotraumas a radiological examination before autopsy is helpful.

Mutilated remains, especially those from mass disaster should be X-rayed, for example in fire victims where external damage makes dissection difficult. Where bombs or explosive devices are involved it leads to identification of any part of mechanism that may be embedded in the soft tissue. CT is a useful and complementary method to autopsy.[10]

Radiology and Battered Child

A distinguished pediatric radiologist Caffey in 1946 reported his observation of a common association of subdural haematoma and abnormal X-ray changes suggestive of trauma in the long bones. Later Selverman and Woolley opined that the trauma noted was largely fractures of the long bones in these immature victims wilfully inflicted by parents or siblings Dr. Henry Kempe named it '*battered child syndrome*'.

Radiographic examination of the entire skeleton prior to autopsy is required whenever child abuse is suspected. CT scan of the head is equally helpful in visualizing skull fractures (Fig. 29.7) among the victims of child abuse fatalities. Where the radiographic findings are not very obvious another X-ray after removing of the organs is helpful. Multiple skeletal injuries in different stages of healing are commonly encountered. They include separation of the metaphyses of long bones due to rough handling and violent shaking. This is observed more often in children under one year of age. Destruction of the adjacent end of the bone can usually be seen in about two weeks if they are not immobilised after the injury. Injuries of the metaphyses are most typical skeletal manifestation of child abuse. Another common finding radiologically is separation of the periosteum from the underlying bone. Resulting subperiosteal hemorrhage may be also apparent. Calcium deposits may be identified radiologically within 2 to 3 weeks. They are usually seen on arms and thighs. The help of a radiologist should always be taken who can interpret the age of the bony injuries from which treatment has been with held. Another common injury that is seen with child abuse is fracture of the posterior ribs, which may be visualised on the radiograph (Fig. 29.8). For a better visualisation, the soft tissue should be dissected and a radiograph be taken, which reveals the fractures better.

Radiology and Firearm Injuries

In case of a shotgun fire a radiograph can helps in locating the projectiles. Not only this, it can also give an idea about the dispersion pattern of the pellets. This can be applied to the estimation of the range of firing. X-ray is used for locating the bullet in the body (Figs 29.9A and B). The bullet track also can be identified if the bullet is jacketed when it leaves a trail of metallic particles from the jacket. X-ray is sometimes used to measure the calibre of the bullet, in a living person where surgical removal of the bullet is not indicated. The use of X-ray is of value in the case of skeletonised bodies where a perforating fracture is present in the bone. X-ray may reveal a bullet track through the bone by the presence of metal fragments along the track. Because the use of radiology in modern forensic medicine has been, until today, mostly restricted to conventional X-rays, which reduces a 3D body to a 2D projection, a detailed 3D documentation of a gunshot's wound ballistic effects was not possible.[11] Multi-slice computed tomography (MSCT) and magnetic resonance imaging (MRI) are technologies used for the documentation and analysis of gunshot wounds.[12] CT scans and MRI of the cerebral parenchyma revealed lanes of opaque bone and missile fragments along the course of the missile, which allowed recognition of the missile track in 3D reconstruction. Biometric reconstruction allowed easy determination of the angle of the missile track in all three planes.[13] With the spiral CT and MRI examinations and the subsequent 2D multi-planar reformation (MPR) and 3D shaded surface display (SSD) reconstruction, the entire gunshot-created complex skull fractures and brain injuries (such as wound channels and deeply-driven bone splinters) could be documented in complete and graphic detail.

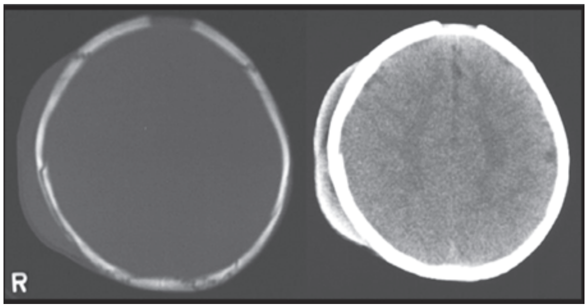


Fig. 29.7: CT scan showing skull fracture in a child abuse incident

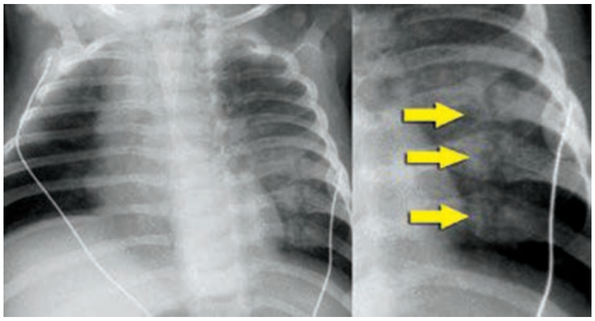


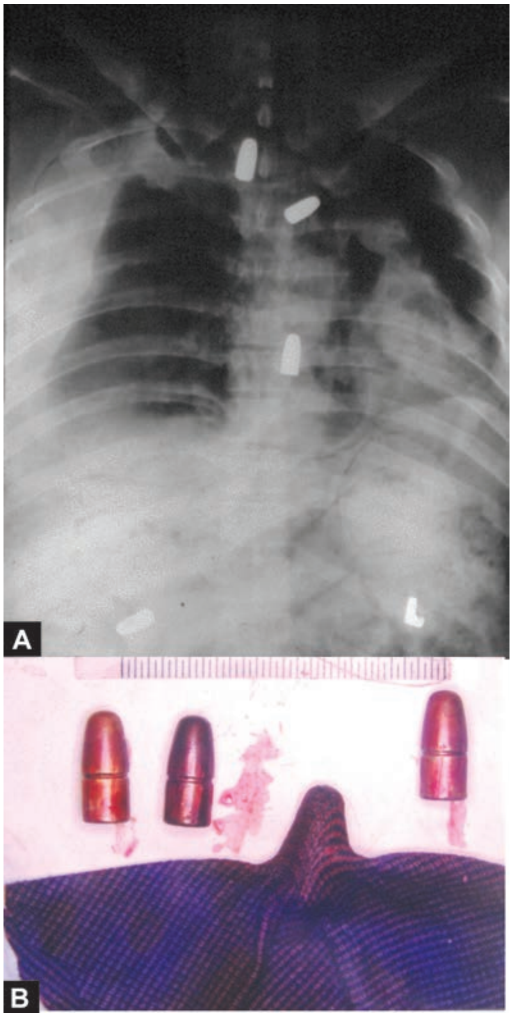
Fig. 29.8: Showing old posterior rib fractures of child abuse

Radiology and Asphyxial Death

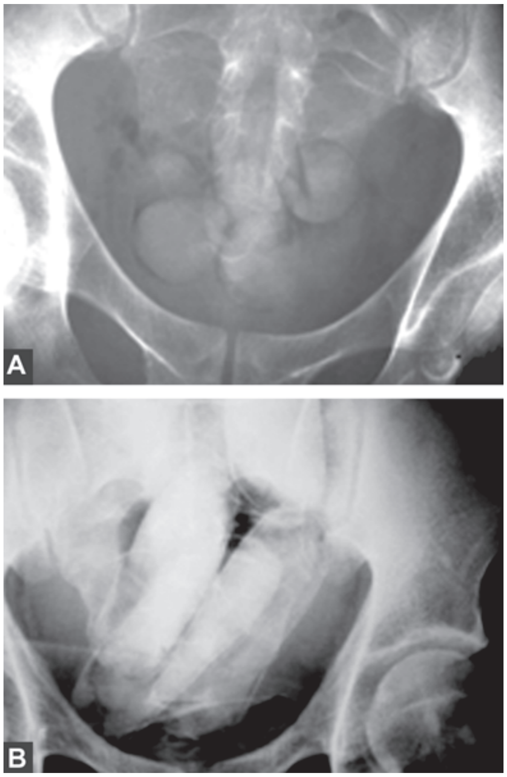
Radiography of the neck may be carried out before any dissection to determine the state of cervical spine and the laryngeal cartilages. An X-ray of the cervical spine in situ both antero posterior and lateral may reveal number of the transverse process of the atlas vertebrae which may be responsible for vertebral artery injury, which passes through it. A radiograph after dissection of the cervical spine reveals the fracture better.

Radiography and Smuggling

Radiologic evaluation may be useful in detection and confirmation of smuggling narcotics which may be enclosed in



Figs 29.9A and B: *Fire arm fatality case:* (A) Radiograph showing rifle fire arm bullets—three in the thoracic and one in the abdominal cavities respectively, (B) These bullets recovered at autopsy



Figs 29.10A and B: *Radiograph of the pelvis showing:* (A) Multiple round-shaped drug packets in rectal area, (B) Close-up view of the pelvic region showing multiple large sausages shaped drug packets in the rectosigmoid region (*Courtesy:* SS Kumar, A Deena, SN Raj and MA Kukkady: Diagnostic Radiographic Findings in Body Packers: A study of 15 cases in Kuwait. The Internet Journal of Radiology 2007;6(2)

plastic containers and swallowed (Read also *"Mini Packer's syndrome"* see page no. 664) (Figs 29.10A and B). Radiologic visualisation is sometimes helpful. Small precious objects as pearls, diamonds and gold may be swallowed in an effort to smuggle them. Radiologic exploration might locate them.

USES OF FORENSIC RADIOLOGY

1. **Suspicious death or murder**: Prior to autopsy, radiographic techniques can assist the pathologist in determining the potential cause of death and may alert them to a previously unsuspected suspicious death. Autopsy technique may need alteration given the prior knowledge of specific radiographic findings.
2. **Analysis of adverse medical events**: During the course of hospital admission or medical care, adverse events may occur. Radiographic imaging is an integral part of the medical process and provides a permanent record of the patient's condition. Specialist review of these images in the light of autopsy findings and clinical audit can aid in the prevention of future mishap.
3. **Legal matters**: Following injury or assault radiological imaging is commonly obtained by medical practitioners. Expert interpretation of these images may provide useful evidence in criminal and civil jurisdictions.
4. **Child abuse**: As part of the workup of children with suspected abuse, radiological techniques are mandatory in providing evidence of both acute and chronic injury. Some of the findings are very specific for abuse and crucial for conviction of culprits.
5. **Drug trafficking:** Increasingly sophisticated methods are used by drug traffickers to avoid detection. This includes the deliberate ingestion or cavity insertion of drugs wrapped in protective materials (known as *"body packing"*). These packets are readily identified on CT scans and can assist law enforcement agencies in screening suspected individuals.
6. **Body identification**: Along with dental and DNA analysis, radiographic images be used in the identification of unknown victims. This requires the securing of premortem examinations, e.g. CT scan or radiographs from a suspected individual and the matching of specific anatomical details with similar postmortem studies.
7. **Disease identification (e.g. Marfan's):** during the course of an autopsy there may be findings that can have consequence to the deceased family i.e. the detection of possible genetic disease. In order to confirm such a pathological suspicion, radiographic procedures can provide confirmatory evidence and as such lead to the deceased's family being offered genetic screening.

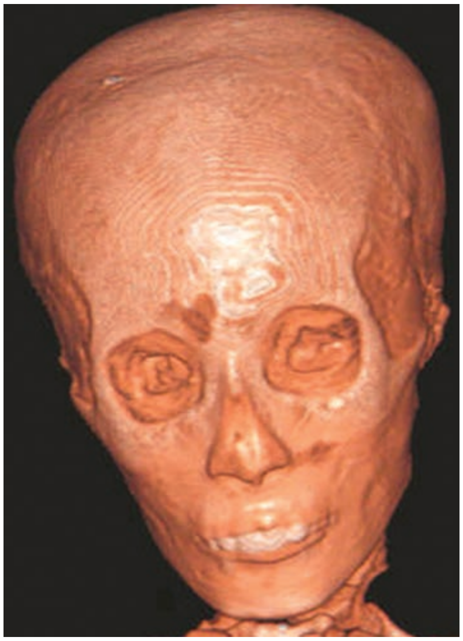


Fig. 29.11: 3D rendering of the skull of the Egyptian king

8. **Donor bank analysis**: all body parts donated for use as grafts in particular bone must be carefully screened prior to use in order to avoid the insertion of "diseased" material into a recipient. This can be accurately performed "non-destructively" utilising radiographic techniques.

FINAL COMMENTS

Tutankhamen, the last pharaoh of the XVIIIth dynasty, died unexpectedly at approximately age 18 years.[14] A cause of death has never been established, but theories that the young king was murdered by a blow to the head have been proposed based on skull radiographs obtained by a team from the University of Liverpool in 1968. Although both the original examination and the subsequent X-rays revealed much about the life of the king, they also left many questions open, and have provided fuel for much speculation. Recently a CT scan of the sarcophagus revealed extensive data which gave appropriate answers to these speculations.[14] *CT and MRI scanning* have recently been used to conduct *"virtual autopsies"*.[12] CT scanning is a non-invasive tool that can scan the whole body in a very short time, and can differentiate between various types of soft tissue and bone in three-dimensional images (Fig. 29.11). Conventional X-rays can see two planes only and cannot clearly distinguish the soft tissues. The scope and ability of CT scanning to diagnose and differentiate between diseases is also far superior. The body also does not need to be moved repeatedly, as is the case for X-rays. The technology is also allowing radiologists to unveil some amasing secrets within the body as if doing a scopy within the body – *"virtual autopsy"*.

Radiological methods of MSCT (Multi slice CT) and MRI have the potential to become a routine *"virtual autopsy"* tool in the future.[15]

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Chapter 30

Forensic Engineering

INTRODUCTION

The attack on the World Trade Centre (WTC) can be described as the most recent and probably the most appropriate example of forensic engineering. Whether the twin towers were waning in their strength or the blast impact was too much for them to bear with and perish; the forensic engineers are required to answer these and other such questions after the attack on the WTC. This example can give an idea about the kind of work a forensic engineer is expected to do.

Forensics engineering is essentially a failure analysis programme for litigation support. The goal of such a programme is to positively identify the sequence of events leading to ultimate failure. After an accident, forensics engineers examine broken parts and bring together a list of probable failure mechanisms to be investigated. Interviews are conducted to determine a sequence of events. Drawings, specifications, and operational procedures are reviewed. As-built dimensions and operating parameters are compared to design requirements. The final step in forensics engineering is to use analytical and testing tools to confirm the findings of fact. The forensics engineer becomes an expert witness in support of the findings. He is also responsible for reviewing the technical aspects of the opposition's case.[1-3]

DEFINITION

Forensic engineering is defined as the application of engineering principles, knowledge, skills, and methodologies to answer questions of fact that may have legal ramifications.

EXPLANATION

Forensic engineering is the investigation of materials, products, structures or components that fail or do not operate/function as intended, causing personal injury or damage to property. The consequences of failure are dealt with by the law of product liability. The subject is applied most commonly in civil law cases, although may be of use in criminal law cases. Generally, the purpose of a forensic engineering investigation is to locate cause or causes of failure with a view to improve performance or life of a component, or to assist a court in determining the facts of an accident. It can also involve investigation of intellectual property claims, especially patents.[4-6]

CASE EXAMPLE HANDLED BY FORENSIC ENGINEERS

A forensic engineer is called upon to analyze car accidents, building collapses, fires, explosions, industrial accidents, and various calamities involving injuries or significant property losses. This can be taken as the best example of a truly multidisciplinary field. That's because the very name forensic engineering is made up of two totally different fields. A forensic engineer has to have the basic knowledge of forensic medicine, advanced understanding of forensic sciences, preliminary knowledge of mathematics, various principles of physics, a working understanding of statistics and of course a good experience of engineering. The inclusion of all these branches may suggest that this is a very difficult field, which indeed it is.

Forensic engineering is too vast a topic to be covered in just one chapter. As already mentioned, it includes the investigation of various kinds of accidents like explosions, building collapses, vehicular accidents, etc. And each of these involves the use of different methods, equations, terminology, etc. As a result, it's very difficult to generalize these features. And there is also the constraint of space. So authors have done the best thing possible, describing the more common occurrences in detail while giving just a passing reference to the other less common instances. The equations that are involved in solving these problems like the Newton's Laws, thermodynamics, and properties of liquids and gases, etc., have been deliberately omitted to facilitate easy learning and understanding of the topic.

Forensic engineering is the application of engineering principles and methodologies to answer questions related to fact and law. A forensic engineer can also be called as the "*reverse engineer*". This is because when he is called upon, the event has already occurred and he has to find the cause of that occurrence. Because they *reconstruct the event*, they are also sometimes known as "*reconstruction experts*".[7] In short a forensic engineer does the following things while working on a case:

- Assess the condition at present, i.e. the condition after the event or the post event condition.
- Predict the condition before the occurrence of the event, i.e. the pre-event condition (he uses both reconstruction and history for this purpose).
- Hypothesize the various ways in which the event could have occurred, i.e. the ways in which the pre-event condition that has become post-event condition.
- Search the various evidences that can either support or refute the hypotheses.

This can be said to be the working principle of a forensic engineer. The importance of forensic engineer lies in the fact that he has to apply the various scientific principles to the facts that he has gathered to prove that the evidence and the hypothesis correlate with each other. For this reason the scientific investigation and analysis is structured like a pyramid (Fig. 30.1).

INVESTIGATION OF DAMAGE DUE TO FIRE

The damage caused due to fires is one of the most common damage in India. As a result we are going to discuss it first. It

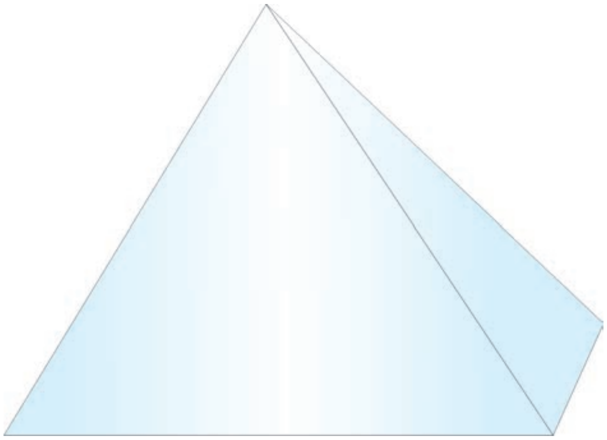


Fig. 30.1: Structure of a pyramid

is also one of the most difficult of cases to be dealt with. The reason for this is not difficult to find. First of all, the fire itself causes the loss of crucial evidences. This makes fire an important method used by the perpetrators of crime to destroy the evidences. Secondly the fire extinguishing process causes further loss of evidences. But this cannot be avoided, as the extinguishing of fire is the first and the foremost requirement to prevent further damage to the structure and to prevent further loss of precious lives. As a result the investigation of the scene of fire requires the investigator to use all the skills at his disposal.[2]

Chemistry of Fire

Fire is defined as the rapid oxidation process with the evolution of heat and light. The components necessary for fire are described by the fire tetrahedron that involves four components: fuel, heat, oxygen and uninhibited chemical chain reactions. Out of these components, oxygen is present in the atmosphere in abundance except in the close environments like attics, storerooms, etc.

Secondly every structure has abundant inflammable material in the form of clothes, wood, papers, and plastic, etc. The chemical reaction involved in fire is a self-propagating reaction that goes on by itself, once started. So the only component that is missing is the heat necessary for starting the combustion process. The finding of this heat source generally makes up the first step in the investigation. This heat source also leads to the point of origin of fire. This point of origin can be found out by finding "*the low point*". This low point is based on the principle of various rates of propagation of fire in different directions. The rate of propagation of fire is maximum in the vertical direction followed by the horizontal direction and least in the downward direction. This differential rate of propagation causes fire to move in the form of a "V" (Fig. 30.2). This "V" is formed on the walls. On the furniture and other materials, this forms a three dimensional "V", in other words a cone. In Figure 30.3 we have assumed the burnt area as the coordinate system and origin as the point of origin of fire. Then the differential rate of propagation of fire leads to the formation of the pattern as shown.[2]

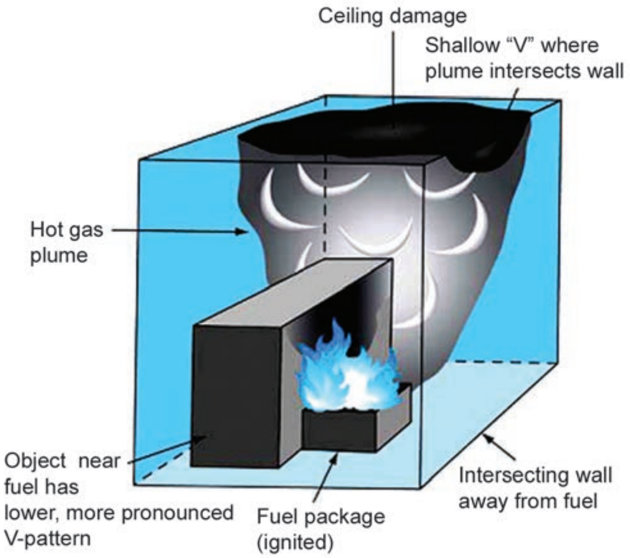
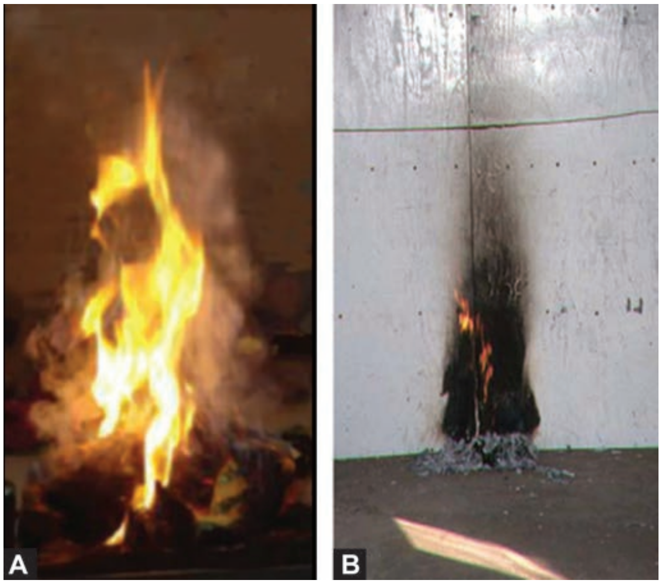

Ceiling damage
Shallow "V" where plume intersects wall
Hot gas plume
Object near fuel has lower, more pronounced V-pattern
Fuel package (ignited)
Intersecting wall away from fuel

Fig. 30.2: Schematic representation of the formation of "V" patterns by a fire plumes (*Source:* DJ Icove and JD DeHaan. "Forensic Fire Scene Reconstruction," Prentice Hall, Upper Saddle River, NJ, 2004)



Figs 30.3A and B: An inverted "V" shaped fire pattern during and at self-extinguishment (*Source:* Photos courtesy of Susan Sherwin, Maricopa County Attorney's Office)

Getting Started

While investigating the damages caused by fire, one should start from the unburnt areas. This has two advantages. Firstly, it tells that the point of origin of the fire is not at that point. As already mentioned, a forensic engineer makes some theories and then goes on to prove or disprove them. Examining the unburnt areas helps to dispel some of the theories regarding the point of origin of the fire. Secondly, fires damage the structures and they may fall any time during investigation. So by observing the unburnt areas the investigator can get an idea about the strength of the standing structure and prevent loss of lives of the investigators. After examining the unburnt area, one has to then proceed from the less burnt area to the more burnt area in the form of a circle of decreasing diameter.

Let us now describe the various methods that are used to investigate a scene of occurrence of fire, especially the point of origin.[1]

Centroid Method

This method is more useful in cases in which the building is of only one story high and is made of uniform material. In such cases the midpoint of the burnt area is the point of origin of fire. That is because the presence of homogenous material means that the fire spreads with same speed in all directions. The shortcoming of this method is encountered when the fire starts at the corner of a wall. In such cases we cannot find the centre of the fire. In such cases the use of a mirror is made. Here a mirror is placed near the wall from where the fire appears to have started. This gives the investigator an idea about the extent up to which the fire would have spread had it started in the middle of the room.

Warehouse or Box Method

In this method the damage that fire causes to the walls or the room is used. It is based on the fact that the wall on which fire reaches first suffers the maximum damage, followed by the one on which fire reaches second and so on. Consequently by using simple mathematical calculations one can find the point of origin of fire. The point of intersection of perpendiculars drawn from fire-damaged area on each wall gives the site of origin of fire.

Weighted Centroid Method

This method is similar to the centroid method. This method assumes that the point that is subject to the longest duration of fire and so is the most severely damaged, is the point of origin of fire. So the areas that are most damaged are the most likely to be the point of origin of fire. As a result these areas are given more importance while investigating the scene. The problem that is encountered in this method is that all buildings don't burn homogenously. This is all the more important when there is a gas cylinder or a can full of kerosene is kept that can explode when fire reaches that area. In such cases the area in which such a material is kept is burnt more than the primary area or the point of origin. Consequently the proper history regarding the places where such material could have been kept becomes important.

Sequential Analysis

In this method the point of origin of fire is determined by following in reverse order the trail of fire from where it ends to where it began. The point where various trails merge is the point of origin. The various indicators of temperature and fire include wood, paint, finishes, and coatings, etc. The advantage of this method is that the homogeneity of the material is not to be assumed as is required in other methods. The disadvantages of this method are twofold. Firstly this method is investigator dependent. This is because it is based on the skill and knowledge of the investigators. And as we all know, all investigators are not equally good and that makes this method highly subjective. Secondly, this method assumes that there are significant markers present to indicate the direction of spread of fire. This may not be true in all cases. The markers may be lost in the debris or may have been damaged due to the extensive fire. In such cases the trail may not be complete and may be discontinuous.

Combination of Methods

One single method is not complete in finding the cause and the point of origin of fire. So the combination of methods is used to find the point of origin of fire. In such cases the investigator starts off with one method and then goes on to another method depending on the findings of that particular case.[1,2]

Arson and Incendiary Fires

Arson can be defined as the malicious burning of homes, residences, buildings, or other types of real property. These are very important as arson may be used many times to conceal some other crime that may have been committed or to take undue profit or advantage from the insurance companies. Here we are not going to discuss the incendiary fires in detail but will describe a few characteristics of these fires.[2]

1. The presence of multiple origins of fire especially when they are not connected to each other.
2. The point of origin is an area where the source of ignition is not possible.
3. The presence of inflammable materials at the site like gasoline, kerosene, etc.
4. The presence of trailers. These are the areas of burn that exhibit a deliberate pattern of pouring inflammable material between two areas of fire.
5. The presence of heaps of material that can burn easily. This indicates that the material had been deliberately placed to enhance the chances of them catching fire.
6. The finding of tampering of fire protection and alarm systems.
7. The presence of unnatural fire patterns; a fire pattern that does not follow the rules and has burned in an unusual sequence.
8. The presence of evidence of tampering with heating and air conditioning equipment to enhance fire spread.

ELECTRICAL SHORTING

Electrical shorting is an important aspect of investigation of cause of fire. That is the reason we have mentioned it at this point. Most of the fires that break out in homes and offices are due to electrical short circuit. The reasons why a short circuit causes fire are manifold.[1,2] Firstly short circuit causes the electrical conductors to heat up excessively. This causes the insulating material, that is usually made of plastic, to heat up and melt, leaving the conductor bare. When an inflammable material comes in contact with this bare conductor, it catches fire. Secondly if the conductor is heated sufficiently, it may melt and fall down. The presence of any inflammable material below can ignite fire. Lastly when the inflammable material is very close to the shorting, it may be heated up due to the transfer of heat directly by any of the methods viz. conduction, convection or radiation. This can then start a fire.

Shorting is of two types – primary and secondary. Primary shorting is that shorting that has started the fire while secondary shorting is the one that is caused due to fire. Many times the investigator, when unable to find a cause for fire, puts the blame on shorting although it may be secondary shorting. So it is imperative that one is aware of the differences between the two.

Characteristics of Primary Shorting

- It occurs at or close to the point of origin of fire.
- Heat damage to the interior of the conductor is much more than exterior.
- The severest damage occurs in an area near the short.

Characteristics of Secondary Shorting

- It occurs away from the point of origin of fire.
- Heat damage to the interior of the conductor is less than the exterior.

The presence of primary and secondary shorting is not exclusive and both may be present in the same conductor albeit at different locations.

MOTOR VEHICLE ACCIDENT

Motor vehicle accidents are the latest epidemic that has gripped the modern world. The number of deaths that occur in the motor vehicle accidents far outweigh those due to any other cause. Coupled with the amount of insurance money involved in it, it is a very important investigation that has to be undertaken by a forensic engineer. The things that have to be found out in a motor vehicle accident are generally whether the vehicle was over speeding or not, whether there was any fault in the maintenance of the vehicle, whether the accident was a result of carelessness or was a deliberate sabotage and so on. For this the examination of the vehicle and the site of accident have to be undertaken. The examination of the skid marks assumes a great importance.[1,4,5]

Skid Marks

Skid marks are vital evidence for vehicular accident reconstruction, when their size and shape can reveal much about vehicle speed and forces of acceleration or deceleration (Fig. 30.4). They are one form of trace evidence. They represent a form of contact evidence produced according to Locard's exchange principle. The length of the skid mark is usually closely related to the vehicle speed at the instant of braking, so measuring the marks yields an estimate of original speed.[4]

Let us now see how this examination is undertaken.

Examination of Skid Marks

The skid marks are produced due to the force of friction acting between the vehicle that is decelerating and the ground. The basic principle that is involved is as follows: Every moving body has some kinetic energy. When brakes are applied this kinetic energy has to be dissipated in some way so that the body decelerates and finally comes to a stop. This dissipation of energy takes place in the form of heat that is produced due to friction when brakes are applied. This heat energy that is produced in the tires raises their temperature. This increase in temperature causes the tires to burn leading to the formation of the skid marks.[4] The examination of the skid marks tells about the velocity of the vehicle at the time when the brakes were applied. This is achieved by using the basic laws of motion. When the body comes to a standstill after the application of the brakes, the final velocity becomes zero. Since the deceleration is constant and we are aware of the distance traveled by the vehicle after application of the brakes, we can find the initial velocity. But the problem comes when the body hits some other object, moving or stationary, before stopping. How such cases are dealt with will be discussed later.

All skid marks are not the same.[5] Some marks may be simple as has been described above. But there may be times when the marks may be produced due to special situations. Some of these situations include curved skids, skids on slopes (both upward and downward), presence or absence of weight on the vehicle, etc. These cases are dealt with differently and it is also more difficult to solve them. But they can be solved using some extra knowledge and application.

And now a few words about Antilock Braking System (ABS).[2] Antilock braking system has been devised to prevent the tiers from locking when the brakes are applied. In other words, they prevent skidding of the vehicle when the brakes are applied suddenly. But they are not 100 per cent skid proof. What it means is that due to ABS the vehicle skids and runs alternatively when the sudden brakes are applied. This gives a pattern of skipped skids. Also this skid is less faint than that of the non-ABS vehicles. Still the skids are present and their examination is done in the same manner as that of the other vehicles.

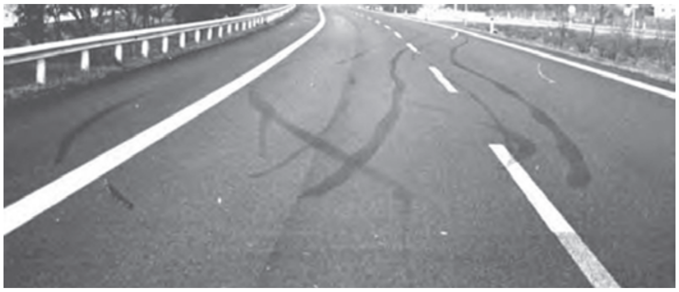


Fig. 30.4: Skid marks on the road

Methods of Examination

In this section we are going to discuss the various methods that are used to examine the scene of accident and the theories that are brought in to play to find the various parameters that are required in the investigation. We will also discuss the cases in which the vehicle strikes a body before coming to a halt.[2]

Momentum Method

This method is based on the principle of conservation of momentum. When two bodies collide, the combined momentum of the system, i.e. the sum of momentum of the two bodies before and after the collision remains same. Using this principle, and knowing the amount of deformity of each vehicle, we can find the initial velocity of each vehicle. When the bodies collide, there is collision. Theoretically, collision can be either elastic or plastic. But in practice there is no such thing as a perfect elastic collision. So the properties of both elastic and plastic collision are used to find the initial velocity when this method is used. Whenever there is a collision there is some twisting and rotation of the vehicles. So the use of angular momentum, torque and other equations of rotational motion are used.

Energy Method

This method is based on the principle of conservation of energy. When two bodies collide the combined energy of the system, i.e. the sum of potential and kinetic energy of both vehicles remains same. This means that the deformation that takes place in each vehicle depends on the kinetic energy of the system. By noting the deformation that each vehicle has taken, we can deduce the initial velocity. The maximum deformation that takes place in either vehicle is taken as the amount of deformation that has occurred.

BUILDING COLLAPSE DUE TO ROOF LEAKAGE

Collapse of buildings due to damage of their supporting structures is a common cause of building collapse in India.[2,9] The climatic conditions in this part of the world increase the chances of such damage. This amounts to a huge loss of capital for the country. The importance to know the causes of such collapses is twofold. *Firstly* it is needed so that the buildings can be designed in such a way that the further loss can be avoided. *Secondly* it has to be noted whether the collapse was due to the natural wear and tear of the building or was a result of some *sabotage*. And this cannot be found out unless the investigator is aware of the ways in which the natural damage to the building occurs.

There are two types of loads that a building has to bear.[9] These are the *static loads* and *dynamic loads*. *Static loads* are the loads of the building itself. These are further subdivided into *dead loads* and *live loads*. Dead loads are the loads that don't change with time. These include the weight of the floors, roofs, walls, etc. Live loads include those weights that may change with time of the day or season like furniture, machinery, human beings, etc. *Dynamic loads* include the loads that change in a very short period of time. These include strong winds, earthquakes, drilling machines that can shake the building, etc. Since these forces act for a short duration, they cause a lot of damage to the buildings. The ways in which both these forces act on the buildings to weaken it and lead to its failure is beyond the scope of this book. But what we are going to discuss is the ways in which the normal wear and tear causes damage to the building.

Every time there is rainfall the water seeps into the building. When the building is new, this doesn't cause much perceptible damage. But when it grows old, the damage that has been caused earlier comes to light. This water leakage leads to the weakening of roof and walls. It also causes damage to the wooden material present in the building. This water causes bacteria and fungi to colonize the walls and the roofs. It causes softening and weakening of the wooden material. These softened beams cannot hold the weight of the building leading to its collapse.[2] Another way in which water causes damage to the building is by causing chemical reaction with the soda lime present in the walls. This leads to the formation of highly alkaline solution in a highly exothermic reaction. This dissolves the calcium hydroxide present in the walls. As a result the mortar becomes a highly porous material that can easily fall off causing weakening of the walls and their imminent collapse.[2,9]

EXPLOSIONS

An explosion is a sudden, violent release of energy. It is usually accompanied by a loud noise and an expanding pressure wave of gas. Explosions are of three types:[2,10] These are Deflagrating Explosion, Detonating Explosion and third unnamed variety of explosion in which there is sudden expansion of high pressure gases as occurs in a ruptured high-pressure vessel of pipe.

Deflagrating Explosion

These are the type of explosions in which there is a relatively slow, progressive burn rate of the explosive material. This type of explosion causes damage by pushing things around by pressure differentials. What this means is that there is a formation of a pressure gradient between the site of explosion and the surrounding areas. This pressure gradient pushes the things that come in its way causing damage like walls, ceilings, large furniture, etc. An important property of this type of explosion is that small objects lying near the epicentre of the explosion are left undamaged. This occurs because the pressure wave that is formed goes around them.

Detonating Explosion

These are the type of explosions in which there is high burn rate, high energy release and a high peak explosion pressure. Consequently these explosions are associated with higher damaging properties than deflagrating explosions. These explosions tear apart the objects lying near their epicentre and may be useful in blasting work.

The reason why these two types of explosions have been mentioned is because the distinction between the two is important in their investigations. The common explosions that occur in households are generally of the deflagrating type while those that are generally involved in arson, sabotage and terrorist activities are of detonating types.

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Part V: Forensic Toxicology

Chapter 31

General Principles

INTRODUCTION

The knowledge of poison is perhaps as old as the history of mankind. *Hippocrates* and *Aristotle* had mentioned the details of all the known poisons according to ancient literatures.[1] *Socrates* is said to have done a detailed study on *hemlock*, the *state poison* of the Greeks, in his time.[2] *Dioscorides*, a Greek physician in the court of *Nero*, had classified poisons, into *plant, mineral* and *animal poisons*, which is still a convenient format today. Early Romans had developed the concept of deliberate poisoning into a profession like *'contract killers'* or *'hit men'* of current time. However the most powerful historical influence on the development of the science of toxicology was probably by *Phillipus Aureolus Paracelus* (real name – *Theophrastus Bombastus von Hohenheim*), who in 16th century contributed his visionary realization of poisoning in terms of dose and the difference between therapeutic and toxic doses of a substance.[1] Recent days have seen tremendous update in the field of toxicology, especially with the development of chemical and biological warfare.[3] Search in literature confers the fact that increasing number of developments in the industrial and agricultural use of chemicals has been occuring, globally. In spite of laying several laws on poison, environmental pollution of land, ocean and air by these poisons shall not only haunt us now, but will keep continue to haunt the future of mankind too.

DEFINITIONS

Defining terminologies are of immense significance. It is easier to understand terminologies but they are difficult to define. Review of literature provides definitions as defined by others or redefined with little modifications.[4-7]

Toxicology

Toxicology is the branch of medicine that deals with properties, action, toxicity and lethal dose, estimation of, treatment and autopsy findings of poisons.

Forensic Toxicology

Forensic toxicology deals with medical and legal aspects of the harmful effects of the chemicals on the human body.

Clinical Toxicology

Clinical toxicology refers to human diseases caused by or associated with abnormal exposure to chemical substances.[5]

Toxinology

Toxinology refers to toxins produced by living organisms, which are dangerous to man, e.g. venom of snakes, spiders and bees, bacterial and fungal toxins, poisonous plants, etc.[4,8]

Drugs

Drugs are natural or synthetic substances, which are used to exert physiological or psychological effect on the consumer.[4,6]

Poison

According to Webster's Dictionary, 'poison' means a substance, which through its chemical action usually kills, injures or impairs an organism.[5] Poison is defined as any substance which when administered by any route can cause disease, deformity or death.[4] Poisoning thus connotes clinical symptomatology. Poisoning also implies that the toxic exposure was accidental, unintentional, or unknown to the patient as in victim of intended homicide.[5] According to law statutes,[9] any substance, irrespective of its quality or quantity, when given with intention to endanger, injure or kill a person is called poison.

Overdose

The terminology overdose in general implies an intentional toxic exposure.[5]

CLASSIFICATION OF POISONS

According to the main symptoms produced, poisons are basically classified into four groups, namely: corrosives, irritants, systemic poisons and other poisons,[4-7] (Tables 31.1 to 31.4).

ACTION OF POISONS

Poisons act usually by three ways: locally, remotely and both locally and remotely.[3-7]

- *Locally acting*: These act only at the site of application such as skin/mucosa, e.g. corrosive poisons.
- *Remotely acting*: These act only after being absorbed into the circulatory system, e.g. narcotic poisons, cardiac poisons, etc.
- *Both locally and remotely acting*: These act by local and remote actions, synergistically, e.g. carbolic acid, etc.

Factors Influencing the Action of Poison

Several factors influence the action of poison[4-7] and they are:

Route of Administration

A poison could be administered orally, hypodermically, intramuscularly, intravenously, endemically (rubbing), per-

Table 31.1: Corrosive poisons

Strong acids		*Strong alkalies*	
Inorganic or mineral acids	**Organic acids**	**Hydrates of**	**Carbonates of**
• Sulphuric acid	• Carbolic acid	• Sodium	• Sodium
• Nitric acid	• Oxalic acid	• Potassium	• Potassium
	• Hydrochloric acid		

Table 31.2: Irritant poisons

Inorganic	*Organic*	*Mechanical*
Nonmetallic • Phosphorus • Halogens	**Vegetable** • Abrus, castor, croton • Calatropis, ergot, etc	• Diamond dust • Glass powder • Hair • Nails • Pins, etc
Metallic • Arsenic, mercury • Lead • Copper, etc.	**Animal** • Snake or insect bites/and stings	

Table 31.3: Systemic poisons

Central nervous (Neurotoxics)	*Cardiovascular*	*Lungs (Asphyxiants)*
Cerebral **Somniferous** • Opium, pethidine **Inebriants** • Alcohols, anaesthetics • Sedative hypnotics • Insecticide, hydrocarbons • Benzodiazepines, etc **Delirients** • Datura, cannabis • Cocaine, etc. **Spinal** • Strychnine, gelsemium, etc. **Peripheral** • Curare, conium, etc.	• Oleanders • Aconite • Nicotine	• Carbon monoxide • Carbon dioxide • Irrespirable gas • Cyanogen gas • Amol cyanides

Table 31.4: Miscellaneous/other poisons

Domestic poisons	Kerosene, diesel, petrol, cleansing agents, soaps, detergents, disinfectants, cosmetics, etc.
Therapeutic substances	Salicylate, paracetamol, antidepressants, sedatives, antipsychotics, insulin, etc.
Food poisoning	Bacterial, viral, mushrooms, chemical, etc.
Drugs of dependence	Alcohol, tobacco, hypnotics, hallucinogens, stimulants, organic solvents, etc.

Table 31.5: Toxicity rating of poisons

Fatal dose schedule	*Toxicity rating*
< 5 mg/kg	6 (Super toxic)
5 to 50 mg/kg	5 (Extremely toxic)
50 to 500 mg/kg	4 (Very toxic)
500 mg to 5 gm/kg	3 (Moderately toxic)
5 to 15 gm/kg	2 (Slightly toxic)
>15 gm/kg	1 (Non-toxic)

rectally, per vaginally, per vesically (urinary bladder), by inhalation, etc. For a rapid action, a poison may be better given intravenously than orally.

Idiosyncrasy

Idiosyncrasy means unexpected allergy or intolerance. It brings about untoward effects or ill health or death, e.g. allergy to certain drugs like penicillin, certain foodstuffs like eggs, shellfish, etc.

Age

Age has got considerable relationship to dosage for any poisonous substance, e.g. dosage required for children is usually half that in an adult.

Addiction

Taking small quantity of a poison or a drug for a long duration can lead to its diminished effect. This is called addiction or habit formation, e.g. addiction to morphine, barbiturates, etc.

Dose

Depending on dosage, a poison could be either useful or harmful, e.g. aspirin, morphine, etc. which with a proper dose can act as an analgesic, but with higher dose can induce fatality.

Health of Individual

A healthy, normal individual can withstand a poison ingested for a longer duration than an individual unhealthy and debilitated.

Concentration of Poison

This factor is highly responsible for the development of classical effects of any poison or drug, e.g. sulphuric acid burns are developed only with concentrated form of the acid.

Chemical State of Poison

These factors are responsible to derive the rate of solubility, absorption, etc. for a poison to get into the various systems of the living body.

Table 31.6: Usual fatal dose (UFD) of common toxic agents

Toxic agents	*UFD*	*Toxic agents*	*UFD*
Acids (Mineral)	10-15 ml	Isopropanol	200-250 ml
Aconite roots	01 gm	Iron	200 mg/kg
Aluminium phosphide	03 gm	Lead acetate	10 gm
Arsenic trioxide	250 mg	Lindane	05-30 gm
Aspirin (Sodium salicylate)	15-20 gm	Malathion	01 gm
Atropine	10 mg	Mercuric chloride	01-02 gm
Barbiturate (Long acting)	03 gm	Methanol	60-250 ml
Barbiturate (Short acting)	01-02 gm	Morphine	200 mg
Carbon monoxide (COHb)	50-60%	Nicotine	60 mg
Castor seeds	05-10	Oleander roots	15 gm
Cocaine	01-02 gm	Oleander leaves	05-15
Copper sulphate	30 gm	Opium	500 mg
Curare	60 mg	Oxalic acid	15–20 gm
Cyanide	200-300 mg	Paracetamol	12-20 gm
Datura seeds	50-75	Phenol	20 ml
DDT	15-30 gm	Parathion	100 mg
Diazinon	01 gm	Phosphorus	60-120 mg
Ethanol	05-08 gm/kg	Strychnine	50-100 mg
Ethylene glycol	100 ml	Salicylic acid	70-80 gm
Formaldehyde	30-60 ml	TEPP	100 mg
Heroin	50 mg	Thallium	01 gm

Physical State of Poison

This means the state of existence of a poison, i.e. gas, liquid or solid state. For gas, inhalation is the best route for rapid onset of action. For liquids, onset of action is more rapid than solids when administered orally. Among solids, fine powder acts faster than coarse powder.

Toxicity Rating of Poisons

Based on fatal dose of a poison, toxicity rating of poisons is derived for the purpose of assessing the progress of a case of poisoning clinically.[4,5] Toxicity rating by Gosselin RE and his colleagues[10] is presented in Table 31.5, it may be presumed from this table that higher the toxicity, greater is its potency and worse is the prognosis. However, other factors also affect this.

Lethal Dose (Fatal Dose)

Lethal dose is usually difficult to fix due to various factors that can affect the action of a poison. This renders lethal dose for a given poison only in an "*Approximately Fatal Dose*" (*AFD*), which is essentially to help the treating physician to assess the prognosis of a case. This can also be called as "*Usual Fatal Dose*" (*UFD*).[11,12]

Usually the UFD is based on *minimum lethal dose* (MLD), which is usually indicative of the lethal dose that is fatal to 50 per cent of animals (LD 50). Table 31.6, enumerates the UFD of common toxic agents and is meant for a quick reference for the reader.

It may be also remembered here that for most of the poisons dealt with in this book throughout, lethal doses mentioned are thus just an Approximately Fatal Dose (AFD)/Usual Fatal Dose (UFD) that too meant for an adult unless otherwise mentioned for children.

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EPIDEMIOLOGY OF POISONING

According to the University of Newcastle Medical Dictionary.[1] epidemiology means the study of the distribution and determinants of health-related states and events in populations and the control of health problems, the study of epidemic disease. It also refers to field of medicine concerned with the determination of causes, incidence, and characteristic behavior of disease outbreaks affecting human populations and includes the interrelationships of host, agent, and environment as related to the distribution and control of disease. The toxicologic epidemiology is the study of the distribution and determinants of health related states and events in populations and the control of poisoning related health problems. It is a silent but growing field in public health.

Many Indians consider that taking life of either a human being or an animal by blood-shed is a greater sin.[2] This belief has led to the use of poisons for the purpose and it is a sad fact that cases of poisoning are increasing day by day. The incidences of poisoning in this country are mainly:

- Suicidal
- Homicidal
- Exhibitional (for creating sympathy)
- Stupefying (to induce the condition of unawareness)
- Aphrodisiacal (A drug or other agent that stimulates sexual desire and also known as love philter, love pations, etc.)
- Abortifacient (to induce abortion)
- Accidental
- To kill animals — cattle (to obtain hides), rodents, stray dogs, etc.
- To kill insects (as insecticides).

While considering suicide or homicide with poison, it is an observed fact that the suicide victim prefers to terminate life as quickly as possible with least agony; while for an act of homicide, the criminal would like to kill his victim, without creating any suspicion in the mind of the victim or people around. The criminal mind also prefers to use such a poison, which would allow him sufficient time to escape. This implies that whether it is for suicidal purpose or for committing homicide, only a few selected poisons are used. Table 31.7, enumerates the characteristics of poisons which are considered as 'ideal suicidal poison' or 'ideal homicidal poison'.[2-5]

Among the developing countries, Sri Lanka presents suicide rates of 40 cases per 100 000. This greatly exceed those of the United Kingdom (7.4/100 000), United States (12/100 000), and Germany (15.8/100 000).[6,7] A leading method of committing suicide in Sri Lanka is ingestion of pesticides, which are readily available in rural farming households. Self-poisoning kills more people in rural Sri Lanka than ischaemic heart disease and tropical diseases combined.[8] Although acute pesticide poisoning occurs at alarmingly high rates in Sri Lanka; it is also a major problem throughout the developing world. The worldwide incidence is three million cases and 220000 deaths each year.[9]

In USA, 430 Poison Information and Control Centre receive more than 1.5 million calls yearly with reference to exposure to potentially toxic substances.[6,7] Ninety per cent of all exposure cases have occurred in the home.[10,11] Children under 5 years of age account for 60 per cent of these exposures.[5,10] Accidental poisoning accounts for 89.9 per cent and intentional 8.2 per cent. In 79.2 per cent cases, poison was ingested, 6.3 per cent occurred through dermal absorption, 5.4 per cent from eye, 5.4 per cent from inhalation and 3.7 per cent from bites and stings. Deaths were most often associated with antidepressants, analgesics, sedatives, hypnotics and carbon monoxide. Out of 1,10,000 calls received only 20,000 patients required hospitalization.[11]

The epidemiology of acute poisoning in India among children aged 0-15 years presented distinct patterns: accidental poisoning in under 11's and adult pattern and self-poisoning in children over 11's. More children belonged to urban areas (72.3%) as compared to rural areas. In both decades reported more than 50 per cent of children belonged to middle income group followed by lower income group and least to upper income group. The overall mortality was low (12.5%), with majority of deaths (78%) is occurring in older children, highlights the unacceptable fact that a high rate of preventable accidental poisoning is reported among young children (0–1 years age group) and suicide and para suicide in older children (11-15 years age group).[12]

European data are similar to American data with the majority of poison centre calls involving children less than 5 years of age with peak exposure between 2 and 3 years of age. Passive poisoning (inappropriate administration of medication by care providers such as parents) is the common cause of poisoning less than 1 year, whereas toddlers' exposures most often result from inquisitive behaviour. Accidental ingestion beyond the age

Table 31.7: Characteristics of ideal suicidal and homicidal poisons[2]

Characteristics	*Suicidal*	*Homicidal*
Accessibility to the poison	Easy and free	Not particular
Antidotes availability	Nil	Nil
Clinical diagnosis	Difficult	Difficult
Cost of the poison	Cheap	Not particular
Death	Painless	Definite
Metabolism and excretion	Not particular	Rapid
Onset of signs and symptoms	Quick	Slow
Postmortem detection	Difficult	Difficult
Signs and symptoms	Nil/few	Resemble diseases
Solubility in food/drinks	+ve	+ve
Examples*	Opium, barbiturates, Organophosphrus compounds	Arsenic, aconite, thallium, etc

* Though no single poison would fulfil all the characteristics mentioned in the Table 31.6, a few examples mentioned above are often considered

of 5 is unusual and typically reflects the mistaken consumption of a substance from a mislabeled container. Sporadic reports of children poisoned by their parents do occur. The average victim is 3 years old, with a range of 2 to 94 months.[2,13]

Exposure to substances between the age of 5 and 9 years may reflect intrafamily stress or suicidal intent. Beyond the age of 9 years, voluntary exposure is the rule. Poisoning mortality is lowest between the ages of 5 and 14 years. The incidence of suicide in children between 5 and 12 years of age reportedly is between 0.2 and 0.4 per hundred thousand. Most exposures in the elderly over 60 years result from accident (83%), 14.8 per cent with suicidal intent and 1.7 per cent due to drug abuse, which are more common in young adults.[2]

There were 21,600 hospitalizations from poisoning in children between birth and 9 years of age in USA. The hospitalization rate was 65.1/1,00,000, with a maximum in the age group of 1 and 2 years and minimum in the age range of 5 to 9 years. Out of the total fatalities in the US, 49.7 per cent were suicides and 39.5 per cent were accidental in 1980. A dramatic decline was found in deaths from unintentional ingestion in children less than 5 years of age, which peaked in 1959. It further decreased in 1981 and accounted for only 1 per cent of poisoning related deaths. It is also noted that the tendency was towards smaller single and accidental ingestions in children under 5 years of age as compared with larger, multiple and intentional ingestion in adults. The overall US suicide rate changed little between 1970 and 1980. Although it changed little, rates for older persons decreased and rates for younger persons increased.[3] A decrease in suicide rate was seen in women except between 15 to 24 years and there was an increase in the rate for men. Most women preferred firearms than poisoning to commit suicide in 1980. This reversed the trend from 1970.[2]

Forty per cent of the exposures in children less than 5 years of age were due to pharmaceuticals followed by cleaners, polishes and plants. The drug products most often involved vary from region to region. While Salicylates were preferred in some places, acetaminophen and cough and cold preparations were preferred elsewhere. However, poisoning patterns have changed dramatically as a result of changing physician and consumer preferences, availability of new drugs and the passage of child resistant packaging law.[2,3]

In the US medicinal substances accounted for 45 per cent of the childhood hospitalizations in 1984 with aspirin and the analgesics as the most common drug group. Lead was the substance most often responsible for hospitalization among the remaining nonmedical category. The 1980 FDA data on poisoning mortality indicated that pharmaceutical products caused 55.5 per cent of the United States poisoning fatalities, while gases accounted for 33 per cent and other solids and liquids accounted for the rest. Most poisoning deaths in 10 to 17 years age group were as a result of carbon monoxide.

Recently, surveys show that cyclic antidepressants have replaced barbiturates and shows 4-fold increased fatalities due to it in last 10 years. Drugs of abuse have become more important in the 18 and 19 years old patients. Half of all drug deaths are caused by multiple ingestions and 50 per cent of overdose results from suicide attempts. The vast majority of poisoning fatalities (80%) occur prior to arrival at the hospital facility.[2,3]

A spectrum of autopsy study on fatal poisoning for the recent five years in Manipal (January 1993 to December 1997), presents the Indian profile.[14] Out of all fatal cases referred for medicolegal autopsy during the period, 20 per cent constituted deaths due to poisoning. Among these, almost 10 per cent were brought dead cases, rest of the cases received prior treatment at public health centres (PHC) or at some hospitals of either government or private sector. The study revealed gradual upward tendencies from nearly 20 per cent case incidence in 1993 to more than 40 per cent in 1997 involving 35 per cent of the young adult of age group of 20 to 30 years, with a male: female ratio of 2.5:1 is really alarming. A definite preponderance toward suicidal (70%) tendencies among low socioeconomic group of study population, followed by accidental (24%) deaths is not only astonishing but also disgusting.[14]

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OCCUPATIONAL AND ENVIRONMENTAL TOXICOLOGY

Human beings live in a chemical environment. Estimates indicate that more than 60,000 chemicals are in common use and about 500 new chemicals are said to enter the commercial markets annually. Pollution has paralleled technologic advances. Industrialization and creation of large urban centres have led to the contamination of air, water, and soil. The principal causes of pollution are related to the production and use of energy, the production and use of industrial chemicals, and increased agricultural activity. Scientific study concerning this constitutes new subdivisions of toxicology, namely the "Occupational toxicology' and 'Environmental toxicology'.[1-5]

OCCUPATIONAL TOXICOLOGY

Occupational toxicology deals with the chemicals found in the workplace. Industrial workers may be exposed to these agents during the synthesis, manufacturing, or packaging of these substances or through their use in an occupational setting. Agricultural workers, for example, may be exposed to harmful amounts of pesticides during their application in the field. The major emphasis of occupational toxicology is to identify the agent of concern, define the conditions leading to their safe use, and prevent absorption of harmful amounts. Guidelines have been elaborated to establish safe ambient air concentrations for many chemicals found in the workplace.[1,2]

The American Conference of Governmental Industrial Hygienists periodically prepares list of recommended threshold limit values (TLV) for about 600 such chemicals. Three different categories of air concentrations, expressed in parts per million (ppm) or milligrams per cubic meter (mg/m^3) have been elaborated to cover various exposure conditions.

1. Threshold limit value—time-weighted average (TLV-TWA) is the concentration for a normal 8-hour work/day or 40-hour work/week to which workers may be repeatedly exposed without adverse effect.
2. Threshold limit value—short-term exposure limits (TLV-STEL) is the maximum concentration (a value greater than the TLV-TWA) that should not be exceeded at any time during a 15-minute exposure period.
3. Threshold limit value—ceiling (TLV-C) is the concentration that should not be exceeded even instantaneously. These guidelines are reevaluated as new information becomes available.

ENVIRONMENTAL TOXICOLOGY

Environmental toxicology deals with the poten-tially deleterious impact of chemicals, present as pollutants of the environment, to living organisms.[1,2,6] Environmental toxicology is defined as the science that deals with effects of pollutants on environment and wildlife.[10] The term "environment" includes all the surroundings of an individual organism, but particularly the air, soil, and water. A "pollutant" is a substance that occurs in the environment, at least in part as a result of human activity, and which has a deleterious effect on living organisms. While humans are considered a target species of particular interest, other terrestrial and aquatic species are of considerable importance as potential biologic targets.

Air pollution is a product of industrialization, technological and biological development, increased urbanization and indoor sources. Humans may also be exposed to chemicals used in the agricultural environment as pesticides or ingredients in food products.[2] In many people's minds, air pollution is only associated with contamination of urban air from automobile exhaust and industrial effluents. However, in developing countries, the problem of indoor air pollution far outweighs the ambient air pollution.[6]

Indoor air pollution in India is a major environmental and health concern. A recent report of the World Health Organization (WHO) asserts that a pollutant released indoors is one thousand times more likely to reach people's lung than pollutant released outdoors.[6,7] It has been estimated that about half a million people die each year from indoor air pollution.[7] The indoor pollutants are derived from four sources namely: (i) combustion (ii) building material (iii) the ground under the building (iv) bioaerosols.[6] In developed countries the most important indoor air pollutants are radon, asbestos, volatile organic compounds, pesticides, heavy metals, animal dander, mites, moulds and environmental tobacco smoke.[6,7]

The Food and Agriculture Organization (FAO) and the World Health Organization (WHO), Joint Expert Commission on Food Additives adopted the term acceptable daily intake (ADI) to denote "the daily intake of a chemical which, during an entire lifetime, appears to be without appreciable risk on the basis of all the known facts at the time". After evaluation of the pertinent scientific data, the FAO/WHO periodically lists ADI values (expressed in milligrams per kilogram of body weight per day) for many pesticides and food additives that may enter the human food chain. These guidelines are re-evaluated when new information becomes available.

ECOTOXICOLOGY

Ecotoxicology has evolved relatively recently as an extension of environmental toxicology.[1,2] Ecotoxicology is defined as a specialized area of environmental toxicology dealing with effects of pollutants on population dynamics in an ecosystem.[10] It is concerned with the toxic effects of chemical and physical agents on living organisms, especially in populations and communities within defined ecosystems; it includes the transfer pathways of those agents and their interactions with the environment. Traditional toxicology is concerned with toxic effects on individual organisms, has no important impact on populations or an ecosystem. Thus, the terms "environmental toxicology" and "ecotoxicology" are not interchangeable.

TOXICITY, HAZARD AND RISK

Toxicity

Toxicity is the ability of a chemical agent to cause injury. It is a qualitative term. Whether or not these injuries occur depends on the amount of chemical absorbed (severity of the exposure, dose).[1,2]

Hazard

Hazard on the other hand, is the likelihood that injury will occur in a given situation or setting, the conditions of use and exposure are primary considerations. To assess hazard, one needs to have knowledge about both the inherent toxicity of the substance (qualitative aspect) and the amounts to which individuals are liable to be exposed (quantitative aspect). Humans can safely use potentially toxic substances when the necessary conditions minimising absorption are established and respected. The presence of a potentially toxic substance in the work place or in the environment does not necessarily mean that a hazardous situation exists.[1,2]

Risk

Risk is defined as the expected frequency of the occurrence of an undesirable effect arising from extrapolation from the observed relationships to the expected responses at doses occurring in actual exposure situations. The quality and suitability of the biologic data used in such estimates are major limiting factors. A number of mathematical models have been devised and are often used for calculating risk of carcino-genesis; they may also be used to assess the risk involved with other forms of toxicity.

Routes of Exposure

The route of entry for chemicals into the body differs in different exposure situations. In the industrial setting, inhalation is the major route of entry. The transdermal route is also quite important, but oral ingestion is a relatively minor route. Consequently, preventive measures are largely designed to eliminate absorption by inhalation or by topical contact. Atmospheric pollutants of water and soil, oral ingestion are the principal routes of exposure for humans.

Duration of Exposure

Toxic reactions may differ qualitatively depen-ding on the duration of the exposure.[1-5] A single exposure or multiple exposures occurring over 1 to 2 days represent(s) acute exposure. Multiple exposures continuing over a longer period of time represent a chronic exposure. In the occupational setting, both acute (e.g. accidental discharge) and chronic (e.g. repetitive handling of a chemical) exposures may occur, whereas with chemicals found in the environment (e.g. pollutants in ground water), chronic exposure is more likely. Society is also concerned with the possible harmful effects of contact with small concentrations of chemicals over long periods of time; this type of chronic situation is called low-level, long-term exposure. The appearance of the toxic effects after acute exposure may appear rapidly or after a variable interval; the latter is called delayed toxicity. With chronic exposures, the toxic effect may not be discernible until after several months of repetitive exposures. The harmful effect resulting from either acute or chronic exposure may be reversible or irreversible. The relative reversibility of the toxic effect depends on the recuperative properties of the affected organ.

Presence of Mixtures

Humans normally come in contact with several (or many) different chemicals concurrently or sequentially.[3-5] This complicates the assessment of potentially hazardous situations encountered in the workplace or in the environment. The resulting biologic effect of combined exposure to several agents can be characterized as additive, supra-additive (synergistic), or infra-additive (antagonistic). Another type of interaction, potentiation (a special form of synergism), may be observed. In case of potentiation, one of two agents is increased. All these types of interactions have been observed in humans. In the absence of contrary evidence, one usually assumes that the toxic effects of mixtures of chemicals are likely to be additive.

Environmental Considerations

Certain chemical and physical characteristics are known to be important for estimating the potential hazard involved for environmental toxicants. In addition to information regarding effects on different organisms, knowledge about the following properties is essential to predict the environmental impact.[1,10]

The facts like,degradability of the substance, its mobility through air, water, and soil, whether or not its bioaccumulation occurs; and its transport and biomagnification through food chains need to be considered. Chemicals that are poorly degraded (by abiotic or biotic pathways) exhibit environmental persistence and thus can accumulate. Lipophilic substances tend to bioaccumulate in body fat, resulting in tissue residues. When the toxicant is incorporated into the food chain, biomagnification occurs as one species feeds upon the other and concentrates the chemical. The pollutants that have the widest environmental impact are poorly degradable, are relatively mobile in air, water, and soil, and exhibit bioaccumulation potential of organic chemicals found in the environment. In ecotoxicology there are three interacting components: the toxicants, the environment, and the organisms (community, population, or ecosystem). Ecotoxicologic studies are designed to determine emission and entry of the toxicant in the abiotic environment, including distribution, entry and fate of the toxicant in the biosphere, and the quantitative and qualitative toxic consequences to the ecosystem.[1-6]

CLASSIFICATION OF OCCUPATIONAL/ ENVIRONMENTAL POISONS

Occupational/environmental poisons are classified into five types (Table 31.8).[1,10-12.]

Air Pollutants

The major substances that account for about 98 per cent of air pollution are carbon monoxide, sulfur dioxides, nitrogen dioxide and ozone.[1,2,4,9,10-12] Sources of these chemicals include transportation, industry, generation of electric power, space heating, and refuse disposal. Considerable progress has been made in our understanding of the chemistry of urban air pollution. Sulfur dioxide and smoke resulting from incomplete combustion of coal has been associated with acute adverse effects, particularly among the elderly and individuals with pre-existing cardiac or respiratory disease. The association of acute adverse effects, other than severe irritation of the eyes, is less striking with the "oxidising or photochemical type" of pollution

Table 31.8: Classification of occupational/ environmental poisons

Types	*Examples*
Air pollutants	Carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3).
Solvents	Halogenated aliphatic hydrocarbons, aromatic hydrocarbons
Insecticides	Chlorinated hydrocarbon insecticide, organophosphorous insecticide, carbamate insecticide, and botanical insecticide.
Herbicides	Chlorophenoxy herbicide, bipyridial herbicide.
Environmental pollutants	Polychlorinated biphenyls.

Table 31.9: Threshold limit values (TLV) of common air pollutants and solvents

Compounds	*TLV (ppm)* *TWA*[1]	*STEL*[1]
Benzene	010.0	N/A
Carbon monoxide	025.0	N/A
Carbon tetrachloride	005.0	N/A
Chloroform	010.0	N/A
Nitrogen dioxide	003.0	005.0
Ozone	000.1	N/A
Sulphur dioxide	002.0	005.0
Tetrachloroethylene	050.0	200.0
Toluene	050.0	N/A
1,1,1-Trichloroethane	350.0	450.0
Trichloroethylene	050.0	200.0

N/A – None assigned

[1] See text for definitions

Table 31.10: Chlorinated hydrocarbon insecticides

Chemical class compounds	*Toxicity Rating*[1]	*ADI*[2]
DDT and analogues	4	0.005
Methoxychlor	3	0.1
Tetrachloro-diphenylethane (TDE)	3	–
Benzene hexachlorides (BHC; hexachloro-cyclohexane)	4	–
Lindane cyclodienes	4	0.01
Aldrin	5	0.0001
Chlordane	4	0.001
Dieldrin	5	0.0001
Heptachlor toxaphenes	4	0.0005
Toxaphenes (camphechlor)	4	–

[1] Toxicity rating: Probable human oral lethal dosage for class 3 = 500-5000 mg/kg, class 5 = 5-50 gm/kg (see Gosselin reference)
[2] ADI = Acceptable daily intake (mg/kg body weight/d).

(hydrocarbons, nitrogen oxides), which has been implicated as a contributing factor in bronchitis, obstructive ventilatory disease, pulmonary emphysema, bronchial asthma, and lung cancer.

Carbon Monoxide

Carbon monoxide (CO) is a colorless, tasteless, odorless, and nonirritating gas, a by product of incomplete combustion. The average concentration of CO in the atmosphere is about 0.1 ppm; in heavy traffic, the concentration may exceed 100 ppm.[12,13] The recommended threshold limit values (TLV-TWA and TLV-STEL) are shown in Table 31.9.

Sulphur Dioxide

Sulphur dioxide (SO_2) is a colorless, irritant gas generated primarily by the combustion of sulphur containing fossil fuels. The TLV-TWA and TLV-STEL are given in Table 31.9.

Nitrogen Oxides

Nitrogen dioxide (NO_2) is a brownish irritant gas sometimes associated with fires. It is formed also from fresh silage; exposure of farmers to NO_2 in the confines of a silo can lead to "silo filler's disease." The 1993 TLV-TWA and TLV-STEL values are show in Table 31.9.

Ozone

Ozone (O_3) is a bluish irritant gas that occurs normally in the earth's atmosphere, where it is an important absorbant of ultraviolet light. In the workplace, it can occur around high-voltage electrical equipment and around ozone-producing devices used for air and water purification. It is also an important oxidant found in polluted urban air refer Table 31.9 for TLV-TWA and TLV-STEL values.

SOLVENTS

Halogenated Aliphatic Hydrocarbons

These agents find wide use as industrial solvents, degreasing agents, and clearing agents.[1,2] The substances include carbon tetrachloride, chloroform, trichloroethylene, tetrachloroethylene (perchloroethylene), 1,1,1-trichloroethane methyl chloroform refer Table 31.9 for recommended threshold limit values.

Aromatic Hydrocarbons

Benzene is widely used for its solvent properties and as an intermediate in the synthesis of other chemicals. The recommended TLV-TWA and TLV-STEL values are given in Table 31.9. Toluene (methylbenzene) does neither possess the myelotoxic properties of benzene, nor has it been associated with leukemia. However, it is a central nervous system (CNS) depressant (Refer Table 31.9 for the TLV-TWA and TLV-STEL values).

INSECTICIDES

Chlorinated Hydrocarbon Insecticides

These agents are usually classified into four groups: DDT (chlorophenothane) and its analogues, benzene hexachlorides, cyclodienes, and toxaphenes[1, 2, 4] (Table 31.10). They are aryl, carbocyclic, or heterocyclic compounds containing chlorine substituents. The individual compounds differ widely in their biotransformation, and capacity for storage; toxicity and storage is not always correlated. They can be absorbed through the skin as well as by inhalation or oral ingestion. There are, however, important quantitative differences between the various derivatives: the skin poorly absorbs DDT in solution, whereas dieldrin absorption from the skin is very efficient.

The chlorinated hydrocarbon insecticides are considered "persistent" chemicals. Degradation is quite slow when compared to other insecticides, and bioaccumulation, particularly in aquatic ecosystems, is well documented. Their mobility in soil depends on the composition of the soil, whereas adsorption is poor in sandy soils. Once adsorbed, they do not readily desorb. Because of their environmental impact, use of the chlorinated hydrocarbon insecticides has been largely curtailed in North America and Europe. Some of them are still used, however, in tropical countries.

Organophosphorous Insecticides

These agents, (some of which are listed in Table 31.11), are utilized to combat a large variety of pests. They are useful pesticides when in direct contact with insects or when used as 'plant systemics', where the agent is trans-located within the plant and exerts its effects on insects that feed on the plant. Some of these agents are used in human and veterinary medicine as local or systemic antiparasitics or in circumstances in which prolonged inhibition of cholinesterase is indicated. The compounds are absorbed by the skin as well as by the respiratory and gastrointestinal tracts. Biotransformation is rapid, particularly when compared to the rates observed with the chlorinated hydrocarbon insecticides.

Table 31.11: Organophosphorus insecticides

Compounds	*Toxicity rating*[1]	*ADI*[2]
Azinphosmethyl	5	0.0025
Chlorfenvinphos	–	0.002
Diazinon	4	0.002
Dichlorvos	–	0.004
Dimethoate	4	0.020
Fenitrothion	–	0.005
Leptophos	–	–
Malathion	4	0.020
Parathion	6	0.005
Parathion-methyl	5	–
Trichlorfon	4	0.010

[1] Toxicity rating = Probable human oral lethal dosage for class 4 = 50-500 mg/kg. (See Gosselin reference)

[2] ADI = Acceptable daily intake (mg/kg body weight/d).

Table 31.12: Carbamate insecticides

Compounds	*Toxicity rating*[1]	*ADI*[2]
Aldicarb	6	0.005
Aminocarb	5	–
Carbaryl	4	0.01
Carbofuran	5	0.01
Dimetan	4	–
Dimetilan	4	–
Isolan	5	–
Methomyl	5	–
Propoxur	4	0.02
Pyramat	4	–
Pyrolan	5	–
Zectran	5	–

[1] Toxicity rating = Probable human oral lethal dosage for class

[2] ADI = Acceptable daily intake (mg/kg body/weight/d)

In mammals as well as insects, the major effects of these agents are inhibition of acetyl cholinesterase, because of phosphorylation of the esteratic site. The signs and symptoms that characterized acute intoxication are due to inhibition of this enzyme, resulting in the accumulation of acetylcholine. Some of the agents also possess direct cholinergic activity.

Organophosphorous insecticides are not considered to be "persistent" pesticides. As a class they are considered to have a small impact on the environment inspite of their acute action on organisms.

Carbamate Insecticides

These compounds (Table 31.12) inhibit acetyl cholinesterase by carbamylation of the esteratic site. Thus, they possess the toxic properties associated with inhibition of this enzyme as described for the organophosphorus insecticides. The carbamate insecticides are considered to be 'no persistent' pesticides in the environment, and they are thought to exert a small impact on the environment. Generally speaking, the clinical effects due to carbamates are of shorter duration than those observed with organophosphorus compounds. The range between the doses that cause minor intoxication and those that result in lethality is larger with carbamates than that observed with the organophosphorus agents. Spontaneous reactivation of cholinesterase is more rapid after inhibition by the carbamates.

Botanical Insecticides

These insecticides derived from natural sources include nicotine, rotenone, and pyrethrum. Nicotine is obtained from the dried leaves of Nicotiana tobacum and Nicotiana rustica plants. It is rapidly absorbed from mucosal surfaces; the free alkaloid, but not the salt, is readily absorbed from the skin. Nicotine reacts with the acetylcholine receptor of the postsynaptic membrane (sympathetic and parasympathetic ganglia, neuromuscular junction), resulting in depolarization of the membrane. Toxic doses cause stimulation, rapidly followed by blockade of transmission. Treatment is directed toward maintenance of vital signs and suppression of convulsions.

Rotenone is obtained from Derris elliptica, Derris mallaccensis, Lonchocarpus utilis, and Lonchocarpus urucu. The oral ingestion of rotenone produces gastrontestinal irritation. Conjunctivitis, dermatitis, pharyngitis, and rhinitis can also occur. Treatment is symptomatic.

Pyrethrum consists of six known insecticidal esters: pyrethrin I, pyrethrin II, cinerin I, cinerin II, jasmolin I, and jasmolin II. Synthetic pyrethroids account for about 30 per cent of worldwide insecticide usage. Pyrethrum may be absorbed after inhalation or ingestion; absorption from the skin is not significant. The esters are extensively biotransformed. Pyrethrum insecticides are not highly toxic to mammals. When absorbed in sufficient quantities, the major site of toxic action is the central nervous system (CNS); excitation, convulsions, and tetanic paralysis can occur by a sodium channel mechanism resembling that of DDT. Treatment is with anticonvulsants. The most frequent injury reported in human's especially contact dermatitis, results from the allergenic properties of the substance. Cutaneous paresthesias have been observed in workers spraying synthetic pyrethroids in China resulting in marked effects on the central nervous system, including convulsions.

HERBICIDES

Chlorophenoxy Herbicides

2,4-Dichlorophenoxyacetic acid (2,4–D), and their salts and esters are the major compounds of interest as herbicides used for the destruction of weeds. They have been assigned toxicity rating of 4 or 3, respectively, which place the probable human lethal dosages at 50 to 500 or 500 to 5000 mg/kg, respectively.

In humans, 2,4-Dichlorophenoxyacetic acid (2,4–D), in large doses can cause coma and generalized muscle hypotonia which may persist for several weeks. With 2, 4–D, coma may occur, but the muscular dysfunction is less evident. In laboratory animals, signs of liver and kidney dysfunction have also been reported. There is limited evidence suggesting that occupational exposure to phenoxy herbicides is associated with an increased risk of non-Hodgkin's lymphoma. The evidence for soft-tissue sarcoma, however, is considered equivocal.

The toxicological profile for these agents, particularly with 2,4–D, has been confusing because of the presence of chemical contaminants (dioxins) produced during the manufacturing process. The presence of 2, 3, 7, 8-tetra-chlorodibenzopdioxin (TCDD) is believed to be largely responsible for the teratogenic effects detected in some animal species as well as the contact dermatitis and chloracne observed in workers involved in the manufacture of 2,4–D. In spite of exhaustive studies, it has been very difficult to document long-term toxic effects of TCDD in humans.

TCDD as a contaminant in herbicides has stimulated interest in its possible carcinogenic property, in humans. Soft tissue sarcomas and malignant lymphomas have received considerable attention. A causal role for TCDD in malignant melanoma, however, appears unlikely, and the evidence for soft tissue sarcoma is considered unconvincing, a role for TCDD in the etiology of other cancers remains to be evaluated.

Bipyridyl Herbicides

Paraquat is the most important agent of this class. It has been given a toxicity rating of 4, which places the probable human lethal dosage at 50 to 500 mg/kg. A number of lethal human intoxications (accidental or suicidal) have been reported.

In humans, the first signs and symptoms after oral exposure are attributable to gastrointestinal irritation (hematemesis and bloody stools). Within a few days, however, respiratory distress may appear (delayed toxicity) with the development of congestive hemorrhagic pulmonary oedema accompanied by widespread cellular proliferation. Evidence of hepatic, renal, or myocardial involvement may also be present. The interval between ingestion and death may be several weeks. Because of the delayed pulmonary toxicity, prompt removal of para-quat from the digestive tract is important. Gastric lavage, the use of cathartics, and the use of adsorbents to prevent further absorption have all been advocated; after absorption, treatment is successful in fewer than 50 per cent of cases. Oxygen should be used cautiously to combat dyspnea or cyanosis, as it may aggravate the pulmonary lesions. Patients require prolonged observation, because the proliferative phase begins 1 to 2 weeks after ingestion.

ENVIRONMENTAL POLLUTANTS

The polychlorinated biphenyls (PCBs) have been used in a large variety of applications as dielectric and heat transfer fluids, plasticizers, wax extenders, and flame-retardants. Their industrial use and manufacture in the USA was terminated by 1977.[1,4,8,12] Unfortunately, they persist in the environment.

The products used commercially were actually mixtures of PCB isomers and homologues containing 12 to 68 per cent chlorine. These chemicals are highly stable and highly lipophilic, poorly metabolized, and very resistant to environmental degradation, therefore they bioaccumulate in food chains. Food is the major source of PCB residues in humans.

A serious exposure to PCBs, lasting several months, occurred in Japan in 1968 as a result of cooking oil contamination with PCB-containing transfer medium (Yusho disease). Possible effects on the fetus and the development of offspring of poisoned women were reported.[12] It is now known that the contaminated cooking oil contained not only PCBs but also polychlorinated dibenzofurans (PCDFs) and polychlorinated quaterphenyls (PCQs).

Consequently, the effects that were initially attributed to the presence of PCBs are now thought to have been largely caused by the other contaminants.

Workers occupationally exposed to PCBs have exhibited the following clinical signs:

- Dermatologic problems (chlorance, folliculitis, erythema, dryness, rash, hyperkeratosis, and hyperpigmentation),
- Some present with hepatic involvement and elevated plasma triglycerides are also observed in some.
- Effects of PCBs alone on reproduction and development, as well as carcinogenic effects, have yet to be established in humans, even though some subjects have been exposed to very high levels of PCBs.

The bulk of the evidence from human studies indicates that PCBs pose little hazard to human health except in situations where food is contaminated with high concentration of these congeners.

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CLINICAL MANAGEMENT OF POISONING

Management of poisoning cases includes diagnosis of poisoning, duties of doctor towards suspected poisoning case, general measures and specific measures.

DIAGNOSIS OF POISONING

A standard clinical examination is carried out in every poisoned patient:[1,2,4]

- Record the vitals — This includes recording of pulse, respiratory rate, blood pressure, body temperature and pupillary manifestations.
- Note the odor — A number of toxins have a characteristic odor (Table 31.13) and may help in detection of poison consumed. However, the odour may be subtle and the ability to smell the odor may vary (e.g. only about 50 per cent of the general population can smell the 'bitter almond' odor of cyanide).
- Note the color of urine Observing the colour of urine passed, may also give vital clue towards the diagnosis of type of poison (Table 31.14).

- Clinical features–Certain clinical manifestations which point to the substance responsible for poisoning in an unconscious or unwell patient are mentioned in Table 31.15.

Note: In addition to above particular care must be taken to look for any needle marks or previous evidence of self harm/ suicide, e.g. razor cut marks on forearms, neck, etc.

Certain Condition Arousing Suspicion of Poisoning

Enumerated below are certain conditions observed in living persons, which should arouse suspicion of poisoning to a physician:

- Appearance of symptoms suddenly in a healthy person or several people at a time.
- Sudden worsening of symptoms in a sick person, who was receiving correct treatment and showing significant improvements or progress.
- Appearance of symptoms soon after ingestion of food or medicine.
- Manner and course of symptoms, e.g. symptoms that run steadily, a downhill course to death or an uphill course to total recovery.
- Detection of poison in remnants of food or vomitus.
- Even on reporting of "no poison" on analysis of the suspected materials, the suspicion of poisoning cannot be ruled out for the reasons such as:
  - Poison may already be eliminated from the body by vomiting or metabolized and excreted through natural channels, leaving no evidence.
  - The analysis technique might be faulty.
  - The case may be a real case of disease and not a poisoning case at all.

DUTIES OF A DOCTOR IN A POISONING CASE

Refer Medicolegal Aspects of Poisoning.

General Measures

This depends on whether the patient is unconscious or conscious.

Dealing with Unconscious Patient

First ensure that the airway is clear and the patient is breathing adequately, circulation is not compromised and there is no depression of CNS. This is known as ABCD of assessing the patient.[1]If the patient is stable then proceed to take history and assessing the depth of unconsciousness. In a poisoned patient, this is important and is preferably done as per the Reeds Classification/ Edinburgh Method (Table 31.16).[2] The Glasgow Coma Scale[3] (Table 31.17) is the most frequently used in assessment of the degree of impaired consciousness, though remarkably it has never been validated for use in poisoned patients.[1,2]

When patient is unconscious and no history is available, the diagnosis of poisoning depends on exclusion of other causes of coma (such as meningitis or encephalitis, trauma, subarachnoid hemorrhage, intracranial haemorrahage, hypogylcaemia, diabetic ketoacidosis, uraemia, encephalopathy, etc.) and consideration of circumstantial evidence.

Dealing with Conscious Patients

Take a brief history. In the vast majority of cases, the diagnosis of acute poisoning is obtained from the history given by the patient.[1,2] However, doctors need to be aware that patients may not always furnish them with the correct answer. This may be because they do not always know what they have taken, not least because they may have been under the influence of alcohol or the drug itself at the time of ingestion or while narrating the

Table 31.13: Characteristic odour of toxins

Odors	*Causes*
Acetone (sweet, like russet apples)	Isopropyl alcohol, acetone, lacquer, chloroform
Acrid (pear-like)	Paraldehyde, choral hydrate
Alcohol (fruit-like)	Alcohol, isopropyl alcohol
Ammoniacal (pungent)	Uremia
Bitter almonds	Cyanide
Burnt rope	Cannabis
Fish or raw liver	Zinc phosphate
Garlic	Arsenic, selenium, thallium, phosphorus, parathion, malathion, thallium, selenium, zinc phosphide
Kerosene like	Organophosphorus, endrin
Phenolic smell	Carbolic acid
Rotten eggs	Hydrogen sulphide, mercaptans, disulfiram
Shoe polish like	Nitrobenzene
Sweet pungent	Ether
Wintergreen	Methyl-salicylate

Table 31.14: Urine colour caused by certain toxins

Urine colours	*Causes*
Green or blue	Methylene blue, e.g. fish tank tablets
Grey-black	Phenols, cresols
Opaque appearance which settles on standing	Primidone crystals
Orange or orange-red	Rifampicin, iron (especially after giving desferrioxamine)

Table 31.15: Certain specific clinical features diagnostic of poisons and poisoning

Clinical features	*Possible causes*
Pupillary constriction (Miosois)	Barbiturates, caffeine, carbamates, carbolic acid (phenol), clonidine, methyl dopa, nicotine, opiates, organophosphates, parasympathomimetics.
Pupillary dilatation (Mydriasis)	Alcohol (constricted in coma), amphetamines, antihistamines, benzodiazepines, carbon monoxide, cocaine, cyanide, datura (atropine), ephedrine.
Hippus (alternate constriction and dilatation of pupils)	Aconite, alcohol, barbiturates.
Nystagmus	Alcohol, barbiturates, carbamazepine, phencyclidine, phenytoin.
Pinpoint pupils, and reduced respiratory rate	Opioids, cholinesterase inhibitors (organophosphorus or carbamate insecticides), clonidine, phenothiazines, Pontine haemorrhage.
Cyanosis	Any CNS depressant or agent causing methemoglobinaemia.
Needle tracks, pinpoint pupils, and reduced respiratory rate	IV Opioids.
Dilated pupils or mid-point pupils, and reduced respiratory rate	Benzodiazepines
Dilated pupils, tachycardia	• Tricyclic antidepressants – dry mouth, warm peripheries, may also be twitchy or have seizures • Amphetamines, ecstasy, cocaine – may also be hallucinating or agitated • Anticholinergic drugs such as benzhexol, benztropine – may also have hyperreflexia and myoclonus • Antihistamines – may also be drowsy
Increased salivation	Organophosphorus or carbamate insecticides
Cerebellar signs; nystagmus, ataxia	Anticonvulsants (particularly phenytoin, carbamazepine), alcohol
Extrapyramidal signs	Phenothiazines, haloperidol, metoclopramide.
Seizures	Tricyclic antidepressants, theophylline, antihistamines, anticonvulsants, non-steroidal drugs, phenol, phenothiazines, isoniazid, cocaine, carbon monoxide, organophosphorus insecticides, strychnine.
Bradycardia	Beta-blockers, calcium antagonists (not dihydropyridines), digoxin, opioids, organophosphorus insecticides, centrally acting alpha agonists, e.g. clonidine
Hyperthermia	Lithium, tricyclic antidepressants, anticholinergics, antihistamines
Hypothermia	• Amphetamines, ecstasy, cocaine (also hypertension, tachycardia, agitation, rhabdomyolysis) • Neuroleptic malignant syndrome (also confusion, fluctuating consciousness, rigidity, tremor, autonomic instability, sweating, rhabdomyolysis) • Serotonin syndrome (also agitation, clonus, tremor, hyperreflexia, sweating, tachycardia) • Salicylates including aspirin (also tachycardia, metabolic acidosis, restlessness, hyperventilation)
Abdominal cramps, diarrhoea, tachycardia, restlessness, hallucinations	Withdrawal from: Alcohol, benzodiazepines, opioids
Cardiac arrhythmia	Amphetamine, arsenic, carbon monoxide, choral hydrate, cocaine, cyanide, digitalis, MAO inhibitors, phenol, phenothiazine, physostigmine, quinine
Severe muscle weakness	Nicotine, curare, succinylcholine, neostigmine, botulism
Hypertension	Amphetamine, ephedrine, cocaine, clonidine, MAO inhibitors, thyroid hormones
Hypotension	Cyanide, alcohol, digitalis, carbon monoxide, narcotics, barbiturates

Table 31.16: The Reed's classification of comatose patient

Unconscious level	*Clinical response*
Group 0	Arousable
Group 1	Respond to painful stimuli and have intact reflexes
Group 2	Do not respond to painful stimuli — most reflexes are normal
Group 3	Do not respond to painful stimuli — most reflexes are absent
Group 4	Deeply comatose, with respiratory and/or circulatory failure

Note: An example of minimal painful stimulus is to pinch lightly, while a suitable maximally painful stimulus is to rub the sternum with knuckles of clenched fist

history. A few patients deliberately mislead doctors, but in our experience this is very rare, except perhaps in the drug abuser population.

Full details of how many and what type of substance that has been taken must be recorded, as well as timing of ingestion or exposure. Ask the patient why the overdose, if any, was taken and take time to listen to the explanation. Often reasons include relationship difficulties, work or school related commitments, problems of addiction, psychiatric illness or bereavement. Beware of those claiming 'accidental overdose'. Whilst this clearly can

Table 31.17: The Glasgow coma scale [3]

Clinical responses	*Scores*
Eye opening:	E4
Spontaneously	4
To speech	3
To pain	2
None	1
Best verbal response:	V5
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Best motor responses:	M6
Obeys commands	6
Localisation to pain	5
Normal flexion to pain	4
Spastic flexion	3
Extension to pain	2
None	1

The responsiveness of the patient is expressed by summation of figures. Thus coma score (E + V + M) = 3 to 15, Max score = 15 (Conscious); Min score = 3 (Deeply comatose)

Reproduced from Teasdale G and Jennett B, Assessment of Coma and Impaired Lancet 1974, 2: 81-84.

Warning: Beware patients feigning unconsciousness. Remember to record best response for GCS.

Table 31.18: Routes of poisoning and methods of removal

Poisoning	*Suggested methods of removal*
Ingested	Gut decontamination*
Inhaled	Breath fresh air, artificial respiration
Injected	Give first aid, followed by specific antidotes, diuretics, dialysis, etc
Contact	Wash with water, neutralize with antidotes, etc

* Discussed below in detail

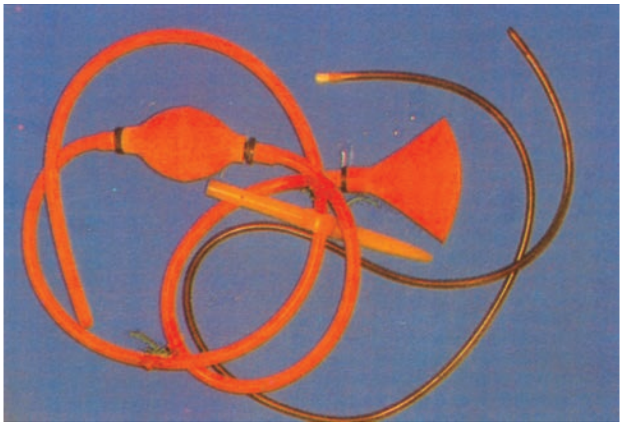

Fig. 31.1: Stomach tube (Boa's tube/gastric lavage tube) and Ryle's tube

occur, e.g. Paracetamol poisoning after toothache, in general all patients presenting with poisoning should undergo psychiatric evaluation.[1]

Details of the past medical history should be recorded. In particular a history of asthma, jaundice, drug abuse (by which routes), head injury, epilepsy, cardiovascular problems and previous psychiatric history or selfharm should be taken. It is important to ask about allergies and alcohol history. Family problems and social history are very important but often missed out in undue haste to clerk the patient.

Specific Measures

Specific measures are in relation to removal of the poison from the body. It depends on whether the poison is ingested, inhaled, injected or a contact poison. Table 31.18 presents an idea regarding routes of poisoning, and methods of removal.

However, toxicologists recommend four principal lines of treatment, and this includes decontamination, emesis, elimination by natural channels, administration of antidotes, and symptomatic line of treatment. Each of these is discussed in detail.

DECONTAMINATION

The majority of patients who present after an overdose require only meticulous supportive care. However, it is important that patients are observed closely for signs of deterioration. Overall the mortality from acute poisoning is less than 1 per cent and it is in patients who have taken significant overdoses that further measures such as decontamination may be required.[1,4]

Decontamination refers to skin/eye decontamination, gut evacuation and administration of activated charcoal. However, removal of poison from the gastrointestinal tract demands maximum attention in managing an overdose case and this constitutes various gut decontamination procedures such as gastric lavage, emesis, administration of activated charcoal, catharsis, and whole bowel irrigation. These procedures should only be used in patients who untreated would risk serious poisoning, as they can be associated with complications.[1,4]

Gastric Lavage

Gastric lavage (stomach washout) should only be undertaken if the patient has ingested a potentially life-threatening amount of a poison and presents within 1 hour of the ingestion. If performed later than this the amount of poison removed is insignificant and lavage can actually make the situation worse by pushing unabsorbed poison into the small intestine.[2,4] Always ensure that the patient's airway is adequately protected before gastric lavage.

The Equipment

Gastric Lavage/Stomach WashEquipment used for the purpose is known as Stomach Tube/ Boa's Tube. Table 31.19 provides brief description of the equipment and Figure 31.1, and Figures 31.2A and B presents the coloured photograph and line drawing of the stomach tube respectively.

Other Requirements

- Oxygen.
- Powerful suction equipment.
- KY jelly.
- A disposable gastric lavage tube (14 mm diameter [36-40 F] in adults)/ red rubber gastric lavage tube/Boa's tube.
- A bucket.
- A jug of 250-500 ml capacity.
- Protective clothes/covering and gloves for the staff.
- Minimum two members of staff.
- Oxygen saturation monitor with probe.
- Cuffed endotracheal tube.
- Gloves, apron and boots for the person performing the procedure.

Table 31.19: Description of the stomach tube (Boa's tube)

Structure/ parts	*Details*
Material	Flexible red rubber
Length	1.5 meter (adult)
Diameter	0.5 cm
Upper end	Has a funnel
Lower end	Blunt and round with 1-2 lateral openings
Mark "50"	On the tube, seen at a distance of exactly 50 cm from lower end (this roughly corresponds to the distance from incisor teeth above to the cardiac/upper end of the stomach below)
Suction bulb	To apply suction and siphon out or aspirate stomach contents
Mouth gag	To prevent the biting of the tube during gastric lavage procedure

Procedure

Gastric Lavage procedure includes certain specific steps[1,2] such as:

Position of the Patient

Proper positioning of the patient is to prevent regurgitation of fluids during lavaging. Two methods adopted:

- Lying left lateral position is often preferred (Fig. 31.2B). The foot of the bed or trolley should be raised by 20 cm.
- Lying supine on the table with head extended and neck stretched by bringing the head at lower level than body at the edge of the head end of the table is also accepted.

Introduction of the Tube

- Remove artificial denture if any in the mouth.
- If the tube is disposable gastric lavage tube, lubricate it with KY jelly
- If the tube is of red rubber material, warm it up to body temperature and lubricate it by applying olive oil.
- Tube may then be inserted/passed by asking the patient to swallow gently. Another approved method is by using a tongue depressor/two fingers, depress the tongue and insert the free end of the tube (which is passed through mouth gag) along the fingers/tongue depressor into the pharynx.
- Push the tube down further into the esophagus till the mark 50 cm on the tube corresponds with incisor teeth. Next remove tongue depressor or fingers and fix the mouth gag so that the teeth do not bite the tube.

Confirmation of Tube in the Stomach

Ensure it is not passed into the airway by keeping the head forward in a flexed position. Confirm the end of the tube is in the stomach by aspirating or blowing air in and auscultating over the stomach. Confirmation may be also done by holding the inverted funnel end on a water column which yields a few air bubbles into the water continuously if it is in the respiratory tract, while if bubbling stops within few seconds, it is in the stomach (stomach also contains little air normally).

Washing/Lavaging the Stomach

Hold the funnel in hand and pour lavage fluids (not more than 250 ml at a time). Now raise the funnel higher up. It becomes empty as the fluid flows into the stomach. Allow few minutes for fluid to act in the stomach and then lower the funnel end into an empty bucket below. Apply suction on the bulb, which in turn would siphon out the contents of stomach into the bucket. Repeat the procedure to continue lavaging. At times specific gastric lavage solutions are preferred rather than water / saline for certain specific poisons (Table 31.20).

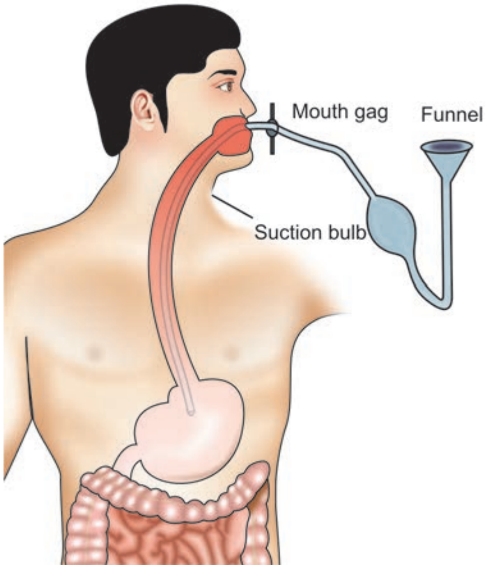

Fig. 31.2A: Line drawing of stomach tube /Boa's tube

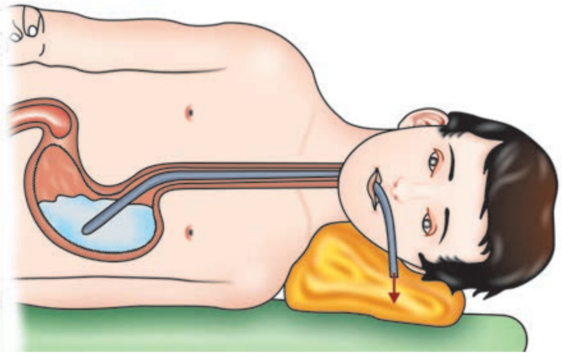

Fig. 31.2B: Line drawing showing left lateral position for stomach washes

Indication to stop lavaging — Lavaging is to be done continuously. The indication for gastric lavage to stop is that the fluid siphoned out is clear and odorless and is of same nature as the fluid poured.

Children — A Ryle's tube is made use of in place of gastric lavaging.

Complications

Gastric lavage is an invasive procedure and has its own merits and demerits. Merits being known already let's try to analyze

Table 31.20: Specific solutions for gastric lavage

Solutions	*Poisons*
Luke warm water or saline	Known/ unknown poisons
Potassium permanganate (1:5000 or 1: 10000)	Poisons which are oxidisable such as alkaloids
Sodium thiosulphate (25%)	Cyanides
Castor oil and warm water (1:2)	Carbolic acid (phenol)
Calcium gluconate	Oxalates
Desferrioxamine (2 gm in 1 litre of water)	Iron

Table 31.21: Gastric lavage complications and how to avoid them

Complications	*Solutions*
rupture of oesophagus/ stomach (rarely)	Do not use force to pass/introduce the tube
Aspiration pneumonitis	Care with water and contents, adequate airway protection, suction apparatus must be present and working/perform endotracheal intubation.
Lipoid pneumonia	Do not wash out petroleum distillates, e.g. paraffin, petrol, furniture polishes
Hypoxia, cardiac arrhythmias	Monitor oxygen saturation, give supplemental oxygen via nasal prongs

the demerits in order to provide relief when observed. Table 31.21 highlights the possible complications of gastric lavage and also the solutions suggested.[5,6] Gastric lavage should never be undertaken as a punitive procedure. Do not wash out patient beyond one hour after ingestion. Beware of worsening cardiovascular status during lavage where hypoxia and pushing the contents beyond the pylorus, which increase absorption and cause sudden deterioration, particularly with trycyclic antidepressants.[1,6,7] Gastric lavage without consent constitutes assault.[1]

Contraindications

- **Corrosive poisons** such as concentrated inorganic acids, e.g. sulfuric acid, nitric acid, hydrochloric acid, etc. which corrode and soften the stomach wall, making it prone for perforation (exception carbolic acid which hardens the stomach wall and gastric lavage is done safely).
- Convulsive poisons It may be allowed only after the convulsions are controlled.
- Comatose patients It may be done only if the intubation is made visible radiographically.
- Other conditions — Volatile poisons, upper gastrointestinal tract diseases like oesophageal varices, patient in hypothermia, etc.

Note: The first three in the list are considered as absolute contraindications for gastric lavage.

EMESIS (VOMITING)

Emesis or vomiting may be induced by several methods, which are tickling the throat, administering the oral emetics and parenteral emetics.

Table 31.22: Oral emetics

Emetics	*Doses*
Common salt	1 tablespoon
Mustard	2 tea spoon
Tincture iodine	20 drops
Zinc sulphate	250 mg
Ammonium carbonate	1 gm
Tartar emetics	100 mg
Powder ipecac	1 gm
Syrup ipecac *	30 ml

* Ipecacuanha (Ipecac) induced emesis is no longer recommended as it cannot evacuate the poisons in the stomach significantly and the vomiting induced by it can mask the clinical features and also limit the use of activated charcoal [8]

Tickling of the Throat

Tickling of throat is the simplest method. But it is not possible when the patient is unconscious.

Oral Emetics

There are certain drugs (Table 31.22), which are dissolved in half a glass of lukewarm water and administered orally, to induce vomiting.

Contraindications

Oral emesis is contraindicated in following circumstances:

- Child less than 6 month of age
- Nontoxic ingestion
- Comatose patient
- Patient having seizures
- Ingestion of alkali or acid
- Compromised gag reflex
- Haemorrhagic diathesis such as patient with cirrhosis, varices and thrombocytopenia
- Ingestion of sharp and solid material such as glass, nail, razor, etc.

Precautions

If vomiting is not produced, these drugs may get absorbed and produce their own side effects and complications, e.g. hypernatraemia with common salt, cardiac depression with ipecac, nephrotoxicity and haemolysis with copper sulphate, etc.

Parenteral Emetics

These are drugs, which induce vomiting when given parenterally, e.g. apomorphine hydrochloride — 6 mg subcutaneous injection. However these drugs are contraindicated in cases with respiratory distress.

Activated Charcoal

Activated charcoal is black slurry material, which owing to its large surface area is highly effective at adsorbing many toxins. It is highly effective at adsorbing most poisons with a few exceptions owing to its large surface area and porous structure. It should be given to all patients who present within 1 hour of ingestion of a potentially toxic amount of poison, which would binds to charcoal. In certain circumstances it may be warranted to give activated charcoal more than 1 hour after ingestion, e.g. following an exceptionally large overdose of a substance that slows gastric emptying (e.g. tricyclic antidepressant, opioids) provided that the airway is protected. Activated charcoal can be given via a nasogastric tube if the patient is not able to swallow activated charcoal or has a reduced level of consciousness. The airway must be adequately protected before activated charcoal is given. Repeated doses of activated charcoal are also effective in increasing the elimination of some poisons from the body.

However, certain agents cannot be adsorbed by activated charcoal and they are:

- Metal salts, e.g. iron, lithium, potassium.
- Alcohols, e.g. ethanol, ethylene glycol, methanol.
- Other agents, e.g. cyanide, hydrocarbons, solvents, acids, alkalies, fluoride.

Contraindications

Contraindications to administration of activated charcoal are:

- More than 1 hour since ingestion
- Substance not bound to charcoal (see above)
- Airway cannot be protected
- Oral antidote is given.

Brands Available

Available formulations of activated charcoal vary from one country to another.

- Medicoal® is an effervescent preparation containing sodium citrate and povidone and must be mixed with water before use.
- Carbomix® comes in ready to use containers, to which water must be added.
- Liquichar® and Actidose aqua® come as ready to use prepared mixtures.

Dose

Activated charcoal is given in 50 gm doses for adults and in the dose of 1 gm/kg for children. It commonly causes vomiting and so if a patient is vomiting or nauseated prior to its administration, an antiemetic should be given. The dose for multiple-dose charcoal is 50 gm 4-hourly for an adult. Sometimes, for example in salicylate poisoning, charcoal may be given 2-hourly to limit absorption of the drug.

Precautions

In order to maintain the adsorptive power of the charcoal, never encourage the addition of any flavouring or coloring agents. For the same reason ice cream should not be given with charcoal.[1]

Complications

Some of the known complications of activated charcoal and the way how it can be prevented are presented in Table 31.23.

Table 31.23: Activated charcoal complications and how to avoid them

Complications	*Solutions*
Medicoal can cause diarrhoea	Could be regarded as a therapeutic advantage in some cases
Other charcoals cause constipation and rarely bezoars and even intestinal obstruction	Give a purgative when more than one dose of charcoal is given
Adsorption of oral antidotes	Do not give charcoal and oral antidote
Aspiration pneumonitis	Protect the airway, care in administration to unconscious patients

Catharsis

Catharsis is known to reduce the transit time of drugs in gastrointestinal tract.[9] However, there is no evidence to prove the fact that catharsis has reduced the mortality or morbidity in a poisoning case till now. Routinely two types of cathartics are tried in poisoning victims to induce catharsis and they are: Ionic / saline cathartics and Saccharide cathartics.

Ionic/ Saline Cathartics: These cathartics alter the physico-chemical forces within the intestinal lumen leading to osmotic retention of fluid which activates motility reflexes and enhances expulsion. However, excessive doses of magnesium based catharsis can result in hypermagnesemia which is a serious complication. The doses of recommended ionic/saline cathartics are as follows:

- Magnesium citrate—4 ml/kg
- Magnesium sulphate—30 gm (250 mg/kg in children)
- Sodium sulphate—30 gm (250 mg/kg in children)

Saccharides cathartics: This comprise of giving sorbitol (D-glucitol), which is the cathartic of choice in an adult because of its better efficacy than saline cathartics. However this is not indicated in small children as it can result in fluid and electrolyte imbalance (hypernatremia). The recommended dose for catharsis by sorbitol for an adult is 50 ml of 70 per cent solution.

Whole Bowel Irrigation

This means washing the gut rapidly. Whole bowel irrigation involves administration of non-absorbable polyethylene glycol solution to cause a liquid stool and reduce drug absorption by physically forcing gastrointestinal contents.[1, 10,11]

Indications

Whole bowel irrigation is a newer method of gut decontamination that is indicated for a limited number of poisons such as large ingestions of iron, lithium, sustained release or enteric coated drugs, e.g. theophylline, etc, which are not adsorbed by activated charcoal. This is also indicated in cases of body packers, i.e. ingestion of drug-filled packets/condoms.

Precautions

Prior to the procedure check whether the patient has paralytic ileus or not. The procedure usually involves passing a nasogastric tube or persuading the patient to drink the polyethylene glycol.

Commercially available polyethylene glycol preparations, such as Klean-Praeg (60 gm/L polyethylene glycol, with electrolytes often used in surgical units, are recommended.

They are instilled at a rate of 2 L/hour in adults and 0.5 L/hour in pre-school children. Watery diarrhea soon develops and the patient is then asked to sit on a commode. The polyethylene glycol is given until the rectal effluent becomes clear. Patients tolerate the procedure remarkably well. It even appears to be a safe procedure during pregnancy.[12] Polyethylene glycol is not absorbed and does not result in changes in water or electrolyte balance. Never confuse polyethylene glycol for ethylene glycol, which is a highly toxic antifreeze.

Complication

Table 31.24 provides the complications of whole bowel irrigation and the methods of preventing the same.

Table 31.24: Whole bowel irrigation complications and how to avoid them

Complications	*Solutions*
Haemodynamic compromise	Do not use in patient with hemodynamic compromise from bleeding e.g. severe iron poisoning
Obstruction	Do not use in patients with ileus

METHODS FOR ENHANCING ELIMINATION OF TOXINS

In the vast majority of patients who present after an overdose, gut decontamination methods and supportive care are all that is necessary. In a limited number of poisonings it may be necessary to consider the use of one of the methods that are available to increase elimination of poisons these include: Urinary alkalinization, multiple-dose activated charcoal, extracorporeal techniques and diaphoresis.[1,13]

Urinary Alkalization

This is also known as alkaline diuresis and is indicated for serious poisoning with:

- Chlorpropamide
- Mecoprop
- Phenobarbitone
- Phenoxyacetate herbicides
- Salicylates including aspirin.

Prior to the procedure check the patient's plasma potassium concentration and renal function tests. Give intravenous bicarbonate 1 litre of 1.26 per cent (for an adult) over 3 hours. Check plasma potassium, as it is very difficult to produce alkaline urine if the patient is hypokalemic. Also potassium can fall precipitously once adequate urinary alkalinization commences and so it is wise to add 20-40 mmol potassium to each litre of I.V fluids given. Check that adequate urinary alkalization is achieved (aiming for a urinary pH of 7.5-8.5) by checking the pH of the urine with indicator paper.

However, never force a diuresis, as pulmonary edema will ensue. So also never give acetazolamide diuretic to induce alkaline urine as it produces a systemic acidosis which enhances the toxicity of certain drugs such as salicylates.[13]

Multiple-dose Activated Charcoal

Repeated doses of activated charcoal can increase the elimination of some drugs by interrupting their enteroenteric and entero-hepatic circulation.[1, 14] Clinical studies have shown that multiple dose activated charcoal increases the elimination of certain drugs, but no controlled studies have established beyond doubt a clinical benefit on patient outcome, though this would seem logical.

The dose given is 50 gm (1 gm/kg in children) of activated charcoal every 4 hours.

Indications for multiple-dose activated charcoal include life-threatening overdose with:

- Carbamazepine
- Dapsone
- Phenobarbitone
- Quinine
- Theophylline.

Extracorporeal Techniques

Hemodialysis, charcoal hemoperfusion, hemofiltration are some of the extracorporeal techniques. There are a limited number of poisonings in which one of these procedures may be indicated. Before deciding whether or not to undertake one of these procedures in a poisoned patient, the case should be discussed with a clinical toxicologist. However, charcoal hemoperfusion columns being not widely available, there is only a limited data on the use of haemofiltration in poisoned patients and hence at present the method is not recommended. Tables 31.25 and 31.26 present indications and complications of extracorporeal techniques respectively.[1]

Table 31.25: Indications for haemoperfusion and haemodialysis in poisoned patients

Drugs for which haemodialysis may be considered	*Drugs for which charcoal haemoperfusion may be considered*
Salicylates	Theophylline
Ethylene glycol, methanol, ethanol	Phenobarbitone
Theophylline	Carbamazepine
Lithium	

Table 31.26: Extracorporeal technique complications and how to avoid them

Complications	*Solutions*
Hypotension	Gradually increase flow rates in the extracorporeal circuit. Remember that hypotension may be due to the poisoning!
Air embolism	Carefully prime the extracorporeal circuit
Sepsis	Strict aseptic technique during central line insertion
Thrombocytopenia	Anticoagulation with prostacyclin rather than heparin
Bleeding	Careful monitoring of anticoagulation

Diaphoresis

Diaphoresis means inducing excessive perspiration, and poison is excreted through sweat. In most of the cases it is doubtful how far this can speed up the excretion of toxic agents consumed or absorbed. Methods adopted include application of heat by fomenting with hot water bottles or covering the body with thick blankets, administering hot beverages like hot tea, coffee or milk, hot lemonade, etc can induce increased perspiration. Profuse perspiration can be induced by giving subcutaneous injection of 5 mg of pilocarpine nitrate. Giving alcohol, salicylates or antipyretics are also used to produce less marked perspiration.

ADMINISTRATION OF ANTIDOTES

Despite popular misconceptions, antidotes are available for only a small number of poisons.[1] Table 31.27 enumerates the various antidotes available marking their indications.[2,4]

Action of antidotes—antidotes are substances, which, on administration counteract or neutralize the effect of a poison.[2]

Classification According to Action

Antidotes can be classified in three types according to the mode of action.[2,15,16]

- Physical/mechanical antidotes
- Chemical antidotes
- Physical and chemical antidotes
- Physiological antidotes.

Table 31.27: Antidotes and their indications

Antidotes	*Indications*
4-methylpyrazole	Methanol and ethylene glycol
100% oxygen	Carbon monoxide, cyanide, methemoglobinemia
Ammonium chloride	Phencyclidine, amphetamine and strychnine
Amyl nitrate	Cyanide, hydrogen sulphide
Atropine	Carbamate and organophosphorus poisoning
Calcium disodium edetate (EDTA)	Cadmium, chromium, cobalt, copper, lead, magnesium, nickel, uranium, zinc
Calcium gluconate	Precipitates fluorides, magnesium and oxalates
Chlorpromazine	Amphetamine
Deferoxamine	Iron, aluminium
Diazepam	Amphetamine, barbituarate, chloroquine, alcohol withdrawal
Dicobalt edetate[1]	Cyanide
Dimercaprol (BAL)	Antimony, arsenic, copper, lead, mercury, nickel and gold.
Diphenhydramine	Phenothiazines and related drugs
Disodium calcium edetate	Lead
DMPS *, (Unithiol)	Mercury
DMSA **	Lead
D-Penicillamine (Cuprimine)	Arsenic, copper, lead, mercury, chromate, nickel, zinc
Ethanol	Methanol and ethylene glycol
Fab fragment	Digoxin, digitoxin, oleander tea
Glucagon	Propranolol and other beta-blockers toxicity
Hydroxocobalamin[1]	Cyanide
Labetalol hydrochloride	Hypertensive crisis due to cocaine
Methionine	Paracetamol
Methylene blue	Methemoglobinaemia
N-acetylcysteine	Paracetamol, acetaminophen toxicity
Naloxone (Naltrexone)	Narcotic, opiates, CNS depressants
Nicotinamide	Vacor poisoning, phenylurea pesticide toxicity
Pancuronium bromide	Neuromuscular blocking agents
Penicillamine	Copper
Physostigmine salicylate	Coma, convulsions from anticholinergics
Pralidoxime (2-PAM) [1]	Organophosphorus insecticides, nerve agents
Propranolol	Cocaine intoxication, beta adrenergics
Protamine sulphate	Heparin overdose
Prussian blue[1]	Thallium
Pyridoxine (Vit. B_6)	Isoniazid, hydrazine toxicity
Sodium bicarbonate	Urinary alkalinisation for salicylates, phenobarbital
Sodium nitrite[1]	Cyanide
Sodium thiosulphate[1]	Cyanide
Vitamin K	Warfarin

* DMPS (2,3-dimercapto-l-propane sulphonate)
** DMSA (2,3-dimercapto-succinic acid)

Physical/ Mechanical Antidotes

These neutralize the poison by their mechanical action, e.g. egg albumin (act as demulcents) in corrosive and irritant poisons, and animal charcoal (act as an adsorbant) in alkaloid poisons.

Chemical Antidotes

These acts by forming new compounds with the poison, which will be either nontoxic or less active or insoluble, e.g. dilute alkalies (magnesium oxide) in acid poisoning, acetic acid or vinegar for alkalies, etc.

Universal Antidote

It is an antidote, which is a combination of physical and chemical antidote. It can be administered, when the exact nature of poison taken is not known or when one or more poison is/are taken. However, recently use of universal antidote has been criticized and condemned.[17,18]

Composition — It is made up of 2 parts (50%) of powdered animal charcoal or burned toast, 1 part (25%) of magnesium oxide or milk of magnesia and 1 part (25%) of tannic acid or tea.

Dose — 1 tablespoon mixed with one glass of water.

Route — Given orally.

Physiological/Pharmacological (Antagonists) Antidotes

These act by producing exactly the opposite action to those produced by the poison, e.g. physostigmine in pilocarpine poisoning, and atropine in organophosphorus poisoning, naloxone in morphine poisoning, etc.

However, in practice it has been concluded that none of these presumed beneficial effects of antidotes is ever accomplished, other than only a false sense of satisfaction of having given an antidote is generated, which can be disastrous leading to dangerous complacency.[17]

Chelating Agents

These are true physiological agents which act by forming stable and soluble complexes by the inner ring structure which can combine with the poison molecules easily, e.g. British antilewisite (BAL), ethylene diamine tetra-acetic acid (EDTA), pencillamine, etc in metallic poisons like lead, mercury, arsenic, copper, etc. desferrioxamine for acute iron poisoning.

- British Anti-Lewisite (BAL) — BAL is a colorless liquid (100 mg/ml) in peanut oil, given deep intramuscularly. It is administered in a dose of 2.5 mg/kg body weight, four hourly for 2 days followed by six hourly for 2 days and 12 hourly for next subsequent 10 days or till improvement.
- Ethylene diamine tetra-acetic acid (EDTA) — EDTA is rarely used in heavy metallic poisoning, as it is prone to remove blood and bone calcium also producing fatal hypocalcaemia. (However calcium complex compounds of EDTA are now available). It is administered in a dose of 50-70 mg/kg body weight (1 gm in 500 ml. of 5 per cent glucose or normal saline, by slow intravenous drip, twice a day for 3 to 5 days).
- Penicillamine — Penicillamine is obtained by hydrolytic dehydration of penicillin. It is well absorbed from gastrointestinal tract. It is administered in a dose of 30 mg/kg body weight per day orally.
- Deferoxamine—Deferoxamine is a watersoluble compound with great affinity for ferric iron. It is administered in a dose of 8 gm in 50 to 200 ml of water given orally or by nasogastric tube, 1-2 gm intravenously or intramuscularly 4 hourly for 2 days.

SYMPTOMATIC LINE OF TREATMENT

It constitutes certain measures of treatment provided to give relief from poisoning symptoms,[2] e.g.: give atropine sulphate for colicky pain, morphine or pethidine for severe pain, oral or IV fluids for dehydration, etc.

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DIAGNOSIS OF POISONING IN THE DEAD

Diagnosis of poisoning in the dead is usually done by meticulous postmortem examination of the dead body, looking for specific changes in the cadaver, analytical toxicology (of biological fluids), and histopathological examination. Some of the circumstantial evidence also helps in the poison detection process.

1. Certain Specific Changes in the Cadaver—If the dead body is closely observed there will be several changes noticed which help in detection of poison causing death.[1-3] These changes comprise of external as well as internal autopsy observations, and constitute of colour changes imparted to the postmortem lividity (Table 31.28), characteristic odours emitted from the cadaver externally or on opening the body cavities (Table 31.29), and other internal changes (Table 31.30) noticed on autopsy.
2. Analytical toxicology (of biological fluids)—Analytical toxicology is to help not only in diagnosis of the poisoning in a living case in a hospital, but also facilitate poison

Table 31.28: Poison identity by colour changes imparted to postmortem lividity

PM lividity colour changes	*Poisons identified*
Blue	Copper sulphate
Brick red	Cyanide
Cherry red	Carbon monoxide
Yellow/ brownish	Phosphorous

Table 31.29: Poison identity by characteristic odour emitted from the cadaver

Poisons	*Odour emitted*
Arsenic, phosphorus, malathion, thallium	Garlic odour
Cannabis (marijuana)	Burnt rope
Carbolic acid, lysol, etc.	Phenolic odour
Cyanide	Bitter almonds odour
Ethyl alcohol	Fruity odour
Hydrogen sulfide	Rotten eggs
Kerosene, organo-phosphorus insecticides	Kerosene like odour
Zinc phosphide	Fishy (mushy)

Table 31.30: Internal changes giving clue to the poisons

Other changes	*Poisons*
Corrosion of lips, mouth, chin, hands, corrosion, softening or perforation of gastrointestinal mucosa, etc.	Corrosive poisons such as concentrated sulphuric acid, nitric acid, oxalic acid, etc
Xanthoproteic reaction (yellow discoloration of skin and tissues)	Concentrated nitric acid
Injection marks, sting marks, bite marks	Injectable poisons, insects stings and snake bite
Delay/retarding of putrefaction	Arsenic, organophosphates
GI Contents: Tablets,powders fluid, and vegetable remain especially seeds in the stomach or intestines, emitting specific odour.	Specific poisons identified
Subendocardial hemorrhage in the heart	Arsenic, barbiturate, etc.
Necrosis/degeneration of liver, kidneys, etc	Hepatotoxics and nephrotoxics

identification in a dead body in the mortuaries. However, this is accomplished by chemical analysis of the biological fluids (blood, urine, semen, sweat) and viscera/tissues collected from the dead body at Forensic Science Laboratory. (Refer wide infra for Analytical Toxicology).

3. Histopathological examination (HPE)—Histopathological examination of tissues could give clues regarding degenerative change due to certain poisons. Histopathological examination of tissues is usually valuable, as it could furnish corroborative evidence in poisoning death. Viscera have to be thus selected and preserved considering the target organs on which suspected poison act; e.g. liver is the target organ in alcohol abuse, which can present with cirrhotic changes, could certainly substantiate alcohol poisoning as cause of death.[1,4] All viscera to be subjected for histopathology should be always preserved in 10 per cent buffered formalin. Formalin not only preserves the architecture of the viscera but also the disease process/ the changes it has undergone due to the poison.
4. Circumstantial evidence—Circumstantial evidences obtained from the belongings of the deceased such as a suicide note in the pocket of the garments worn by the deceased, an empty container (bottle) in the hand of the dead with a label of poisonous material consumed, e.g. Follidol, Endrin, etc, can certainly give clues regarding the poisoning and its motive.[4,5]

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VISCERA AND CHEMICAL EXAMINATION

In every case of accused poisoning efforts are made to identify the poison by chemical analysis at forensic science laboratory (FSL). Poison identification may also be made in clinical laboratory in a hospital if ideal facilities exist. However, as the FSL reports are accepted in the courts of law, they are often preferred.

Usually analytical methods (refer Analytical Toxicology) detects the poison from the material sent from the victim. When the victim is alive, biological fluid constitutes ideal material for analysis, while the viscera are preferred from a dead body.[1,2]

For chemical analysis at FSL, it is essential that all the materials must be collected and preserved properly with appropriate preservative and dispatched through police. The section here deals with selection, collection and preservation of the viscera for chemical examination.

SELECTION OF VISCERA

Viscera for chemical examination depend on the poison taken. Table 31.31, presents the list of materials (viscera and biological fluids) that has to be collected in an adult for chemical analysis in all case of poisoning (Fig. 31.3) and special viscera in certain special cases of poisoning.[1-3]

COLLECTION OF VISCERA

Collect in clean, sterile and separate containers, close, seal and label (Fig. 31.3) them, properly giving details of name of the deceased, date and place of autopsy, name and signature of the doctor performing it.

PRESERVATION OF VISCERA

Anyone of the following two preservatives may be used for the purpose.[1-2]

- Saturated solution of common salt (NaCl, i.e. sodium chloride) is prepared by adding salt to water till it remains undissolved on vigorous stirring. It is indicated in all poisoning cases
- Rectified spirit is indicated for most of the poisons, exceptions being alcohol, kerosene, phosphorus, paraldehyde, carbolic acid, acetic acid, etc.

PRESERVATION OF BLOOD AND URINE

- For 25 ml blood, add 125 mg sodium fluoride as preservative and 75 mg potassium oxalate as anticoagulant. Refrigeration of the sample may be helpful in alcohol poisoning cases. In case of carbon monoxide poisoning, pour 2-3 cm thick layer of liquid paraffin over blood sample.

Table 31.31: Material to be preserved for chemical examination in adults in routine and special of poisoning with indications

Material		*Quantity*	*Indications*
Routine $^{+}$	Special $^{++}$		
Stomach	–	Entire	All poisons
Stomach contents	–	All contents/ 300 ml	All poisons
Small intestine	–	Proximal 30 cm	All poisons
Small intestinal contents	–	Up to 100 ml	All poisons
Liver	–	Whole or minimum 500 gm with entire gallbladder or all bile	All poisons
Kidney	–	Half of each or full one kidney (both Kidneys in infants)	All poisons
–	Brain	Half (one hemisphere) or a minimum of 500 gm	Volatile and other CNS poisons #
–	Spinal cord	Entire length	Strychnine, gelsemium
–	Lungs *	Half of each or one whole lung	Inhaled poisons
–	Heart	Entire	Cardiac poisons, arsenic, etc.
–	Muscles	Minimum 200 gm	When internal organs putrefied
–	Bones ***	1 long bone or 10 cm length or 10 gm of each long bone total minimum 200 gm	Heavy metals
–	Scalp hairs**	One tuft of minimum 10 gm	Heavy metals
–	Nails	All nails	Arsenic
–	CSF	As much as withdrawn	Alcohol
Blood	–	25 ml of venous blood	All poisons
Urine	–	All quality available	All poisons

* Send in air tight container

** Plucked out

*** Long bones preferably femur

# Barbiturates, CO, anaesthetics, cyanide, strychnine, opiates, alcohol

+ *Viscera to be preserved routinely*

Thus routinely — stomach and proximal 30 cm of small intestine with their contents; 500 gm liver with gallbladder, one whole kidney, blood (25 ml) and urine sample are sent for chemical examination in every case of poisoning (Fig. 31.4). This may be remembered as:

- Two hollow viscera, i.e. stomach and intestine,
- Two solid viscera, i.e. liver and kidneys and
- Two body fluids, i.e. blood and urine.

++ *Special viscera*

All or one of these to be preserved in addition to routine viscera as shown in table column indication.

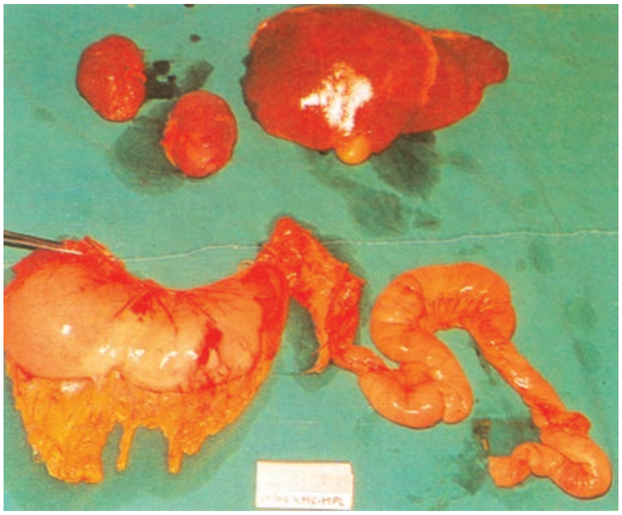

Fig. 31.3: Viscera to be preserved routinely in suspected case of poisoning

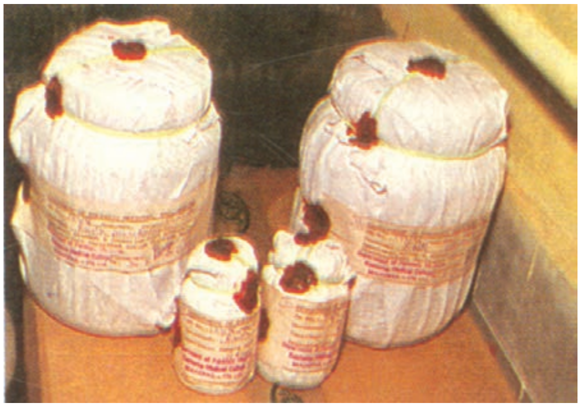

Fig. 31.4: Viscera bottles packed, sealed and labelled for dispatch to FSL

Bottle 1—Stomach with contents and proximal 30 cm of small intestine
Bottle 2—500 gm of liver and half of both kidneys
Bottle 3—Blood 25 ml in sodium fluoride preservative
Bottle 4—Sample preservative used in bottle 1 and 2

- For urine, add thymol as preservative. Other alternatives are saturated solution of sodium chloride or 5 ml of concentrated hydrochloric acid for every 250/500 ml.
- Specimen of bones does not require preservative.

Note

- Always send a sample of the preservative used to chemical examiner in separate bottle.
- Never use formalin as a preservative for sending the viscera to chemical examiner as it hardens (fixes) the tissue, renders difficulty to extract poison (formalin is used as a fixative for histopathological examination of tissues).

Sending Viscera to Chemical Examiner [2]

- Write a requisition
- Send the specimen packed ideally in separate containers, which are closed and sealed from outside, through a police constable (note his PC No)
- Always demand and collect a receipt for delivering the specimen to the police constable.

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MEDICOLEGAL ASPECTS OF POISONING

This depends on whether physician (clinician) who has treated a victim of poisoning when alive or performed medicolegal autopsy on victim of poisoning after death. Accordingly there are two aspects medicolegally, which a doctor has to be aware of:

I. Duties of a doctor towards suspected/actual case of poisoning.
II. Preparation of document from medical witness for an autopsy surgeon.

DUTIES OF A DOCTOR TOWARDS SUSPECTED/ ACTUAL CASE OF POISONING[1-7]

This constitutes medical duties and medicolegal duties:

Medical Duties

- Save life (undertake ABCD of assessing the patient and treat accordingly).
- Remove from the source of poisoning.
- Advice immediate hospitalization of the victim.
- Identify the poison consumed.
- Assess the approximate time of poisoning.
- Assess the approximate dose taken
- Ascertain the route of poisoning.
- Rule out any head injury.
- Appraise the psychiatric status.
- Elicit any history of suicide attempt earlier
- Confirm history of drug abuse.
- Exclude allergic/hypersensitivity reactions to drugs in the past.
- Verify for any other coexisting diseases.
- Take second opinion from a competent professional colleague if necessary.

Medicolegal/Forensic Duties

- Doctor must notify the health authority in case he comes across cases of food poisoning involving several people at same time from same source of a public eating places like hotel, cafeteria, hostel canteen, restaurant, etc. or at a mass eating place such as at a wedding party dinner or any such other group/festival occasions eating in common place.
- Doctor must make every attempt to collect, preserve and dispatch all the suspected materials to the chemical examiner. In the event of non compliance to this, the doctor is held responsible for the loss or disappearance of evidence and punishable (Section 201 IPC).
- Doctors must inform police or magistrate adopting following guidelines:
  – All private medical practitioners must inform every suspected or alleged homicidal poisoning case to the police (Section 39 Cr PC). Failing to do so render the doctor guilty and punishable (Section 176 IPC).
  – Cases of suicidal poisoning or attempted suicidal poisoning, a doctor need not inform about it to the police (Section 309 IPC), however, if an investigating officer (IO) inquires about it, he must divulge in detail (Section 175 Cr PC) or else the doctor is liable to be prosecuted and punished for not giving information (Section 202 IPC). So also, the doctor is punishable if he gives any false information to police (Section 193 IPC).
  – A government medical officer must inform all cases of poisoning to the police at the earliest.
- To arrange for recording dying declaration if the patient is about to die. However if there is any delay in arrival of magistrate, and death is imminent, doctor himself can record the dying declaration (Section 32, Clause 1 of Indian Evidence Act [IEA]).
- If the victim dies withhold issuing of the death certificate and arrange for the medicolegal autopsy examination at the earliest.
- Doctor must always maintain a detailed written record on every case of poisoning treated by him and kept under safe custody labeling medicolegal case (MLC)

PREPARATION OF DOCUMENT FROM MEDICAL WITNESS FOR AN AUTOPSY SURGEON

This document should include three aspects—details on symptoms suggestive/suspicious of poisoning, autopsy findings/ details and analytical details.[1,2]

Details on Symptoms Suggestive/Suspicious of Poisoning. Autopsy surgeon must take care to record details on certain points eliciting proper history/circumstances that demand special attention on:

- Nature of symptoms (history from relatives/friends/hospital records, if any)
- Date of onset
- Order of occurrence: sudden/slow onset

- Whether intermittent/continuous/severe poisoning led to death
- Past history of any diseases
- History of head Injury
- Whether family physician was called, or not, or called on delay (given reasons for any such delay)
- Statements about symptoms by victim (if alive) or by any other with the victim
- Any other person suffering with similar symptoms
- Past history of consuming the alleged substance without any ill effects
- Possibilities of suicide, homicide or accident
- Interval between taking the poison and death
- Probable source of poison
- Probable time since death
- Preserve all suspected articles.

Autopsy Findings/Details

Record must be made on all the details on observations regarding the following.

External Findings

- Position or attitude of the body
- Findings on the clothing
- Colour of postmortem lividity (hypostasis)
- Corrosion of lips, mouth, etc.
- Any distinct odour
- Evidence of violence
- Evidence of injection marks
- Rectal temperature
- State of rigor mortis
- Things around the victim.

Internal Findings

- All organs must be examined and contents must be preserved for chemical examination to identify the poison.
- Detailed information as observed must be entered in the report, giving importance to all the organs examined thoroughly.

Analytical Details

In every case of poisoning deaths, this is a must to confirm the diagnosis of poisoning proposed on autopsy examination. To have this in the record, doctor should take proper measures at the level of conducting the postmortem examination himself, by taking proper steps to arrange for dispatching the viscera collected from the deceased for chemical examination. Chemical examination of viscera is usually done at Forensic Science Laboratory (FSL) or Regional Forensic Science Laboratory (RFSL) situated in the capital of each State or district head quarters, respectively.

Cause of Death

Cause of death can be usually deduced on FSL Report. At times even if a FSL report is negative stating 'no poison is detected in the materials sent',one must not be uncertain to write cause of death based on the autopsy examination findings, history provided and findings in the clinical case sheet whenever possible. Conversely, it is also relevant to mention here that in every case it may not be possible to conclude on cause of death and under such circumstances doctor must not to hesitate to write the fact.

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LAWS OF POISONS IN INDIA

In the court of law, in a case of poisoning, intention with which the substance was administered is more important. Any substance administered with intent to injure or produce ill health or death, constitutes an offense by the court of law and is punishable. Poisons, which can kill human beings, are often useful in medical practice, given in small doses and are then considered as medicine. Thus a medicine in toxic dose is a poison and a poison in small dose may be a medicine. This rendered, it is essential to establish stringent laws on drugs and poisons. Some of the Indian statutes of importance in relation to drugs and poison[1-5] in this country are:

- The Indian Penal Code (IPC)
- The Criminal Procedural Code (CrPC)
- The Indian Evidence Act (IEA)
- The Narcotic Drugs and Psychotropic Substance Act, 1985
- The Drug and Magic Remedies Act, 1954
- The Drug Control Act, 1950
- The Pharmacy Act, 1948
- The Drugs and Cosmetics Rules, 1945
- The Drugs and Cosmetics Act, 1940
- The Drugs Act, 1940
- The Poison Act, 1919

THE INDIAN PENAL CODE (IPC), CRIMINAL PROCEDURAL CODE (CRPC) AND INDIAN EVIDENCE ACT (IEA)

Under the Indian Penal Code (IPC), sections 284, 299, 300, 304A, 306, 307, 309, 324, 326, and 328 and under the Criminal Procedural Code (CrPC) sections 39, 40, and 175 deals with poisons in India and Table 31.32 highlights facts regarding some of these sections briefly. Section 32, Clause 1, under the Indian Evidence Act (IEA) allows a doctor to record dying declaration

Table 31.32: Facts regarding laws (Sections under Cr PC/IPC) relevant to poisoning

Sections under Cr PC	*Sections under IPC*	*Details/Relevances*
Section 39, 40	Section 176	Doctors must report all cases of homicidal poisoning to police, if not they are punishable
	Section 193	Doctor is punishable for giving false information about poisoning case
Section 175	Section 201	Doctor must divulge about a poisoning case to IO
	Section 202	Doctor is punishable for concealing facts about a case of poisoning treated to IO
	Section 284	Lays down penalty for any person causing harm by rash and negligent handling of a poison
	Section 299	Culpable homicide (by any method including poisoning)
	Section 300	Murder (by any method including poisoning)
	Section 304A	Rash and negligent—homicide by method including poisoning
	Section 324	Causing hurt by dangerous weapons or means (including poisoning)
	Section 326	Causing grievous hurt by dangerous weapons or means (including poisoning)
	Section 328	Causing hurt by means of poison with intention is punished by imprisonment (10 yr) and fine.

when the death of the patient is imminent and arrival of magistrate is delayed.

NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCE ACT, 1985 (NDPS ACT)

This Act consolidates and amends the law relating to narcotic drugs (The Opium Act, 1878, 1957; and The Dangerous Drug Act, 1930 — both are repealed by this Act), drugs of abuse, penalties for the drug trafficking offenses and control over psychotropic substances. Narcotic dugs under this act include opiates, cannabis, and cocaine. The psychotropic drugs under this Act refers to mind alerting drugs such as LSD, phencyclidine, amphetamines, barbiturates, methaqualone, benzodiazepines, mescaline, psilocybin, and designer drugs (MDMA, DMT, etc)

THE DRUG AND MAGIC REMEDIES ACT, 1954

This Act bans the advertisements of magical remedial drugs ('manthras', 'kavachas', etc) for curing conditions such as venereal diseases, impotency, menstrual disorders, infertility and abortion, misconception and insanity.

THE DRUGS CONTROL ACT, 1950

This Act controls the fixing of prices of drugs for the manufacturer or dealer, sale, supply and distribution of drugs.

THE PHARMACY ACT, 1948

This Act makes provision for regulation of the profession of pharmacy and for the purpose of constitution of Pharmacy council of India, which regulates study of pharmacy throughout the country. Individual states have State Pharmacy Councils for registration of pharmacist.

THE DRUGS AND COSMETICS RULES, 1945

This is a derivative of Drugs and Cosmetics Act 1940; and it covers all kinds of drugs used in therapeutics under allopathic, ayurvedic, unani, and siddha preparations. The rule deals mainly with the standard and quality of drugs. It also controls the drugs by specific regulation laid down for their storage, display, sale, dispensing, labelling, prescribing, etc. by classifying them into several schedules, such as:

Schedule C — Biological and special products such as sera, vaccines, etc

Schedule E — This schedule lists poisonous substances under ayurvedic, siddha and unani systems.

Schedule G — List includes hormone preparations, antihistamines, and anticancer drugs.

Schedule H — These are drugs or poisons which need to be labeled as 'Schedules H Drug Warning — to be sold by retail on the prescription of Registered Medical Practitioner only.' Barbiturates, amphetamines, reserpine, ergot, and some of the sulphonamides are listed under this schedule.

Schedule J — Drugs, which should not be advertised for certain diseases which cannot be announced. This covers list of drugs which are claimed to be cure of conditions such as appendicitis, blindness, cancer, cataract, epilepsy, hydrocoele, etc.

Schedule L — Antibiotics, antihistaminic and other chemotherapeutic agent of recent origin subjected to same restrictions as Schedule H drugs.

THE DRUGS AND COSMETICS ACT, 1940

This Act was amended in 1964 and it regulates the drugs of articles of cleansing (except soap), beautifying and promoting attractiveness or altering appearances. This act also demands the fact that every patented or proprietary medicinal preparation under this act must display a label on the container mentioning the exact formula or list of ingredients in it.

THE POISON ACT, 1919

This was amended in the year 1958 and repealed in 1960. It regulates launching, licensing, importation and sale of poisons in India.

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ANALYTICAL TOXICOLOGY

Analytical toxicology services, has objectives of providing support for diagnosis, identification of poison, management/treatment and prognosis of poisoning.[1-4] These are variable and depend on local arrangements. Two more parameters that can be added to this are law enforcement and education/ research (Box 31.1).

In developed countries, they may be provided by specialized laboratory attached to a clinical toxicology unit, by a hospital biochemistry laboratory, an analytical pharmacy unit, and a university department of forensic medicine. In many of the developing countries, such services are not available on a regular basis, and where they are available, the physician is generally dependent on a national or regional health laboratory established for other purposes and operating only part of the time. In India mostly the government forensic science laboratory (FSL) caters the required services, while a very few laboratories in private may also be doing the services in a few places. There are however, many simple analytical techniques that do not need sophisticated equipment or expensive reagents, or even a continuous supply of electricity. Such tests could be carried out in the basic laboratories with basic equipments (Table 31.33) that are available to most hospitals and health facilities, even in developing countries.[5-7] With training, hospital laboratory staff could use these techniques to provide an analytical toxicology service to the physicians treating poisoned patients.

APPARATUS AND REAGENTS

Analytical toxicology services can be provided in clinical biochemistry laboratories that serve a local hospital or accident and emergency unit. Certain basic laboratory equipment, are needed (Table 31.33) are usually available in all biochemistry laboratory. A continuous electricity supply is not essential for operating these equipments. No reference has been made to the use of more complex techniques that may be made available along with. However, certain specialized expensive equipments,[3,5] which are also considered essential, at the analytical toxicology centre, are mentioned in Table 31.34. Table 31.35 enumerates the steps of undertaking an analytical toxicological investigation.

REFERENCE COMPOUNDS AND REAGENTS

- Relatively pure compounds for use as reference standards are essential if reliable results are to be obtained. However, expensive reference compounds of a very high degree of purity are not needed.[5] Some drugs such as barbital, caffeine and salicylic acid and many inorganic and organic chemicals and solvents are available as laboratory reagents with adequate degree of purity through normal laboratory chemical suppliers.
- Although the apparatus required for performing the tests described here are relatively simple, several unusual laboratory reagents are needed in order to perform all the tests.

Box 31.1: Objectives of analytical toxicology

- Diagnosis of poisoning
- Identification of poison
- Management/treatment
- Prognosis
- Law enforcement
- Education/research

Remembered by a mnemonic – DIMPLE

PRACTICAL ASPECTS OF ANALYTICAL TOXICOLOGY

It is assumed that some practical aspects of analytical toxicology are essentially considered at this juncture and these include:

- Clinical chemistry— comprises reagents and drug standardization, balances and pipettes, chemically pure water availability, etc.
- Basic laboratory operations— quality assurance, recording and reporting results
- Laboratory health and safety measures— usually all analytical toxicology centres handle highly toxic chemicals, needing careful preservation and handling of such items in the laboratory. This necessitates a chemical examiner in the laboratory to maintain certain guidelines and these include:
  - Strong acids and alkalies should never be preserved together

Table 31.33: Summary of basic equipment required for toxicological analyses *

- Calibrated laboratory balance (top-pan and analytical)
- Bench-top centrifuge (electrical/hand driven) for separating blood samples, solvent extracts
- Vortex—mixer or other forms of mechanical or hand driven shaker
- Water-bath and heating block
- Spirit lamp or butane gas burner
- Refrigerator (electrical or evaporative) for storing standards/samples
- P^H meter
- Range of automatic and semiautomatic pipettes
- Low power polarising microscope
- An adequate supply of laboratory glassware, including volumetric apparatus and adequate cleaning facilities
- A supply of chemically pure water
- A supply of compressed air or nitro
- A supply of thin layer chromatography plates or facilities for preparing such plates
- Facilities for developing and visualising thin-layer chromatograms, including an ultraviolet lamp (254 nm and 366 nm) and a fume cupboard
- Single beam or dual beam ultraviolet/visible spectrophotometer and associated cells
- Conway micro diffusion apparatus
- Porcelain spotting tile
- Modified Gutzeit apparatus

* Basic analytical toxicology—International Programme on Chemical Safety, Manual, WHO, Geneva, 1995.

Table 31.34: Essential specialized equipments, for analytical toxicology

- High-performance liquid chromatography (HPLC)
- Gas chromatography (GC)
- Atomic absorption spectro-photometry
- Immunoassay facility — radio immunoassay (RIA) and enzyme-mediated immunoassay (EMIA)

Table 31.35: Steps to be undertaken in an analytical toxicological investigation

Phases and Steps
Pre-analytical phase
Step 1 Obtain details of current admission, including any circumstantial evidence of poisoning and results of biochemical and blood investigations
Step 2 Obtain patient's medical history, if available, ensure access to the appropriate sample(s), and decide priorities for the analysis
Analytical phase
Step 3 Perform the agreed analysis
Post-analytical phase
Step 4 Interpret the results and discuss them with the clinician looking after the patient
Step 5 Perform additional analysis, if indicated, on the original samples/ on further samples from the patient

  - Strong acids or alkalies should always be added to water and not vice versa
  - Organic solvents should not be heated over a naked flame but in water bath
  - Use fume cup boards/hoods when organic solvents are heated.
- Laboratory staff should be aware of local policies regarding the health and safety and especially regulations regarding the processing of potentially infective biological specimen.
- Analysis actual of viscera preserved at an autopsy of suspected fatal poisoning case or any biological material sent from a living person suspected of poisoning in the casualty. This is done to detect the poisonous substance in it and usually dealt under wet chemistry.

WET CHEMISTRY

Traditional wet chemistry refers to the wet chemical methods of analysis. This comprises a painstaking testing and retesting of unknown material using different reagents, until the composite behavior patterns give a clue to the identity of the material/ poison. Wet chemistry also refers to the analytical techniques in which various chemical reagents, such as acids, bases and salts are applied to a sample, identified on the basis of its reactions with the reagents. These tests are further of two types.

- Qualitative analytical tests
- Quantitative analytical methods

Qualitative Analytical Tests

This includes certain biochemical tests where-in the nature of the poison is identified using the biological material samples from the victim of poisoning (such as blood, urine, faeces, saliva, CSF, gastric lavage material, vomitus, scene residue, etc.) by certain biochemical tests. Routinely certain color tests (bedside tests) are in common practice. Twelve qualitative colour tests are described in Table 31.36 and are based on simple colour reaction and cover number of important drugs and other poisons detection.

Colour Tests (Bedside Tests)

Principle

Many of the drugs and other poisons, if present in sufficient concentration and in the absence of interfering compounds, give characteristic colors with appropriate reagents.

Advantages

Many of these tests can be performed satisfactorily in clear glass test tubes. However, use of spotting tile (a white glazed porcelain tile with a number of shallow depression or wells in its surface) gives a uniform background against which to assess any colors produced, and also minimize the volumes of reagents and sample that need to be used.

Disadvantages

Some of these tests are, for practical purposes, specific but compound containing similar functional groups will also react, and thus interference from other poisons, metabolites or contaminants is to be expected. Further complications are that color description is very subjective, even in people with normal color vision, while the colors produced usually vary in intensity or hue with concentration, and may be unstable.

Precautions

When performing color tests it is always important to analyze concurrently with the test sample.

- A reagent blank (control sample), i.e. an appropriate sample known not to contain the compound(s) of interest, e.g. if the test is to be performed on urine, then a blank (analyte free) urine should be used, otherwise clean water is adequate.
- A known positive sample at an appropriate concentration: If the test is to be performed on urine, then ideally urine from a patient or volunteer known to have taken the compound in question should be used. However, this is not always practicable and then spiked urine (blank urine to which a known amount of the compound under analysis has been added) should be used.

Quantitative Analytical Methods

This comprises of methods where in the tests are done not only to exactly identify the poison but also to estimate the concentration of the poison in the body of the poisoned victim, e.g. a quantitative analysis carried out on whole blood or plasma to confirm the poisoning. However, these tests may not be possible in laboratories where facilities are limited. Enumerated below are quantitative analytical assays:[1,3,5,7]

- Chromatography
- Ultraviolet spectrophotometry
- Mass spectrometry.

Chromatography

Basic Principle

Different kinds of molecules have varying degree of surface attractions for each other. When the surface attraction is relatively strong, the molecules of one substance cling to the surface molecules of the other substance without being drawn into the internal chemical structure of the second substance. This surface phenomenon is known as adsorption.

Chromatography is a term used to describe the analytical techniques, which separate, the various compounds in a mixture according to relative adsorption potential of their molecules. This technique is used in the analysis of organic substances. The two main systems are:

Table 31.36: Recommended qualitative colour tests (bedside tests)*

1. Trinder's test: (Salicylic acid, including Acetyl salicylic acid - aspirin. Also known as ferric chloride test) Add 100 ml of Tinder's reagent (a mixture of 40 gm of mercuric chloride in 850 ml of water and 120 ml of aqueous hydrochloric acid (1 mol) and 40 gm of hydrated ferric nitrate, diluted to 1 l with water) to 2 ml of urine and mix for 5 seconds. A violet color indicates the presence of salicylate. If only stomach contents or scene residue are available, first hydrolyze the same by heating with 0.5 mol/L hydrochloric acid on a boiling waterbath for 2 minutes, and neutralize with 0.5 mol/L sodium hydroxide before performing the test. If a positive result is obtained in this test, carry out a qualitative assay on plasma/serum.
2. FPN test: (Phenothiazines) Add 1 ml of FPN reagent (a mixture of 5 ml of aqueous ferric chloride solution (50 gm/L), 45 ml of aqueous perchloric acid (200 gm/kg) and 50 ml of aqueous nitric acid (500 ml/L) to 1 ml of sample and mix for 5 seconds. Colors ranging from pink to red, orange, violet or blue suggest the presence of phenothiazines. TLC should confirm positive results.
3. Forrest test: (Imipramine and related compounds) Add 1 ml of Forrest reagent (a mixture of 25 ml of aqueous potassium dichlorate (2g/L), 25 ml of aqueous sulfuric acid (300 ml/l), 25 ml of aqueous perchloric acid (200 g/kg) and 25 ml of aqueous nitric acid (500 ml/L) to 0.5 ml of sample and mix for 5 seconds. A yellow-green color deepening through dark green to blue indicates the presence of imipramine or related compounds. TLC should confirm positive results.
4. Fujiwara test: (Trichloro compounds, including chloral hydrate, chloroform, dichloral phenazone and Trichloroethylene) To three 10 ml tubes labeled A, B and C, add respectively 1 ml portion of – sample, purified; water (blank test—essential); and aqueous Trichloroacetic acid (10 mg/L). Add 1 ml of sodium hydroxide solution (5 mol) and 1 ml of pyridine to each test-tube. Mix carefully and heat in a boiling waterbath for 2 minutes. An intense red/purple color in the top (pyridine) layer of the tube A as in tube C indicates the presence of Trichloro compounds; tube B should show no coloration.
5. O-cresol/ammonia test: (Paracetamol, Phenacetin) Add 0.5 ml of concentrated hydrochloric acid to 0.5 ml of sample, heat in a boiling waterbath for 10 minutes and cool. Add 1 ml of aqueous Ocresol solution (10 gm/L) to 0.2 ml of hydrolysate, add 2 ml of ammonium hydroxide solution (4 mol/L), and mix for 5 seconds. A strong blue to blue-black color, which forms immediately, indicates the presence of Paracetamol or Phenacetin. If a positive test result is obtained in this test, carry out a quantitative assay for Paracetamol on plasma or serum.
6. Dithionite test: (Paraquat, diquat) Add 0.5 ml of aqueous ammonium hydroxide (2 mol/L) to 1 ml of test solution, mix for 5 seconds and add about 20 mg of solid sodium dithionite. A strong blue to blue-black color indicates Paraquat; diquat gives a yellow-green color, but this is insignificant in the presence of Paraquat. If the color fades on continued agitation in air and is restored by adding further sodium dithionite, Paraquat or diquat is confirmed.
7. Dichromate test: (Ethanol and other volatile reducing agents) Apply 50 ml of potassium dichromate (25 gm/L in aqueous sulfuric acid (500 ml/L) to strip of glass fiber filter paper in the neck of test-tube containing 1 ml urine. Lightly stopper the tube and place in a boiling water-bath for 3 minutes. A change in color from orange to green indicates the presence of volatile substance. If a positive result is obtained in this test, carry out a quantitative assay for ethanol on blood.
8. Diphenylamine test**: (Chlorates and other oxidising agents) Carefully add 0.5 ml of diphenylamine (10gm/L in concentrated sulfuric acid) to 0.5 ml of filtered stomach contents or scene residue. A strong blue color, which develops rapidly, indicates the presence of oxidising agents.
9. Ferricyanide/ ferrocyanide test**: (Ferrous and Ferric iron) To 50 ml of filtered stomach contents or scene residue add 100 ml of aqueous hydrochloric acid (2 mol/L) and 50 ml of aqueous potassium ferricyanide solution (10 gm/l). To a further 50 ml of sample add 100 ml of hydrochloric acid and 50 ml potassium ferrocyanide solution (10 gm/L). A deep blue precipitate with potassium ferricyanide or ferrocyanide indicates the presence of ferrous or ferric iron. If a positive result is obtained in this test, carry out a quantitative assay for iron on serum.
10. Meixner test: (Poisonous mushroom) this test is done with stool sample. To a stool sample add methanol and centrifuge and filter it. Place one or two drops of this on a piece of filter paper and then add few drops of hydrochloric acid to them. A bluish coloration indicates the presence of anatoxins, which is present in most of the poisonous mushrooms.
11. Reinsch test: (Arsenic, antimony, bismuth and mercury) Applicable to urine, stomach contents and scene residues. Take a copper foil or mesh (5×10) or wire (2-3 cm), which is cleaned in nitric acid until the copper acquires a bright surface. Rinse the same with purified water and add 10 ml of concentrated hydrochloric acid and 20 ml of test solution in a 100 ml conical flask. Heat on a boiling waterbath in a fume cupboard for 1 hour. Maintain the volume of the solution by adding dilute hydrochloric acid as necessary. Color staining on the copper can be interpreted as follows:
    - Purple black: antimony
    - Dull black: arsenic
    - Shiny black: bismuth
    - Silvery: mercury.
12. Marquis' reagent test: (Morphine and other opium) Applicable to scene residue (fragments of suspected residue), stomach contents. Place a drop of Marquis' reagent (prepared by adding 3 ml of concentrated sulfuric acid and 3 drops of Formalin) on the suspected scene residue or add few drops to stomach contents. A purple coloration which gradually turns into violet and finally to blue color indicates the presence of opium and its derivatives.

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* First nine tests are from, Basic Analytical Toxicology-IPCS', WHO: Geneva, 1995.

** Tests for stomach contents or scene residue only.

- Gas chromatography (GC)
- Thin layer chromatography (TLC).

Gas Chromatography (GC)

Structure and Basics

Gas chromatography is essentially a thermal chamber or oven (Fig. 31.5) of precise temperature control, in which a long thin spiral tube called column, is installed. The interior of the column is packed with finely divided solid onto which a viscous liquid is coated. This liquid is known as stationary phase. One end of the column is equipped with an injection port, a self-sealing rubber septum, into which a sample of compound to be analyzed is injected by means of a gastight syringe. Once injected, the sample is forced through the column by pressurized streams of carrier gas known as the mobile phase. The carrier gas is usually helium. The sample can be either a gas or liquid form. If it is a liquid, the internal temperature of the injection port must be high enough to vaporise the liquid instantaneously.

Procedure

Box 31.2 summarises the procedure. As the sample passes through the column; its molecules are adsorbed by the liquid stationary phase. The molecules of different kinds have different degrees of attraction for the liquid. As the fresh carrier gas comes along, it tends to detach or desorbs, the molecules, which had been adsorbed by the stationary phase. Consequently, the molecules of the sample are continuously being adsorbed and desorbed as they move along the column. During this process the molecules of the sample separate and sort themselves out according to their attraction to the stationary phase. In general, lighter weight molecules move through the column faster than heavier weight molecules do (see Fig. 31.5).

When a test sample reaches the end of the column, the sample is separated into bands of like molecules. As they leave the column these bands pass through a detector. The most commonly used detector is the flame ionization detector. The bands of gas molecules pass through a flame of burning hydrogen gas, which ionises them in an electrical field. The ionized gases cause fluctuations in the resistance of the electrical field, thereby generating an electrical signal. The electrical signal is amplified and used to power a mechanical stylus (pen), which inks the read out, or chromatogram, into a strip of moving chart paper (see Fig. 31.5). By comparing this chromatogram with chromatograms of known reference standards made under identical conditions, the analyst is able to identify the compound present in the unknown mixture. The position of the peak along the chart paper (peak retention time) identifies the substance. The area of the peak indicates its approximate concentration.

Pyrolysis Gas Chromatography

The chromatograph by itself can analyze only liquids and gases but if a pyrolyzer is attached to the chromatography, organic solids can also be identified. Pyrolysis is the decomposition of solid materials by high heat into simpler volatile components. The pyrolyzer used in conjunction with the gas chromatograph in a small heating chamber that decomposes organic solids into gases in an oxygen free atmosphere. If oxygen were present, the gases would simply burn. These gases are then injected into the chromatograph and analyzed like any other gaseous sample. This process is generally known as pyrolysis gas chromatography.

Thin Layer Chromatography (TLC)

Thin layer chromatography is used to analyze a mixture of soluble organic compounds. This is mainly used to identify drugs, inks, etc.

Principle

Same as gas chromatography, but method differs. In the stationary phase there is a granular material that is coated on

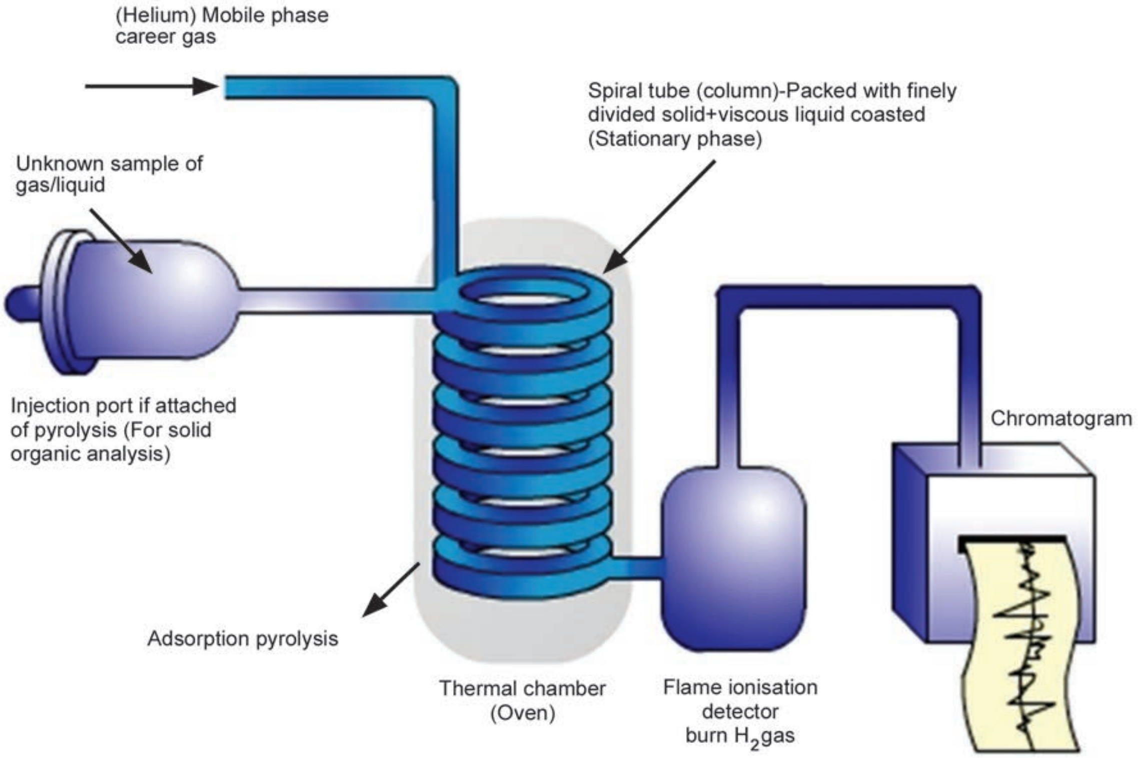


Fig. 31.5: Gas chromatography

Box 31.2: Summary of gas chromatography procedure

IN COLUMN
Molecules of sample gas/liquid — Undergo continuously adsorption,
desorbtion + separation – According to stationary phase
↓
Those with lesser tendency to adsorb move ahead of those with greater tendency, i.e. compounds with lighter molecular weight move faster than heavier weight molecules in bands, when made to pass through detector
↓
IN DETECTOR
Bands of gas molecule pass through burning H_2 gas and get ionized → in an electrical field → electrical signal
↓
IN CHROMATOGRAPHER
Amplified and used to power as pen writes a chromatogram (Graph)

a flat supporting plate, usually glass. The most common stationary phase material is silica gel. The mobile phase can be any one of a number of liquid solvents. TLC can analyze more than one substance at a time.

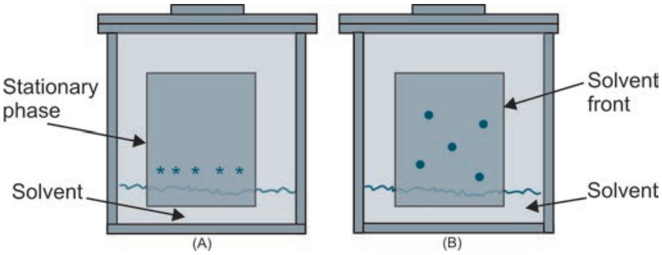
Procedure

The samples to be tested are applied in spots along a start line near the bottom of the plate. In a tightly closed developing chamber, the bottom edge of TLC plate is immersed in a small amount of the (mobile) phase solvent. The solvent begins to migrate up the plate by capillary action that is by a progressive surface attraction of the solvent to silica gel granules. When the solvent front reaches the start line where the test samples are spotted, the solvent dissolves them and carries them along with it as it migrates up the plate. The different molecules contained in the sample now show varying tendencies to become adsorbed to the silica gel and desorbed by moving solvent phase. After the solvent front has migrated a certain distance up the plate, usually 100 millimeters, the plate is considered to be developed and is removed from the chamber and dried in the air. During the development, the component parts of the samples become separated into individual spots at different distances from the start line (Figs 31.6A and B).

The distance that each compound travels up the plate during the development period is given a numerical value called the Rf value. The Rf value is determined by dividing the distance of the spot centre from the start line by the distance of the solvent front from start line. For example, if the spot of one compound has migrated 25 millimeters and solvent front has migrated 100 millimeters, the Rf value for that spot is 0.25, i.e.

$$\text{Rf} = \frac{25}{100} = 0.25$$

Rf values are always between 0.00 and 1.00. As long as the same stationary and mobile phases are used, a given compound will regularly reproduce the same Rf value. In most cases, the spots produced by the developed compound are completely colorless and therefore invisible without some detection aid. The first step in the detection process is to view the plate under ultraviolet light (UVL). Since many organic compounds show fluorescence under UVL, the position on the plate can be outlined with a pencil. The plates can be sprayed with a variety of chemical reagents that produce different color reactions with different compounds. Since the color reactions are often specific to certain organic compounds, they are considered along with Rf values in the identification of substance.



Figs 31.6A and B: *TLC Plates:* (A) At the beginning of tests and (B) After the development

Mass Spectrometers

Equipment

These are the most sensitive and accurate instruments used to identify organic compounds. Because of the prohibitive cost, only advanced laboratories have them. Each type of instrument has four systems.

- A system for introducing the sample
- An ionization system
- A system for sorting the ions according to ratio of their mass to their electric charge
- A detection and read out system.

Procedure

The sample compound must be introduced in gaseous form at a steady measured rate into the ionization chamber, where the gas is bombarded at right angles by a stream of energized electrons, which strike the molecules of the sample, causing these molecules to break up into ionic fragments with electrical charges. Since these fragments bear charges, they can be accelerated by being subjected to electrostatic forces. Positively charged particles fly towards the negative poles and negatively charged particles fly towards positive poles.

By varying the combinations of electrostatic forces in the path of accelerated particles, particles of any given mass to charge ratio can be selected out from all other particles. The selected particles are accelerated through the system until they strike a collector, but those that have a different mass to charge ratios are drawn off along the way. As the electrostatic fields are progressively changed, the particles of different mass to charge ratio are drawn to the collector. The particles striking the collector set up weak electrical signals, which are detected and amplified and converted into visual readout. The readout may be in the form of oscilloscope, an oscillogram, or a computerized digital printout. The given compound under the same conditions of ionization, temperature and pressure is always fragmented into the same kind of particles, and therefore can be very precisely identified by its mass spectra.

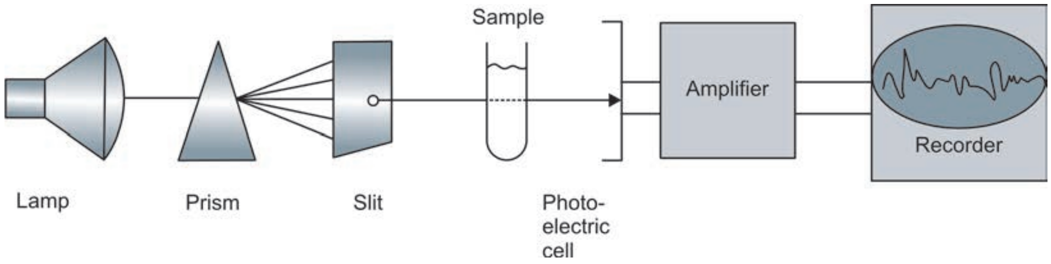


Fig. 31.7: Schematic diagram of a spectrophotometer

The gas chromatograph as seen earlier above is an ideal instrument for separating mixtures into pure bands of their component compounds. The main difficulty that had to be overcome was the gas chromatograph operates under high pressure and the mass spectrometer operates nearly at vacuum. Several interfacing systems exist now for collecting a pure sample of gas from GC, separating it from the carrier gas, and introducing it under appropriate pressure into the mass spectrometer.

Spectrophotometers

The materials of the same chemical composition always absorb the same wavelength of light, the absorption characteristics of an unknown substance can be used to help identify it or compare it with a known standard. This field of study is called absorptiometry, the analytical instrument based on it *are spectrophotometers.*

The principle absorption cannot be entirely explained by the wave theory of light because they concern the energy present in light. The light waves are made up of particles of energy called photons. The higher frequency wavelengths of light towards the blue end of the spectrum are more energetic than those of lower frequency towards the red end of the spectrum. When substance absorbs light, its atoms and molecules are actually absorbing energy in the form of photons and each substance can only accept photons that reach it at certain specific wavelengths.

Ultraviolet, Visible and Infrared Spectrometers

All the organic materials we have to deal with absorb light in either the ultraviolet, visible or infrared range. When a photon is absorbed it causes the atom that absorbed it to rise from its normal, or ground state to more highly energized state with ultraviolet and visible light. This is accomplished by the shift of electron from a lower to higher energy orbit. In the infrared range, the increased energy causes an increase in vibrational stretching and bending of atoms or groups of atoms continuously taken place within every molecule. The basic operating principles of ultraviolet, visible light and infrared spectrometers are fairly simple.

Procedure

The energy of a spectrophotometer is a lamp that radiates a band of wavelength appropriate to analytical system. The radiation is passed through a prism, which disperses it into component wavelengths. The dispersed beam is projected against a barrier that has a slit in it, through which the required wavelength of beam is allowed to pass (Fig. 31.7).

This beam is then made to pass through the sample, which absorbs practically its characteristic wavelength, and then falls on the photoelectric cell and the intensity measured. A series of such readings with different wavelengths are measured and a curve constructed. Each substance has a characteristic curve by which it is identified and is called as “absorption spectrum.” The ultraviolet, visible light and infrared Spectro-photometery is especially suited for analysing organic compounds.

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THE POISON INFORMATION AND CONTROL CENTRE SYSTEM

The poison information centre (PIC) movement started first in Netherlands in 1949, followed by United States in 1950’s. Guy’s Hospital, London, and the Illinois Chapter of American Academy of Pediatrics at Chicago, Illinois, established National Poison Information Centres, almost simultaneously in early 1960’s, creating an sentience of need and formation of similar centres in other parts of the world, and rendering invaluable service of promoting awareness on poisons and poisoning, promoting diagnostic and therapeutic assistance to the physician in managing poisoning cases as needed through these centres.[1-7] The early canters, called the poison control centres (PCC), were cataloguing services providing information to the physician about the contents and toxicity of common hazardous products.[2]

The current system of care of the victim of poisoning, which has evolved over the last four decades, focuses through a regional poison information centre network, on imparting the most recent

Table 31.37: Global information on countries with poison information centres (PIC)

Australia	India	Netherlands *	South Africa	USA
Brazil	Indonesia	New Zealand	Spain *	Zimbabwe
England #	Iran	Pakistan	Sri Lanka	
France #	Ireland *	Philippines	Turkey	
Germany #	Kenya	Singapore	Uruguay	

* PIC works integrated with Diagnostic Labs;
# PIC works in conjunction with Acute Poisoning Treatment Unit.

information on poisoning management. The system also integrates training and certification of poison centres and related health care givers, with a national data collecting system and a computerized poison centres and related health care givers, with a national data collecting system and a computerized poison information database.[7]

The first poison control centre opened in 1953 in Chicago, and initially developed a loose leafed guide to the ingredients in common household products and the treatment of poisoning with these product. The National Clearing house for Poison Control Centres, which became operational in 1957, provided a centralized focal point for collecting and disseminating information among the 17 then existing poison control centres.[2]

The official name of the centre has been debated and changed to “Poison Information Centre” which in a way reflects the role of the centres, while at another level, as this will perform Poison Control which is the another role of these centres. Developed in collaboration with the Rocky Mountain Poison and Drug Centre and University of Colorado Health Science Centre, the POISINDEX system has been published since 1974. Today it provides information on more than 800,000 poisonous substances to the needy in no time, by computerized information resource network.[2,7]

India established her first National Poison Information Centre at All India Institute of Medical Sciences, New Delhi, in 1994. The INTOX is computer software on poisons by WHO is available for use in such centres presently. Ahmedabad hosts the second such centre in the country at the National Institute of Occupational Health. Table 31.37, provides the list of countries of the world possessing a Poison Information Centre.[7]

ROLE OF POISON CENTRES

The primarily its role is to get the public’s primary access to poison centre services is through a network of toll-free telephone systems. Poison centre toxicologists and information specialists are consulted here daily. Basically the centre has to play a preventive role of poison centres:

Poison centres are the frontline responders to poison emergencies and are instrumental in the surveillance of adverse effects of marketed products, drugs and food and in the promotion of poison prevention.

STAFFING PATTERNS AND FINANCIAL CONSIDERATIONS [2]

A 1989 survey found that a typical regional poison centre is located at a university medical centre or a hospital with a university affiliation, serves a population of approximately 4 million people; handles about 35,000 human exposure cases every year. It is staffed by a physician, a medical director, a pharmacist or a nurse who acts as the technical director, an administrator, an average of 10 poison information specialists, a secretary and a staff member devoted to community education, with a total operating budget of US$ 650,000. According to the American Association of Poison Control Centres (AAPCC) Toxic Exposures Surveillance System (TESS) Data for 1992, more than 1.3 million human poison exposures were managed by poison centres at home or site of exposure, thus the economic justification for funding poison centres becomes very clear.

Traditionally, poison control centre programs relied on individual sponsoring hospitals for the majority of their operating funds. Today, increasing fiscal constraints imposed on the health care system has produced challenges for the poison centres.

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Chapter

32

Corrosive Poisons

Corrosive poisons are those substances, which corrode (means –'*to eat away*') and destroy tissues through direct chemical action. They almost always act locally and have few systemic effects. They can be classified into two groups:

1. **Strong Acids**
    - *Inorganic acids (mineral acids):* Sulphuric, nitric, hydrochloric and hydrofluoric acids.
    - *Organic acids:* Carbolic, oxalic and salicylic acids. These acids are weaker in action compared to inorganic acids and are usually absorbed into circulation promoting local and remote action.[1]
2. **Strong Alkalies**
    - Anhydrous ammonia
    - Potassium hydroxide
    - Sodium hydroxide
    - Ammonium carbonate
    - Potassium carbonate
    - Sodium carbonate

INORGANIC ACIDS: SULPHURIC ACID, NITRIC ACID, HYDROCHLORIC ACID

Physical Properties

Table 32.1, provides a comparative account of specific characteristics of all the three acids.

Action

Only strong acids and alkalies act as corrosives. Dilute acids and alkalies act as irritants. Strong acids produce *coagulation necrosis* (Fig. 32.1) characterized by the formation of a coagulum (eschar) as a result of the desiccating action of the acid on proteins in superficial tissues.[1] The coagulum formed limits the penetrating ability of acids. On the other hand, strong alkalies create injury to tissue by the mechanism of *liquefaction necrosis*. Alkalies, unlike acids, produce extensive penetrating damage.[1,2] Squamous epithelium of oesophagus is more resistant to acids than columnar epithelium of stomach. On the contrary, squamous epithelium of oesophagus is more sensitive to alkalies than columnar epithelium of stomach. Hence oesophageal strictures are more common in alkali poisoning and pyloric and gastric strictures are more common in acid poisoning.

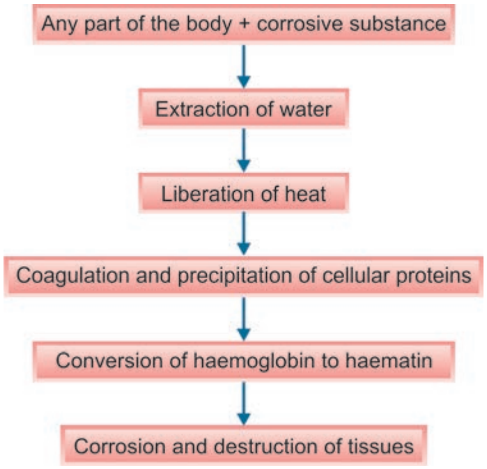

Any part of the body + corrosive substance
Extraction of water
Liberation of heat
Coagulation and precipitation of cellular proteins
Conversion of haemoglobin to haematin
Corrosion and destruction of tissues

Fig. 32.1: Mechanism of coagulation necrosis by mineral acids

Table 32.1: Comparative aspects of specific characteristics of sulphuric, nitric and hydrochloric acids

Acids	*Sulphuric acid (Sulphuric acid)*	*Nitric acid*	*Hydrochloric acid*
Synonyms	Oil of vitriol	Aqua fortis, spirit of nitre	Spirit of salts, muriatic acid
Physical properties	Heavy	Heavy	Heavy
	Colorless	Colourless	Colourless
	Viscid/oily	Not so	Not so
	Non-fuming	Fumes (yellow) in air	Fumes in air
	Gives heat with water	Not so	Not so
	Charring +ve	*Xanthoproteic reaction* +ve (refer below)	Not so
	Burning acid taste	Choking odour	Not so
Fatal dose	5-10 ml	10-15 ml	15-20 ml
Fatal period	12-24 hr	24-30 hr	15-24 hr
Commercial use	Textile, arts and industries	In industries	Cleansing agent

Signs and Symptoms

Certain common gastrointestinal and respiratory signs and symptoms on oral consumption of the corrosives are presented in Figures 32.2 and 32.3 respectively. Figure 32.2 shows the gastrointestinal signs and symptoms seen in all inorganic acid poisonings except hydrofluoric acid, but vary in intensity depending on the type and concentration of the acid. Figure 32.3 shows the respiratory signs and symptoms in all inorganic acid poisons *except* hydrofluoric acid. Oral cavity presents with chalky white teeth, swollen, sodden, blackish tongue and swollen lips. Acid burn (Eschar) is commonly seen as vomited acid trickles down the angle of mouth towards the neck and chest. Abdomen may be distended and tender. Mind remains clear till death.

Treatment

In the management of a case of corrosive poisoning certain common measures are advised, and they are:

Avoid

- Gastric lavage as to prevent gastric perforation (exception: organic acids).
- Administering carbonates, which can yield CO_2, producing distention and perforation.
- Emesis is not attempted for fear of rupture of the stomach.

Give

- *Demulcents:* milk (canned condensed milk), egg white (beaten), vegetable oils, starch solution, barley water, thin gruel, etc.
- Immediate dilution with milk or water within 30 minutes postingestion is widely recommended for oral ingestions despite the fact that tissue injury occurs rapidly. Do not attempt to neutralize the acid with weak bases/dilute alkalizers such as milk of magnesia or lime water (i.e. calcium hydroxide in water), etc., since exothermic reaction may extend the corrosive injury.[2] Also never give strong alkalies or alkalies such as sodium bicarbonate as it can produce gas carbon dioxide which in turn increases the risk of perforation of stomach.

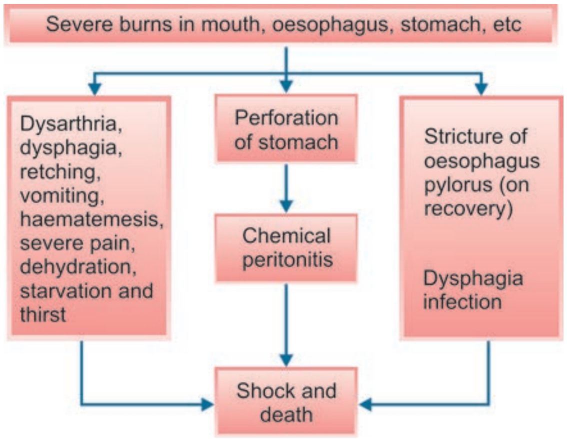

Severe burns in mouth, oesophagus, stomach, etc
Dysarthria, dysphagia, retching, vomiting, haematemesis, severe pain, dehydration, starvation and thirst
Perforation of stomach
Chemical peritonitis
Stricture of oesophagus pylorus (on recovery)
Dysphagia infection
Shock and death

Fig. 32.2: Gastrointestinal signs and symptoms

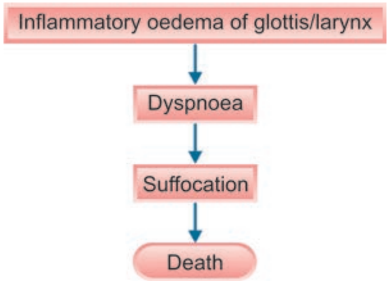


Fig. 32.3: Respiratory signs and symptoms

- Airway maintenance and artificial respiration if there is any respiratory distress.
- Morphine/pethidine for relief of pain.
- Intravenous fluids and electrolytes for dehydration.
- No oral feed until endoscopy confirms the extent of injury.
- Antibiotics for control of infections.
- Corticosteroids although proved to be good in delaying or preventing stricture formation in experimental animal studies; there is a controversy in recommending it for human beings.[2]
- *Skin care* for any skin lesions involves copious saline irrigation. Treat with nonadherent gauze and wrapping. Deep second-degree burns may benefit from topical silver sulfadiazine.[1]
- *Eye care* for any acid injuries to the eyes requires copious irrigation with retraction of eyelids for 20-30 minutes. Antibiotic eye drops can help combat infections.[3] However, referring to an ophthalmologist for further treatment is prudent.

Causes of Death

Causes of death could be immediate or delayed. *Immediate* causes are suffocation and asphyxia due to oedema of glottis; exhaustion; and shock due to circulatory collapse or perforation of stomach, while *delayed* causes are starvation and dehydration due to stricture oesophagus or pylorus. The victim may also die due to peritonitis or secondary infections.[1]

Postmortem Findings

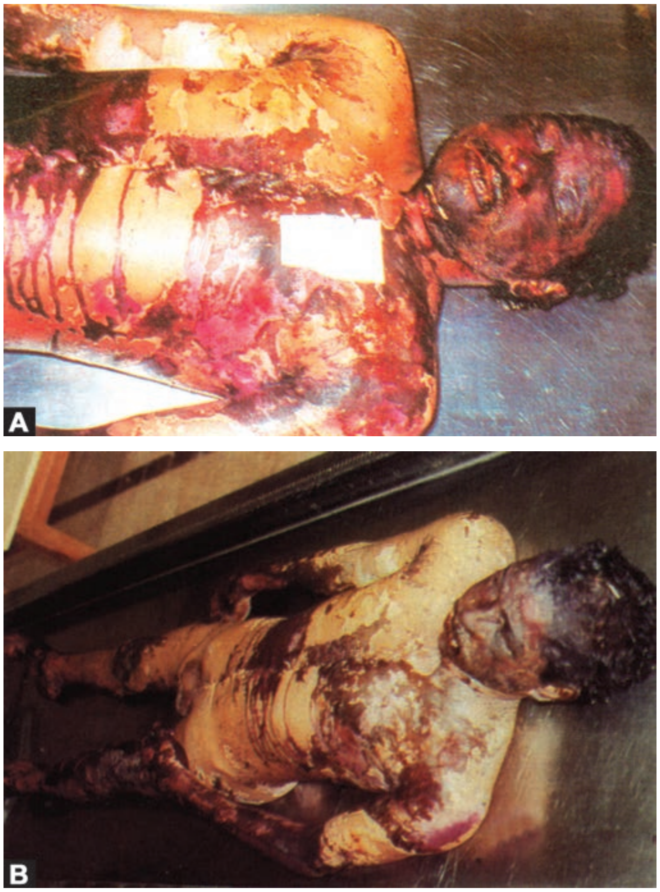
- *External:* Corrosion of parts that come in contact with it (Figs 32.4A and B) especially lips, mouth, throat, chin, angle of mouth, hands, etc. and also clothing, furniture, etc. when spilt over them.
- *Internal:* The findings are restricted to the upper gastrointestinal and respiratory tract and they are:
  - *Pharynx and oesophagus:* Inflammatory changes with oedema and bleeding.
  - *Stomach:* It is converted into a soft, boggy black (sulphuric acid)/yellowish (nitric acid)/brownish (hydrochloric acid), which disintegrates on touch. Contentstarry black or brown (Fig. 32.5).
  - *Surrounding viscera:* Sloughing is seen if stomach wall is perforated. Perforation is most common in case of sulphuric acid.
  - *Larynx and trachea:* These show corrosion and inflammation. Congestion of the respiratory tract is commonest in case of nitric acid poisoning due to inhalation of acid fumes.

Chemical Tests

Sulphuric acid: With barium chloride or nitrate it produces white precipitate of barium sulphate.

Nitric acid: Nitric Acid when added by the side of a test tube containing a mixture of ferrous sulphate and sulphuric acid, a brown ring appears at the interphase.

Hydrochloric acid: When mixed with silver nitrate, it produces a curdy white precipitate of silver chloride.



Figs 32.4A and B: Case of accidental spillage of corrosive acid and burns—autopsy findings (external): (A) Face and trunk, (B) Entire body

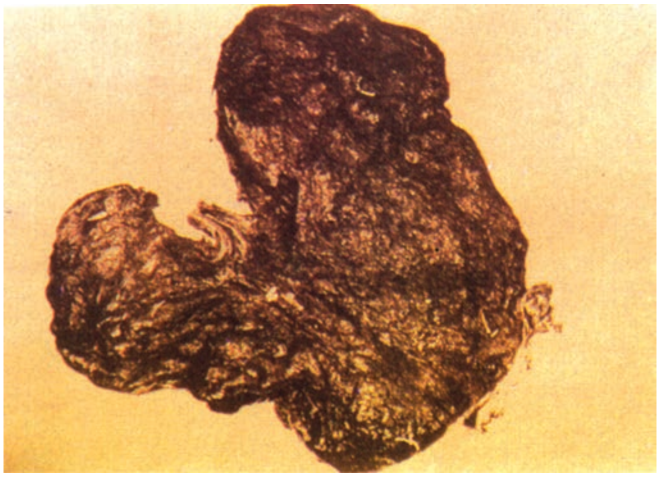
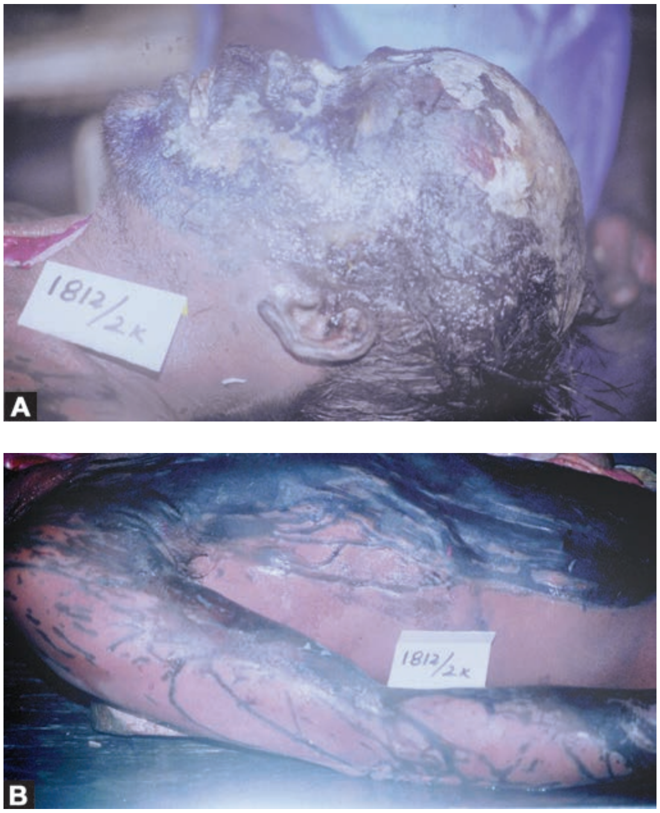


Fig. 32.5: Postmortem appearance of stomach in concentrated sulphuric acid poisoning victim (note—swollen, blackish, corroded, desquamated stomach mucosa)

Medicolegal Importance

1. Most of the reported cases of acid poisoning are of accidental source. Sulphuric acid is often mistaken for glycerine or castor oil and consumed.
2. Rarely used for committing suicide.
3. Extremely rare in committing homicide.
4. Occasionally used as an abortifacient.



Figs 32.6A and B: Case of vitriolage by throwing concentrated sulphuric acid. *Note:* face has been burnt and disfigured completely, front of the trunk also presents tell-tale evidence of acid track. (*Courtesy:* Capt. Dr Santha Kumar, Professor and HOD, Forensic Medicine, Govt. Stanley Medical College, Chennai, Tamil Nadu)

5. Vitriolage (vitriol throwing)—Throwing of concentrated sulfuric acid on the face of a person with an intention to produce damage, disfiguration or death is known as *Vitriolage* (Figs 32.6A and B).

   Clinical appearance—Chemical burns over the face and parts of the face wherever the acid comes in contact.

   Treatment—Comprises of following steps:
   - Wash the area with copious water and soap/mild alkali
   - Give analgesics for relief of pain
   - Use antibiotics to prevent infections.

   Medicolegal Importance
   - It can result in permanent disfiguration (Section 320 IPC—*Grievous hurt*)
   - If it is done out of jealousy or enemity, to facilitate robbery, other injuries may also be present.
6. *Xanthoproteic reaction*—It is a local reaction producing yellowish discoloration of the tissues upon contact with concentrated nitric acid, e.g. yellow discoloration of the skin of fingers on touching the acid.

   Mechanism—Concentrated nitric acid whenever encounters organic matter, reacts and destroys it by oxidation, producing picric acid, which is yellowish orange in colour and stains the tissues deeply.

   Medicolegal Importance—Presence of xanthoproteic reaction change confirms nitric acid poisoning.
7. *Criminal disposal of dead body*—Attempts of disposal of dead body after killing has been reported in history (*Acid bath*

murder case—John George Haigh killed a lady in his warehouse and tried to dispose her dead body by dumping in a steel tank filled with sulphuric acid. However the remains in the tank which were recovered later by police included human foot, few bone fragments, some gall stones and a partially dissolved set of artificial dentures gaving clue to the crime. This was followed by confession of murder by Haigh earlier and disposal the dead in same way.

HYDROFLUORIC ACID

Physical Properties

Hydrofluoric acid is a colorless gas, which becomes a fuming liquid when dissolved in water and is used for etching glass and clouding electric bulbs.

Fatal Dose

About 15 ml.

Fatal Period

Few minutes to 2 hours, However there has been cases reported of death occurring in 7 hours of poisoning.[4,5]

Action

Mode of action of hydrofluoric acid exposure is the major exception to other inorganic acids. The fluoride anion produces a liquefaction necrosis by binding with calcium and magnesium in the tissues. This also results in hypocalcemia, rendering hydrofluoric acid poisoning more serious.[6]

Signs and Symptoms

Following ingestion, patient presents with hematemesis, hypovolemic etanic convulsions, upper airway obstruction, severe hypocalcemia, acidosis, shock and coma.[3] Myocardial irritability and subsequent life threatening cardiac arrhythmias may be due to binding of potassium, magnesium and calcium ions.[6] Skin exposure can result in severe and deep burns, which are extremely painful and slow to heal.[6]

Treatment

Following is recommended:[6]

- Acid burn lesions need copious irrigation with water, application of calcium gluconate gel, and debridement if needed.
- Intra-arterial infusion of 20 per cent calcium gluconate or calcium chloride is effective.
- Oxygen inhalation after removal from fumes and tracheotomy, if needed, should be done.

Postmortem Findings

Lips, tongue and mouth may show white patches or may be charred oesophagus may show shredded epithelium with ecchymosis, inflammation, ulceration, and blackening of the stomach. Liver and kidneys show fatty and parenchymatous degeneration.

Medicolegal Importance

Usually accidental poisoning.

CARBOLIC ACID (Phenol)

Physical Properties

It is a poison that can be identified by smell which is commonly referred to as *phenolic odour* or *hospital odour*. Pure phenol has a colourless, short, prismatic needle-shaped crystalline form. On exposure to air it turns pink and liquefies. It is *fat-soluble*, hence can attack nervous system. It is also soluble in *glycerine, ether, alcohol* and slightly in *water*. It is known specifically for its *antiseptic* or *disinfectant* property.

Other Members of Phenol Group

Phenol has several derivatives, namely cresol, creosote, Lysol, dettol, etc. The toxicological actions of these compounds are similar to phenol but are less severe.

- Cresol—is a methyl phenol with meta, ortho and para isomers. It is used as a disinfectant and antiseptic.[7]
- Creosote—is a mixture of phenols and consists mainly of cresol and guiacol. It is used as household remedy for coughs and is found in many proprietary preparations.[7]
- Resorcinol—is a colourless crystalline substance and is used for the treatment of various skin diseases including ringworm, psoriasis, eczema, etc.[7]
- Lysol—is 50 per cent solution of cresol (3-methyl phenol) in saponified vegetable oil.[7,8]
- Dettol—is chlorinated phenol, parachlorometaxylenol (PCMX 4.8% solution in isopropyl alcohol) and is practically non toxic to adults. However some fatalities have been reported among children.[8]
- Thymol—is an alkyl derivative of phenol obtained from volatile oils of Thymus vulgaris, Monarda punctata or Trachyspermum ammi. It occurs in colourless crystals with characteristic pungent odour and taste. It was used earlier as an antihelminthic (for ankylostomiasis), antifungal and antiseptic.[8]

Routes of Absorption

It is absorbed through:

- Intact skin by local application or spillage
- Gastrointestinal tract by oral ingestion
- Respiratory tract by inhalation
- Per rectum
- Per vaginum.

Action

Phenol is a protoplasmic poison. It enters into loose combination with proteins and penetrates deep into the tissue. When applied to skin or mucosa it causes necrosis and gangrene. The local nerve endings are first stimulated and then paralysed, resulting in anesthesia. After absorption it causes widespread capillary damage and clotting in superficial blood vessels. It also acts on the cells of central nervous system, heart and kidneys.[7]

Metabolism and Excretion

Phenol is metabolized mainly by the kidneys, wherein it gets converted into *hydroquinone* and *pyrocatechol* and excreted in urine (both). These products turn urine *olive green* or *brown* on standing and the phenomenon is called *carboluria*. Complete elimination occurs in 36 hours. However, phenol is considered as a *nephrotoxic drug*. *Other nephrotoxics* comprise of heavy metals, methanol, oxalic acid, salicylates, phenacetin, EDTA and penicillamine.

Signs and Symptoms

Poisoning by carbolic acid is known as *carbolism*. All the signs and symptoms mentioned in Figure 32.2 and 32.3 are presented by carbolic acid. Certain unique ones specific to this poison are presented in Figure 32.7. However, the usual signs and symptoms of *carbolism* are enumerated below.

- Headache, giddiness, tinitus
- Vomiting, diarrhoea and pain abdomen

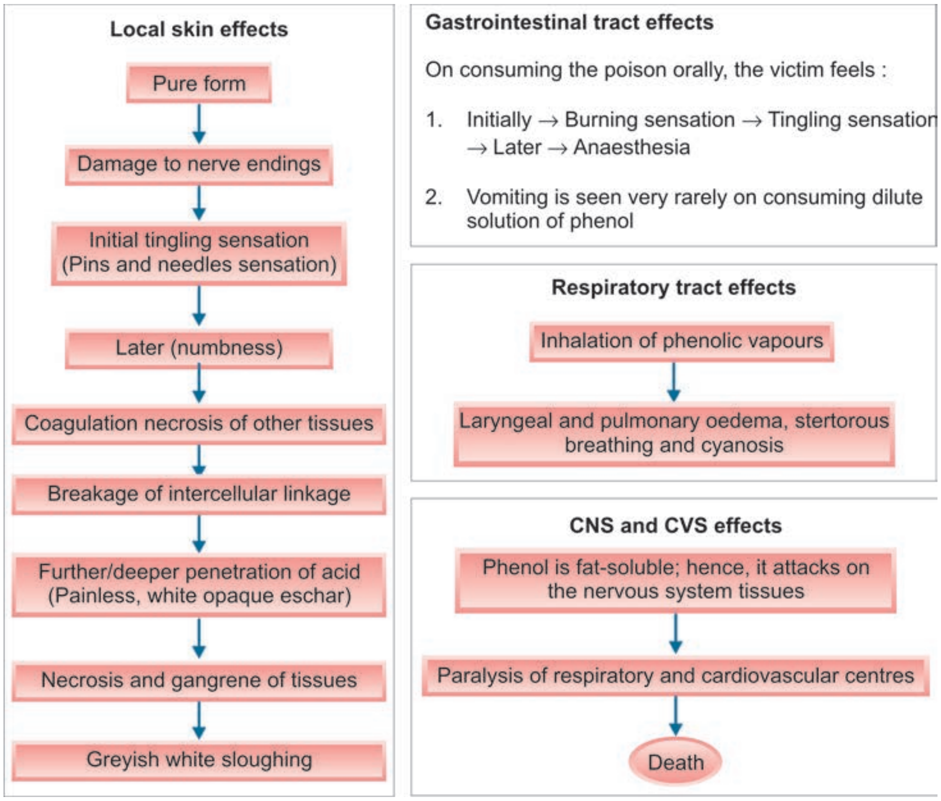

Local skin effects
Pure form
Damage to nerve endings
Initial tingling sensation (Pins and needles sensation)
Later (numbness)
Coagulation necrosis of other tissues
Breakage of intercellular linkage
Further/deeper penetration of acid (Painless, white opaque eschar)
Necrosis and gangrene of tissues
Greyish white sloughing
Gastrointestinal tract effects
On consuming the poison orally, the victim feels :
1. Initially → Burning sensation → Tingling sensatio
→ Later → Anaesthesia
2. Vomiting is seen very rarely on consuming dilute solution of phenol
Respiratory tract effects
Inhalation of phenolic vapours
Laryngeal and pulmonary oedema, stertorous breathing and cyanosis
CNS and CVS effects
Phenol is fat-soluble; hence, it attacks on the nervous system tissues
Paralysis of respiratory and cardiovascular centres
Death

Fig 32.7: Specific signs and symptoms of carbolic acid

- Muscular spasms and convulsions
- Collapse—unconsciousness, coma
- Clammy, cold, sweating body
- Stertorous breathing with cyanosis
- Pupils dilated
- If survives for 48 hr—*carboluria* followed by *anuria*. Victim will pass dark, smoky urine which soon turns olive green on standing.
- Methemoglobinaemia is a characteristic feature in severe cases.
- Death may result from respiratory and circulatory failure.

Diagnosis

- Corrosions on face, around and inside the mouth (grayish white if phenol or brownish, if Lysol)
- Phenolic odour (breath/vomitus)
- Carboluria
- Dilated pupil
- Stertorous breathing.

Fatal Dose

5-15 gm.

Fatal period—2-12 hours (Rapid death if injected intrauterine).

Toxicity rating—4.

Treatment

It depends on route of administration.

In case of poisoning through skin absorption, perform the following:

- Remove the contaminated garments
- Cleanse the site by mopping with wet cloth and wash with soap and water
- Apply olive oil/ methylated spirit/ 10 per cent ethyl alcohol, which can prevent further absorption
- Shift the victim to fresh atmosphere and make him breath in fresh air
- Give normal saline + sodium bicarbonate (I/V- drip).

In case of poisoning through oral route, perform the following:

- Perform gastric lavage: Though phenol corrodes the stomach wall, it also hardens it unlike other corrosive poisons. Hence gastric lavage is performed whenever possible with plenty of lukewarm water containing animal charcoal, olive oil, magnesium or sodium sulphate or saccharated lime, soap solution, 10 per cent glycerine, etc. When the lavage is completed, 30 gm of magnesium sulphate or medicinal liquid paraffin should be left in the stomach.
- Give egg white: Epsom salt/demulcents orally.

Symptomatic

- Artificial respiration.
- Tracheal aspiration of froth/secretions.
- Glucose saline to induce diuresis.

Postmortem Findings

- *External:* Greyish or brownish corrosions at the angle of the mouth, chin tracks, in front of the body, arms and hands (splashes) with characteristic phenolic odour.
- *Internal:* Corrosion of gastrointestinal mucosa, laryngeal and pulmonary oedema has been observed in all orally ingested poisoning cases. However certain *specific findings* observed, as listed.
  - — Stomach changes: Opening the stomach will emit the *phenolic odour*. The stomach wall is usually hardened and due to hardening, the stomach will have a leather

bottle appearance, and is called as *leathery stomach*. Gastric mucosa will present with *marked corrosion* and swelling of mucosal folds with coagulated greyish or brownish silvery mucus on it. Intervening normal mucosal folds appear dark red in colour.
  - — Kidney will show *haemorrhagic nephritis* when the victim survives for some time after poisoning.
  - — Vomitus and gastric lavage collection may show partially detached gastric mucosa.
- *Preservative used for sending viscera for chemical examination:* Supersaturated solution of sodium chloride.

Medicolegal Importance

- Often phenol is used as a *disinfectant* and *preservative* for vaccines and sera.
- Fatalities due to phenol occurred more frequently in the past than at present time. Accidental poisoning still occurs, particularly in homes.[7] Cases of poisoning are usually due to *accidental* consumption or *spillage* over the body.
- Phenol is rarely *suicidal* and *homicidal* due to the strong phenolic *odour.*
- Phenolic solution is used as an *abortifacient* by quacks for syringing it into the uterine cavity to induce *criminal abortion* even today.[7]
- *Ochronosis*—is a condition associated with *alkaptonuria*, an inborn error or metabolism, in which *homogentisic acid* gets deposited in the cartilages, ligaments and fibrous tissues. It is sometimes seen in *chronic phenol poisoning* (infrequent these days) wherein the phenolic metabolites *pyrochatechol* and *hydroquinone* gets deposited in the cartilages and ligaments casuing dark pigmentation.[7]

OXALIC ACID (Acid of Sugar)

Physical Properties

It is a colorless, prismatic crystalline substance, similar to magnesium sulphate ($MgSO_4$) and zinc sulphate ($ZnSO_4$). However, certain differences are depicted in Table 32.2.

Fatal dose—15-20 mg

Fatal period—1-2 hours

Toxicity rating—4

Action

- Acts locally as a corrosive, on both skin and mucosa (more severe)
- Remotely on absorption into blood affects several systems, the important ones are:
  - — Electrolyte → Extracts tissue calcium → Hypocalcaemia
  - — Cardiovascular system → Shock → Death
  - — Renal system → Tubular necrosis → Uraemia → Death

Signs and Symptoms

Presents in three forms:

- *Fulminating*: With large doses (15 gm or more) orally can lead to sour and acidic taste, followed by a sensation of constriction around throat and burning pain from mouth to epigastrium, which radiates all over the abdomen. There will be tenderness in the epigastrium, nausea, followed by vomiting (coffee ground coloured vomitus), severe thirst, diarrhoea, electrolyte imbalance and death.

Table 32.2: Differences between oxalic acid, and magnesium and zinc sulphates

Properties	*Oxalic acid*	*$MgSO_4$/Zn SO_4*
Taste	Sour and acidic	Bitter
Reaction	Strongly acidic	Neutral
On heating with sodium	Sublimates	Not so
On heating with bicarbonate	Effervesces	Not so
On heating with ink stains	Disappears	Not so

- *Acute*: All findings are mainly due to hypocalcaemia such as—muscle irritability, tenderness, tetany and convulsions, tingling of extremities, coma, collapse and death.
- *Delayed*: Findings will be of uraemia. Urine will be scanty with traces of albumin, blood and calcium oxalate crystals (seen microscopically as envelope-shaped crystals).

Treatment

- Gastric lavage with calcium lactate (2 teaspoon per lavage)
- *Antidotes*: lime water, calcium lactate, calcium gluconate, calcium chloride, chalk suspension in water or milk, etc. may be given orally, act as specific antidotes, which form insoluble calcium oxalate and are excreted easily.
- 10 ml calcium gluconate I/V frequently
- Parathyroid extracts: 100 units I/M
- Demulcent drinks
- Bowel wash by enema and purgatives (castor oil)
- Symptomatic measures.

Postmortem Findings

- *External:* No specific findings. However, burns of the face and skin rarely seen.
- *Internal:* (specific findings) Mucosa of the mouth, tongue, pharynx and oesophagus are bleached (whitened/scald/red), if a strong solution is consumed.

Stomach changes

- — The stomach mucosa is reddened and punctate due to erosions giving "*velvety red*" or blackish appearance
- — Wall of the stomach is softened, but no perforations
- — Contents: Gelatinous brown (due to acid hematin formation).

Kidney changes: Swollen and congested. Tubules on histopathological study reveal to be filled with oxalate crystals.

All other viscera: Congested.

Medicolegal Importance

- Usually consumed accidentally (mistaken for magnesium sulphate)
- Suicidal or homicidal uses are rare due to the taste
- As an abortifacient: occasionally used to induce criminal abortion
- Used for illegal erasure of signatures
- It is detected in certain vegetables as oxalates, e.g. beets and in the leaves of spinach, rhubarb, cabbage, etc and many other vegetables,[7] rarely causing poisoning.
- Commercial uses
  - — Cleaning or bleaching leather
  - — Book biding
  - — Calico printing
  - — Removal of iron moulds from linen
  - — Removal of the ink stains.

FORMIC ACID (Methanoic Acid, Formylic Acid)

Formic acid is a colorless liquid with pungent penetrating odour, completely soluble in water.[2,9] It is used as kettle de-scales or bath cleaner. Generally airplane glue makers, cellulose formate workers and tanning workers are exposed to a 60 per cent solution of formic acid.

Signs and Symptoms

On coming in contact with skin it produces brownish discoloration, dermatitis, pustules, vesicles and sometimes sloughing.

Formic acid is unique for its ability in many patients to cause death after a prolonged (several weeks) course of classical acid-induced gastrointestinal damage. Certain other complications include severe metabolic acidosis, intravascular haemolysis, and disseminated intravascular coagulation.[10,11]

Accidental ingestion in children ordinarily does not lead to fatalities since the pungent taste prevents ingestion of lethal dose. It is nevertheless a problem when used deliberately for suicide. It causes acute tracheobronchitis, characterized by cough, sore throat, chest pain and light-headedness. Formic acid skin burns may also result in systemic toxicity.[12] When absorbed by the body, it causes systemic acidosis, haematuria and renal damage. Metabolism of *methanol* can also produce toxic metabolites of formic acid.[2]

Treatment

Treatment is by correction of acidosis by infusion of sodium bicarbonate intravenously and to treat renal failure by haemodialysis.

Medicolegal Importance

- Used in dyeing colorfast wool, in electroplating, coagulating latex rubber, regenerating old rubber, and dehairing and tanning leather; for the manufacture of acetic acid, airplane dope, allyl alcohol, cellulose formate, phenolic resins, and oxalates used in laundry; and in textile, insecticide, refrigeration, and paper industries.[2]
- In South India formic acid is a favourite agent for self-destruction especially among low-income families and laborers.[9]
- In Europe also it is well known, if relatively infrequent, vehicle for suicide.[3, 10]

SALICYLIC ACID

Important therapeutic preparation of this is acetyl salicylic acid (Aspirin) (Refer Chapter 38).

STRONG ALKALIES

Like acids, alkalies also act as corrosive poisons when administered in the concentrated form, but act as irritant poison when diluted.[8] Alkalies are present in a number of household products (e.g. drain cleaners, oven cleaners, dish washer products, some paint strippers, etc.) and are also used in industry.

Mechanism of Action

Alkalies generally contain *hydroxyl groups*, which on dissociation in water produce *hydroxide ions*. Alkali agents create injury to the gastrointestinal tract by the mechanism of liquefaction necrosis whereby saponification of fats and solubilisation of proteins allow deep penetration into tissue. Thus, unlike acids, it produces extensive penetrating damage. This pathogenesis of injury is rapidly progressive, and may extend to weeks after onset.

Alkalies cause more severe corrosive effects on the *esophagus*, than on the stomach as in case with acids. Severe *esophageal damage* can occur, if the pH is lower than 11. However, with deliberate ingestion of large quantity corrosives, effects can be seen anywhere from mouth to the small intestine.[6]

ANHYDROUS AMMONIA

Physical Properties

Ammonia is an irritant gas. It is highly soluble in water. Gaseous ammonia when dissolved in water forms a strong solution of ammonia (Liquor Ammoniae fortis) known as *Spirits of Hartshorn*, which contains 32.5 per cent of ammonia and is a colorless pungent liquid used in agriculture, mining, plastic and explosive industries.[8]

Action

It is absorbed into the respiratory tract and has its pulmonary effects causing pulmonary oedema and direct mucosal injury by alkaline burn.

Signs and Symptoms

Inhalation: Gaseous ammonia causes running nose and increased salivation. It can induce asthma, severe upper respiratory tract irritation, pneumonia, pulmonary oedema, bronchitis, and obstructive lung disease. Death is usually due to bronchopneumonia.

Ingestion: Ammonia ingested can produce intense pain, dysphagia followed by esophageal stenosis.

Eye: Exposures results in watering of the eyes, corneal damage, conjunctivitis and palpebral oedema. Blindness may be the serious consequence in severe cases.

Fatal Dose

Liquid form—10-30 ml.
Gaseous form—0.5 per cent in air.

Treatment

Removal from exposure and rest, including symptomatic treatment; corneal irrigation with water and topical antibiotics should be done.

Postmortem Findings

Following changes are observed:

- Characteristic odour
- Brownish staining of affected skin
- Grayish sloughing of affected mucosa
- Congestion of GI walls (rarely perforations) and respiratory tract and pulmonary oedema.

OTHER ALKALIES

Potassium Hydroxide and Sodium Hydroxide

These are used as drain and oven cleansers.

Fatal Dose

5 grams.

Ammonium Carbonate

It is a translucent, hard crystalline mass with a strong ammoniacal taste and pungent odour.

Potassium and Sodium Carbonate

They are white crystalline powder and are very soluble in water, but not in alcohol. They are used for cleaning and washing purpose.

Fatal Dose—15 grams.

Signs and Symptoms

Taste is acrid and soapy. It gives a heating and burning sensation when ingested. Vomitus and stool may contain altered blood. Later on liver may get damaged.

Tests

- **Carbonate alkalies** produce white precipitate with inorganic acids
- **Hydroxide alkalies** produce yellow precipitate with silver nitrate.

Fatal Period

Usually within 24 hours.

Treatment

- Avoid emesis
- Stomach wash with caution with acetic acid or tartaric acid after mixing with large quantity of water
- Pain relief by morphine
- Raw eggs and milk as demulcents.
- Esophageal strictures should be dilated by means of a bougie. Gastrostomy or esophagostomy, if require.
- Corticosteroid for prevention of strictures at early stages.

Postmortem Appearance

Gastrointestinal tract shows corrosion but not as prominent as in the case of mineral acids. Mucous membrane shows necrosed areas and patchy inflammed chocolate or black areas with blood stained contents. Stenosis is more common at the lower end of oesophagus.

Medicolegal Importance

Mostly accidental. Leakage of ammonia vapours in industrial or godown accidents.

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Chapter 33

Irritant Poisons

Poisons in this category have been classified as '*irritant poisons*', because they mainly produce inflammation on the site of contact, especially in the gastrointestinal tract, respiratory tract and the skin.[1-10] When a poison has a systemic effect and death ensues because of it, then it is classified as a poison affecting that system most, for example, cardiac poison or cerebral poison or a spinal poison.

A case of poisoning with irritant poisons should be differentiated from certain natural diseases of GIT such as cholera, acute gastritis, acute gastroenteritis, perforated gastric ulcer, peritonitis, colics, etc.

Irritant poisons are classified into three groups.

Inorganic Irritants (Figs 33.1A to D)

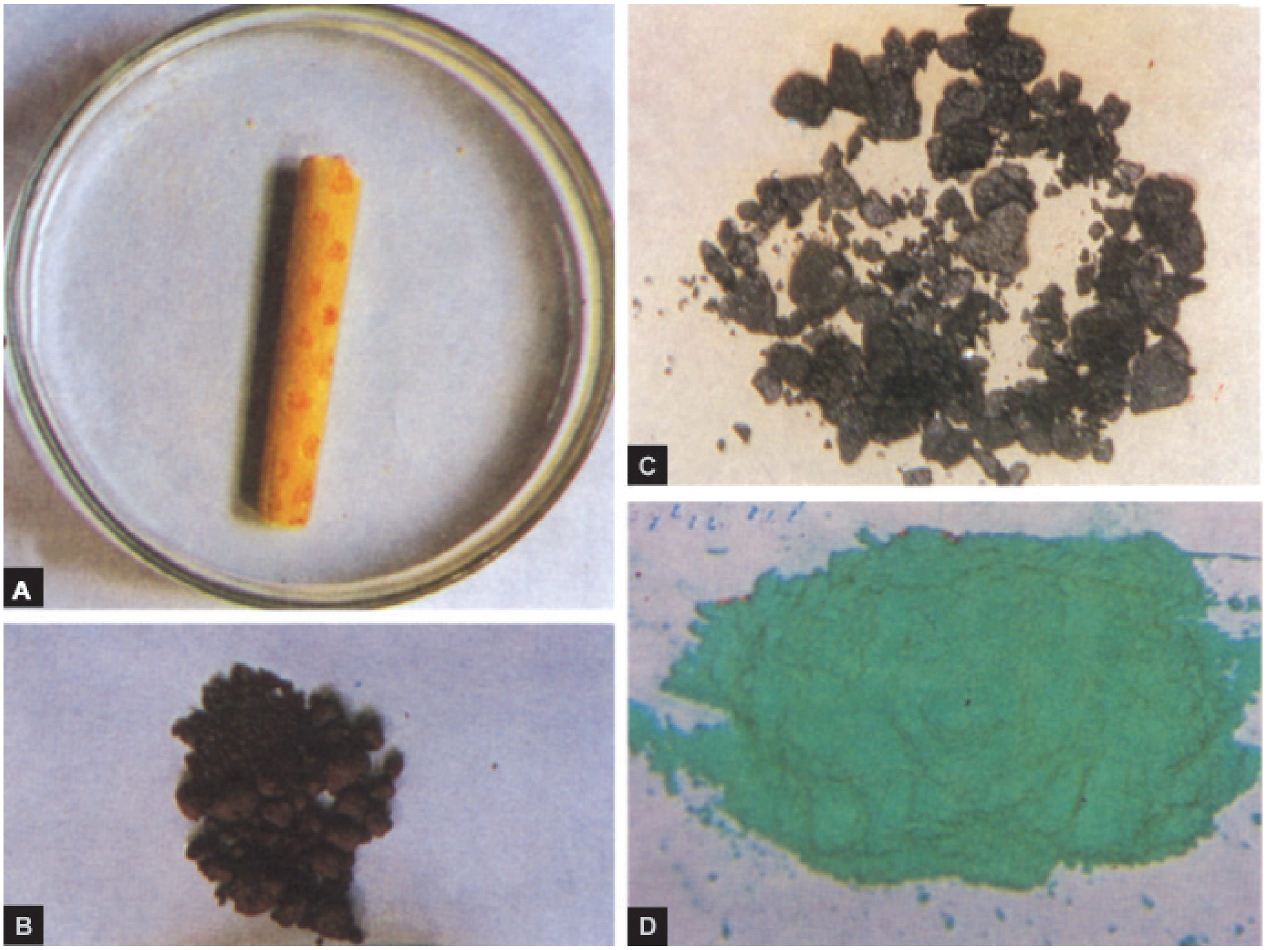
- **Nonmetallic:** Phosphorus, halogens (chlorine, bromine, iodine), formal-dehyde, etc.
- **Metallic:** Arsenic, mercury, lead, copper, iron, etc.

Organic Irritants

- **Vegetable:** Abrus, castor, croton, calatropis, semicarpus, capsicum, ergot, etc.
- **Animal:** Snakes and other poisonous insects.

Mechanical Irritants

- Nails, hair, glass pieces, diamond dust, etc.



Figs 33.1A to D: Inorganic irritants: (A) Yellow-phosphorus, (in Petri dish under water), (B) Red phosphorus, (C) Iodine crystals, (D) Scheel's green (copper arsenite)

GENERAL CHARACTERISTICS OF IRRITANT POISONS

Certain signs and symptoms common to all irritants are discussed first. On consumption, these signs and symptoms will manifest within 30 to 60 minutes and they are:

Common gastrointestinal signs and symptoms (as in case of metallic irritant poisons, phosphorus and most of the vegetable irritant poisons).

- *Burning pain* in mouth, throat, oesophagus, stomach, etc. which radiates all over the abdomen.
- *Intense thirst*, but *dysphagic* due to painful deglutition. So, cannot take water or food which leads to dehydration and starvation.
- *Continuous painful vomiting*. Vomitus shows normal contents initially, but later turns bilious or contain altered blood.
- *Continuous severe diarrhoea and tenesmus*. Stoolsinitially soft, loose but later mixed with mucus and blood.
- *Collapse* due to shock with rapid, feeble pulse, pale anxious face, cold clammy skin, sighing respiration, cramps in leg muscles, etc.
- *Convulsions, loss of consciousness, coma, extreme exhaustion and death* when not treated properly (if survives may develop stricture esophagus later, which can contribute to dysphagia leading to starvation).

Common respiratory signs and symptoms (as in the case of bromine, chlorine and phosphene gas)

- Cough
- Feeling of constriction of chest
- Breathlessness
- Suffocation
- Pulmonary oedema
- Haemoptysis

Common dermal signs and symptoms (as in the case of radioactive substances, insect and snake bites, marking nut, etc.)

- Pain
- Irritation
- Itching
- Redness
- Vesication and blisters

Specific Characteristics of Irritant Poisons

These are discussed in detail with each irritant poison separately.

NON-METALLIC IRRITANTS

Phosphorus

A low birth weight infant of 28 weeks was inadvertently administered 460 mg of phosphorus daily instead of 46 mg as a preventive measure for metabolic bone disease. The infant developed apnoeic attacks with cyanosis, a distended abdomen, and generalized muscle spasm in arms and legs, an extreme hypocalcaemia, and hyperphosphataemia. Calcium supplements lead to the child's recovery.[1]

Phosphorus is a non-metallic, inorganic *hepatotoxic, protoplasmic irritant poison*. It exists in two forms – *white* or *yellow* phosphorus and *red* phosphorus (Figs 33.1A and B). A comparative account of physical characteristics both of these is presented in Table 33.1.

Derivatives of Phosphorus

- Aluminium phosphide
- Zinc phoshide
- Phosphine gas.

Action

Phosphorus acts as a protoplasmic poison due to which normal metabolism is disturbed and cellular oxidation is severely affected. This results in specific changes in liver, bone, kidneys (*acute renal failure*) and lungs.

Liver Changes

The changes observed are called as "*necrobiosis*", which resemble ischemia and prevent cellular metabolism and inhibit glycogen deposition with excess fat deposition, resulting in extensive fatty degeneration and acute *hepatic necrosis*. Thus phosphorus is a *hepatotoxic* substance. (*Some of the other known hepatotoxics are arsenic, alcohol, carbon tetrachloride, chlorpromazine, erythromycin, halothane, iron, isoniazid, naphthalene, nitrofurantoin, paracetamol, paraquat, rifampicin, sulphonamides, and zidovudine*).

Bone Changes

The change observed is called *Phossy jaw.*

Phossy Jaw —is a type of osteomyelitis of jaws observed in chronic cases of phosphorus—poisoning wherein, bone formation under the epiphyseal cartilage, haversian and marrow canals increases, and this can impair the blood circulation to bone, resulting in necrosis and sequestration of bone.[3]

Table 33.1: Comparative account of physical characteristics of white and red phosphorus

Characteristics	*White/Yellow phosphorus*	*Red phosphorus*
Toxicity	Highly toxic	Relatively non-toxic*
Types	Crystalline	Amorphous
Appearance	Translucent, waxy soft sticks	Red, powdery
Odour	Garlicky	Nil
Exposure to light	Turns yellow[2]	Nil
Exposure to air (At 30°C)	Slowly oxidises and ignites** giving dense white fumes of *phosphorus trioxide*[2] (Hence it is always *preserved under water*)	Nil
Solubility	• Insoluble in water • Soluble in alcohol, ether, carbon disulphide, tea, coffee, etc	Alcohol, ether, tea, coffee, etc
Luminescence	+ve	–ve

* *Prolonged contact with red phosphorus can result in dermatitis, loosening of teeth and even bone necrosis.*

** *Always use a forceps to pick it up and not fingers as body heat can ignite it*[2]

Incidence: Common among the workers of phosphorus industries, who suffer *unprotected* chronic exposure to phosphorus fumes for 2-5 years.

Clinical picture: Chief characteristic finding is toothache, originating in *caries* tooth, and failure of the dental socket to heal when extracted, followed by necrosis, and sequestration of jaw,[2,3] often noticed in the lower jaw.[3] *Lesion* is with multiple sinuses open to external surface, producing considerable facial disfigurement.

Pathogenesis: Phosphorus fumes when absorbed into a carious tooth can bring about slow necrosis of the bone, which in 2 to 5 years gets converted into full fledged osteomyelitis with facial disfigurement.

Other findings: Signs of chronic phosphorus poisoning are usually accompanied.

Treatment
- Prevent further absorption of poison
- Treat the lesion surgically

Lung Changes

Phosphene gas (PH_3) reduces oxyhemoglobin in blood and may prove fatal if more than 20 parts of phosphene is present in 100000 parts of air. It can also bring about respiratory inflammation and develop pulmonary symptoms.

Kidneys Changes

Kidney changes constitute—renal damage with *acute renal failure*.

Signs and Symptoms

Signs and symptoms occur in two forms—acute and chronic poisoning.

Acute Poisoning

Massive intake of phosphorus (more than 1 gm) results in *fulminating poisoning*. The chief clinical feature is *peripheral vascular collapse* and *death* in 12 to 48 hours. Acute poisoning presents in *three phases*: *Primary phase*, due to local irritant action on the gastrointestinal tract and *tertiary phase* which is due to action of *absorbed poison*, with a dormant or silent *secondary phase*, of considerable interval in between them.

Primary phase – occurs within 2-6 hours of ingestion and may last up to 3 days. Occasionally, the onset may be immediate. The initial features include garlicky taste, and severe burning sensation in the mouth, throat, retrosternal area and epigastrium, followed by nausea, vomiting and diarrhoea. Breath and vomitus has a garlicky odour. The vomitus and stools will be luminous in dark. There may be haemetemesis. The stools may give rise to faint fumes constituting the *smoky stool syndrome*.

Secondary phase – is a symptom free phase and patient feel well enough and may last so for about 2-6 days or even more after the subsidence of primary phase.

Tertiary phase – is due to systemic effects of the absorbed poison. The original symptoms of primary phase will reappear with increased severity along with manifestations of hepatic damage. There will be *tender hepatomegaly*, *jaundice* followed by an olive green hue, *pruritus*, and *bleeding* from multiple sites and anaemia. Finally hepatic encephalopathy develops, leading to stupor and coma. *Oliguria, haematuria, albuminuria*, and acute renal failure will occur now due to renal damages sustained. Male patients may present with *priapism*, which is common. Death is usually due to hepatic and renal failure.

Chronic Poisoning

Chronic poisoning is usually due to long-term occupational exposure (2-5 years) to the phosphorus fumes in a phosphorus industrial worker, resulting into condition known as *phossy jaw*. Certain specific findings observed in acute and chronic phosphorus poisonings are enumerated in Table 33.2.

Treatment

Acute Poisoning

- Early gastric lavage with 1 in 5000 solution of potassium permanganate or dilute solution of copper sulphate (0.2%).
- Bowel evacuation is helpful.
- Avoid oil, fat, egg, etc orally as phosphorus is soluble in it and would enhance its absorption.
- Intravenous saline is helpful in combating against shock.
- Give dextrose intravenously to protect the liver.
- Vitamin K – 65 mg slow IV drip, can help in hypoprothrombinaemia.
- Vitamin B complex and C as needed.

Table 33.2: Specific findings in phosphorus poisoning

Characteristics	*Acute*	*Chronic*
Garlic like odour and luminescence to vomitus and stools	+ve	+ve
Jaundice* with pruritus	+ve	+ve
Hepatomegaly* with *necrobiosis*	+ve	+ve
Bleeding tendencies from multiple sites and anaemia	–ve	+ve
Bronchitis	–ve	+ve
Bone fragility	+ve	+ve
Phossy jaw (refer above)	–ve	+ve
Burns of hands	–ve	+ve
Dermatitis	–ve	+ve
Conjunctivitis	–ve	+ve
Nasal irritation	–ve	+ve
General prostration	–ve	+ve
Acute renal failure* after scanty, high colored, strongly acidic urine	+ve	+ve
Hypocalcaemia	+ve	–ve

** Occurs in acute cases only when patient survives more than 2-6 days*

- Giving intravenous calcium gluconate (5 to 10 ml of 10% solution) to maintain serum calcium level.
- Symptomatic therapy.

Chronic Poisoning

- Stop further exposure.
- Dental care and regular follow up.

Fatal dose – 60 to 120 mg (1-2 mg/kg body weight)

Fatal period – 4 to 8 days

Toxicity rating – 6.

Autopsy Findings

External

Jaundice, and *haemorrhage* under the skin and from various body natural orifices. Body is emaciated and may emit smell of garlic. Phossy jaw may be observed in chronic cases.

Internal

- Opening the abdomen emits garlicky odor where putrefaction has not taken place.
- Inflammation and erosion of gastrointestinal mucosa (yellowish or greenish-white, softened, thickened, inflamed, eroded and sometimes perforated).
- Liver shows necrobiotic changes (enlarged but sometimes shrunken, doughy, yellow, soft, greasy liver with necrosis and fatty degeneration). In acute yellow atrophy, liver is smaller in size, greasy, leathery and dirty yellow in colour. The capsule is wrinkled and cells are necrosed containing leucin and tyrosin crystals.
- Other viscera like kidneys, heart and muscles- may also show fatty degeneration.
  Preservative used for viscera: Saturated solution of sodium chloride (never use rectified spirit as phosphorus is soluble in it).

Medicolegal Importance

- Accidental poisoning is usual (especially industrial poisoning is common). Children get inadvertently poisoned through fire crackers, rat pastes, etc. Pregnant women often get poisoned accidentally with phosphorus as it is often being used as quack remedy for inducing abortion.
- Homicidal use is rare–due to smell and luminescence (in dark). However, literature review provides several instances in the past by mixing phosphorus in the food items such as soup, jam, etc.
- Suicidal poisoning, which was common in the west in the past prior to the invention of Lucifer match sticks. Popular method in the past was to dissolve the match stick heads in water, brandy, etc and then to consume the potion adding sugar to it.[4]
- Used in industry in manufacture of *fertilizers*, *rodenticides*, and *pesticides* (as insecticides). Also used in manufacture of *fire works*, *gun powder*, *incendiary bombs*, tracer bullets, smoke screen, rescue flares, etc.
- *Lucifer matchstick* — is a type of matchstick, which was made up of yellow phosphorus till the early part of this century (till 1931), which resulted into on occupational hazard among the workers employed in matchstick factories. Apart from this, these matchsticks were being ignited by brushing against any rough surface often resulting in several fire accidents. By dissolving the tip of the same in coffee, alcohol, etc. these matchsticks were used for *homicidal* purposes. As a result "safety match—sticks" were introduced since 1931 and these did not contain yellow phosphorus. The matchstick now has *potas-sium chlorate* and *antimony sulphide*, while matchbox side had igniting surface made up of *red phosphorus* and powdered glass.
- *Workmen's Compensation Act* (regular teeth check-up of workers in phosphorus industry).

CHLORINE

Chlorine is a halogen, which is an inorganic non-metallic irritant poison.

Properties

Chlorine is a yellowish green *gas* with irritating pungent odor.

Action

Chlorine acts as a direct irritant of the mucous membrane of respiratory tract by locally forming hydrochloric acid as it comes in contact with moisture.

Signs and Symptoms

Main symptoms after inhalation are choking, suffocation and a feeling of tightness in the chest with laryngeal spasm. Headache, nausea, sore throat, lacrimation, rhinorrhoea and cough also occur. Breathlessness is due the collection of secretions inside the respiratory passage. Death occurs due to laryngeal or pulmonary oedema.

Fatal dose >400 ppm for few minutes (inhalation) or 1 part of chlorine in 1000 parts of air exposed for 5 minutes.

Fatal period 24 hours of inhalation of pure chlorine gas.

Treatment

- Inhalation of humidified oxygen
- Give bronchodilators—aminophylline/salbutamol
- Wash the affected eye with saline water
- Symptomatic line of treatment.

Postmortem Appearance

- Characteristic odour.
- Massive pulmonary oedema.
- Respiratory epithelium denudation.

Medicolegal Importance

- It is mainly used as a disinfectant and bleaching agent, and in water purification and sewage treatment.
- Most of the cases are accidental poisoning, arising out of occupational exposure
- Poisoning can occur in swimming pool with excess of chlorine added as disinfectant
- Accidental domestic poisoning is also often reported.

BROMINE

Bromine is a reddish brown liquid, volatilizing to red fumes at room temperature and emitting an unpleasant odor.

Action

Bromides are more often in use as medicine, acts as a sedative and cough elixir.

Signs and Symptoms

If taken in liquid form, bromine acts as a corrosive poison. Intense burning pain throughout the gastrointestinal tract, dysphagia, vomiting, eructation of offensive vapors and purging are due to the corrosive action of bromine liquid on the gastrointestinal tract.

If inhaled in gaseous form, bromine causes violent catarrhal inflammation of the respiratory tract. There is cough, feeling of constriction of the chest, pulmonary oedema and haemoptysis, oedema of the glottis and larynx and death from suffocation.

Bromide poisoning is usually a chronic one and is also known as "*Bromism*" and occurs due to repeated administration of bromides of ammonium, sodium and potassium as sedatives in medical doses, over a prolonged period. Clinically, bromism manifests with:

- Skin rashes in the form of red papules *(Bromine rash)*, similar to acne vulgaris, which may transform into a pustular lesion/ ulcerate at the hair roots *(Bromoderma),* on the face, neck and upper part of chest.
- There may be blunting of memory, muscular weakness, and incoordination.
- May suffer from delusion, hallucinations and personality changes in severe cases.

Fatal Dose and Period

Uncertain. The maximum permissible level of vapor in air is 0.1 ppm.

Diagnosis

Diagnosis is by serum bromide level (100-150 mg/100 ml).

Treatment

- Withdrawal of bromide containing product
- Symptomatic line of treatment stomach wash, oral starch, diuretics, caffeine to: combat respiratory failure and haemodialysis.

Postmortem Findings

In cases of oral ingestion of bromine in liquid form, there is inflammation of oesophagus and stomach, which presents as leathery parchment like appearance. Occasionally perforation may be found.

Pulmonary oedema and oedema of glottis are common, where bromine is inhaled.

Medicolegal Importance

- Bromides report poisoning/death rarely.
- More than bromine, bromides are involved in poisoning episodes. These are present in several drugs routinely prescribed by the physicians, which hence might lead to poisoning when taken for long time or as with overdose. Drugs like bromazepam, bromocriptine, brompheniramine maleate, carbromal, dextromethorphan are some of the examples.
- Germans in First World War used bromine in lachrymating and asphyxiating shells.

IODINE

Iodine is a type of halogen, an inorganic nonmetallic irritant poison.

Properties

Iodine is a volatile crystalline substance with purple glittering colour (see Fig. 33.1C), a characteristic odour and an acrid taste. It gives violet fumes/vapors at room temperature.

Action

It acts as an antiseptic. It is a powerful irritant and vescicant.

Signs and Symptoms

Two types are observed—acute and chronic poisoning.

Acute Poisoning

- Burning pain from mouth to epigastrium, intense thirst, excessive salivation, vomiting, purging, giddiness, cramps, convulsions and fainting
- Lips and mouth are stained brownish
- Vomitus and stools are dark yellow/ bluish in colour and show the presence of blood and emits iodine odour
- Urine is suppressed, reddish-brown in colour and shows the presence of albumin
- Pulse—low and weak
- Skin—cold and clammy, the patient passes into a state of uremia and collapse, but consciousness is retained till death.

Chronic Poisoning

Also known as "*Iodism*", where the patient takes large doses of potassium iodide, continuously as medication. It presents with erythema, urticaria, acne, inflammation of all mucous membranes, parotitis, lymphadenopathy, anorexia, insomnia, etc.

Fatal dose 2-4 gm of iodine or 30-60 ml (1-2 ounces) of tincture iodine.[5]

Fatal period Average 24 hours.

Toxicity rating 3-5.

Treatment

- Evacuation of stomach: Administer starch/ flour solution—30 gm/litre of water, milk is helpful
- Sodium thiosulphate solution (1-5%) orally
- Symptomatic line of treatment. Treat dehydration and shock with intravenous fluids.
- Liberal intake of sodium chloride or bicarbonate of sodium, in *Iodism* cases.

Postmortem Findings

- Brownish stains of skin and mucosa
- Characteristic iodine odour
- Congestion of all the viscera. Stomach may show blue content if starchy food is present.
- Heart and liver may show fatty degeneration and kidneys glomeruler/tubular necrosis.

Medicolegal Importance

- Acute poisoning is mostly accidental/ suicidal but occurs rarely.
- Chronic poisoning is usually an occupational hazard.
- It is commonly used in photography.
- *Tincture iodine, Lugol's iodine* and *Povidone iodine* are some of iodine preparations useful in medicine as an antiseptic.

FORMALDEHYDE (FORMALIN, METHYL ALDEHYDE, METHYLENE OXIDE[6])

Formaldehyde is a nonmetallic, irritant poison.

Properties

Formaldehyde is a colourless gas with pungent odour. However, commercially it is available as formalin, which is a 40 per cent aqueous solution of formaldehyde gas.

Action

Formalin is a disinfectant, antiseptic, deodorant, tissue fixative and embalming agent.[6] It has an irritant action also, and can act by all routes of absorption.

Signs and Symptoms

Two types are observed—acute and chronic poisoning.

- *Acute Poisoning*
  - *By inhalation* – Inhalation of vapours can bring about irritation of respiratory tract, resulting in headache, rhinitis, dyspnoea, lacrimation, cough, etc.[7]
  - *By oral ingestion* – Oral ingestion can result in corrosion of GIT, with pain abdomen, nausea, vomiting and diarrhoea. Pupils will be constricted and the face is flushed. It can cause severe acidosis which results from rapid conversion of formaldehyde to formic acid. Coma, hypotension, renal failure, etc are usual complications in severe ingestion cases.[6]
- *Chronic Poisoning* – has been shown to be carcinogenic in animal experiments, but its relationship to occupational cancer is uncertain.[8] Repeated exposure to formaldehyde may cause some persons to become sensitized to it in few days to months or even after first exposure and this can result in asthmatic reaction at levels which is too low to cause any symptoms in normal people.[6]

Fatal dose 30 to 90 ml

Fatal period 24 to 48 hours

Toxicity rating 3.

Treatment

- Dilution with milk or water in alert patients as a first aid measure may reduce its local effects.
- Gastric lavage with 0.1 per cent solution of ammonia, as it reacts with formaldehyde to form harmless methanamide.
- Haemodialysis is life saving in severe cases
- Symptomatic line of treatment.

Postmortem Appearances

- Typical smell on opening the body
- Stomach mucosa may be red, inflamed and eroded; extravasation of blood may make it hard and tough like leather
- Intestine and lungs are congested
- Liver may show fatty degeneration
- Kidneys are inflamed.

Medicolegal Importance

- Fatal cases are reported occasionally due to accidental or suicidal ingestion.
- Accidental poisoning is usually chronic poisoning which commonly occurs as an occupational hazard.
- Cigarettes or marijuana are dipped in formaldehyde (“amp”) before smoking for its alleged hallucinogenic effects.[9]

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METALLIC IRRITANT POISONS

ARSENIC *(Sankhyal, Somalkar)*

Arsenic is a heavy metallic inorganic irritant poison. Metallic arsenic is not poisonous as it is insoluble in water and cannot be absorbed from the gastrointestinal tract. However arsenious oxide or arsenic trioxide (*sankhyal or somalkar*) is poisonous. Two organic arsenic non toxic variants, mostly present in food regularly consumed by humans are *arsenobetaine* and *arsenocholine*.[1] They are found in shell fish, cod, and haddock. Tables 33.3 and 30.4 provide the list of inorganic and organic arsenic compounds with their physical properties and uses.

Sources

Soil, well water, shellfish and arsenic compounds.

Absorption

Absorption is possible through all routes.

Action

Arsenic compounds act by inactivating the sulhydril enzymes, which in turn interfere with the cellular metabolism, in the liver, lungs, intestinal wall, and spleen. Arsenic can replace phosphorus in the bones where it may remain for years. It also gets deposited in the hairs.[1] Epidemiologic studies of arsenic in *drinking water* suggest that arsenic can cause skin, lung, liver, kidney and bladder cancer 1 in 1000 cases.[2]

Fatal dose 100 to 200 mg of arsenious oxide.

Fatal period 2 to 3 days.

Toxicity rating 5 for all arsenic salts, except arsenic trioxide, which has a toxicity rating of 6.

Signs and Symptoms

Arsenic poisoning clinically manifests in three forms:

- Acute fulminating type
- Subacute type (gastroenteritis type)
- Chronic type.

Acute Fulminating Type

Here symptoms appear within half an hour especially when heavy dose of arsenic is taken (3-5 grams). Acute fulminating type occurs due to inhibition of sulphydryl enzyme system which

Table 33.3: Inorganic arsenic compounds and their physical properties and uses[1]

Chemical names	*Synonyms*	*Physical properties*	*Industrial uses or biologic occurrences*
Arsine	Arsenic trihydride, hydrogen arsenide, arseiuretted hydrogen	Colorless gas slightly water soluble, and has garlicky odour	Lead plating, soldering, galvanising, in solid state electronic components
Arsenic (elemental)	Grey arsenic, metallic arsenic	Grey shiny, brittle–looking rhombohedra substance	In alloys to increase hardness and heat resistance
Arsenic trichloride	Butter of arsenic	Yellowish oily liquid, water soluble	Pottery industry and manufacture of chlorine containing arsenicals
Arsenic trioxide	*Arsenic oxide*, white arsenic, arsenic sesquioxide, arsenious anhydride	White amorphous or crystalline powder, slowly water soluble and forms *arsenious acid*, heated sublimes	Manufacture of *glass* and *insecticides* and *rodenticides*. Some other proprietary items containing arsenious oxide are: • *weed killer*, • *fly paper*, and *fly powder* (with metallic arsenic), • *Sheep dip* (with sodium carbonate, soap and sulphur), • *Rough on rat* with barium chloride.
Sodium arsenite	Arsenious acid sodium, meta arsenite	White or greyish hygroscopic powder, highly water soluble	Veterinary uses, insecticide: *fly water*, wood preservative.
Arsenic pentoxide	*Arsenic oxide*, arsenic acid, arsenic anhydride	White amorphous powder, freely water soluble forming arsenic acid	Manufacture of coloured glass, insecticide, wood preservative.
Lead arsenite	Arsenic acid, acid lead arsenate	Heavy white powder, on heating emits toxic fumes	Insecticide with other salts calcium arsenate, sodium arsenate, potassium arsenate
Arsenic sulphide	*Red realgar* (disulphide) *Yellow orpiment* (trisulphide)	Red powder, yellow powder	Depilatory, colour pigment
Copper arsenite	*Scheel's green* (see Fig. 33.1D)	Greenish powder	Coloring agent for toys, *wall paper,* etc.
Copper acetoarsenite	*Paris green* or *Emerald green*	Greenish powder	Insecticide

Modified from Table 67-11, pp 1539, by Ellenhorn MJ, Ellenhorn's Medical Toxicology, Diagnosis and Treatment of Human Poisoning, 2nd edn. Williams and Wilkins, Baltimore, USA, 1997

Table 33.4: Organic arsenic compounds and their uses

Chemical name	*Industrial uses or biologic occurrences*
Arsenobetaine	These *organic arsenicals* are non-toxic variants, are mostly present in food regularly consumed by humans, namely: *shell fish, cod*, and *haddock*.
Arsenocholine Carbarsone Tryparsamide Glycobiarsol Melarsoprol	These *organic arsenicals* were used in *therapeutics* in the *past*.

is necessary for cellular metabolism and also due to its potent capillary poisoning action. It causes marked dilatation of capillaries and myocardial failure resulting in fall of blood pressure, shock and death instantaneously.

Subacute Type (Gastroenteritis Type)

This type of poisoning occurs when small doses of arsenic are given at repeated intervals. It resembles case of cholera or food poisoning. The symptoms are first dyspepsia, cough and tingling in the throat, followed by vomiting, purging with pain abdomen and tenesmus. Stool is at first rice water type, but later becomes bloody. However, the difference between arsenic poison and cholera may be enumerated as follows:

- In arsenical poisoning, vomiting precedes purging (stools are rice water initially and later turn blood stained), there is pain in throat, voice remains unaffected, conjunctiva gets inflamed and vomitus contains mucous, bile and streaks of blood. Arsenic can be detected on chemical examination.
- In cholera, purging precedes vomiting (stools are like rice water throughout and passed in continuous involuntary jet), there is no pain in the throat, voice becomes rough and whistling, conjunctiva is normal and vomitus is watery. Cholera vibrio can be detected on microscopic examination.

Chronic Type

Occurs in persons engaged in smeltering or refining ore or long-term exposure to arsenic compounds. Chronic poisoning with arsenic presents with a sequence of five different set of manifestations.

- *Gastrointestinal* The victim presents with gradual weight loss, malnutrition, fatigue, loss of appetite, cirrhosis of liver, nausea, vomiting, etc.
- *Catarrhal changes* The victim presents with running nose, headache, conjunctivitis, bronchial catarrh, etc.
- *Raindrop pigmentation* It is known to produce *milk and roses complexion* initially, followed by *patchy brown pigmentation* of the skin (especially *face*), which resembles the *raindrops*. It might also show *hyperkeratosis* of the skin of the palm and soles, which is prone to change into *basal cell carcinoma* at a later stage. The scalp may also show *alopecia* (baldness).
- *Meese's lines* The victim's nail manifests with whitish lines 1 to 2 mm breadth across the nail of the finger and toes representing the deposition of the poison as a result of high sulphhydril content of the keratin.
- *Arsenical neuritis* The victim presents with polyneuritis, optic neuritis, anesthesias, paresthesias, atrophy of extensors resulting in wrist and foot drop, etc.

Table 33.5 enumerates all the above findings briefly.

Diagnosis

Urinary level of > 100 mg/ 24 hours suggestive of arsenic toxicity.

Blood level and hair levels are not reliable.[1]

Treatment

- Haemodialysis is the line of choice in massive arsenic poisoning. Chelation therapy with BAL (British Ante Lewisite or dimercaprol) is advised here to control the deleterious effects of arsenic redistribution.[5]
- Perform gastric lavage with warm water or freshly prepared hydrated ferric oxide solution.
- Give butter and other greasy substances which acts as demulcents and prevents further absorption of the poison
- Specific antidotes are BAL– chelation therapy in the dose of 400-800 mg on the first day followed by 200-400 mg on the next two days and then tapering the dose slowly). DMSA (DiMercaptosuccinic acid) or DMPS (DiMercapto propane sulphonate) penicillamine, calcium disodium versnate, etc may also be useful.
- Symptomatic therapy.
- Injection: Vitamin B_1 helps in peripheral neuritis.

Postmortem Findings

External Body will be dehydrated, skin is pigmented or rarely jaundiced, hands and feet cyanosed, with Mee's lines on the nails. Rigor mortis is observed to be unusually longer.

Table 33.5: Clinicopathological findings in acute and chronic arsenic poisoning

System	*Acute*	*Chronic*
Skin	Delayed hair loss (*alopecia*)	Melanosis, Bowen's disease facial oedema, hyperkeratosis, cutaneous cancer, hyperpigmentation
Nails	*Mees's lines* in 2-3 weeks postingestion	
Neurologic	Hyperpyrexia, convulsions, tremors, coma	Encephalopathy, polyneuropathy, tremors, axonal degeneration
GI tract	Abdominal pain, dysphagia, vomiting, *bloody* or *rice water* diarrhea, mucosal erosions	Nausea, vomiting, diarrhoea, anorexia, weight loss
Liver	Fatty infiltration	Hepatomegaly, jaundice, cirrhosis
Kidney	Tubular and glomerular changes – oligurea and uremia	Nephritic finding
Haemetologic	— thrombocytopenia, impaired folate metabolism,	Bone marrow hypoplasia, anaemia, leukopenia, *basophilic stippling* and karyorrhexis
Cardiovascular	Heart will show S-T wave abnormalities, prolonged QT interval, ventricular fibrillations, atypical ventricular tachycardia with *humic acid* in Southwest coast of Taiwan	*Blackfoot disease*[3] due to platelet activation and hypercoagulability of blood in peripheral arteries is observed in endemic areas of aresenicism,
Reproductive system	Maternal ingestion of inorganic Arsenic crosses the placental barrier and infant death shortly after birth[4]	—

Internal

Stomach – *Velvety red* or brownish, patchy areas with small ulceration seen on the stomach mucosa. Gastric contents emit garlicky odour.

Heart – Shows *subendocardial haemorrhage*.

Other viscera – may show fatty degeneration (liver, kidney and heart). Brain may show acute encephalitis with haemorrhagic spots.

Chemical Test

Reinsch's test: Put 1-2 strips of bright copper foils into suspected solution previously acidulated with HCl and then boil for 5-10 minutes. The copper foil becomes coated with steel grey or black deposits of arsenic if present. This foil when washed in water, alcohol and ether and then heated will show white deposits of arsenious oxide (octahedral crystal on microscopy).

Other advanced tests for detecting arsenic are Marsh's test and Gutzeit's test.

Medicolegal Importance

- In India, today most common source of accidental poisoning with arsenic is by consumption of well water. There is sufficient evidence that a sizeable proportion of several Asian countries are exposed to arsenic tainted water, especially the tube well water.
- According to Modi, arsenic was considered as an ideal homicidal poison in the past in India and west as it was cheap, easy to obtain, could be easily mixed and given with food without changing the smell and taste and the symptoms of poisoning would be similar to cholera or gastroenteritis.[6] However, due to legal restriction of sale of arsenic and availability of sophisticated methods of detecting minute quantities of arsenic in blood and tissues from the dead bodies of victims of arsenic poisoning (e.g. *Marsh's test*, which can detect even the traces of arsenic -1/1000 mg dilution), criminal use of arsenic has become extinct now.
- Accidental poisoning instances do occur occasionally with those who consume arsenic for purposes like its *aphrodisiac effects* (quick remedy for impotence), eating arsenic *arsenophagy*, for better respiratory stamina among the mountaineers, etc.
- Arsenious acid, copper arsenite (Scheel's green) (see Fig. 33.1D) copper acetoarsenite (Paris green), liquor arsenicals (Fowler's solution which is 1 per cent arsenious oxide, were used as medicine for treating fever in the past). Potassium arsenite and sodium arsenite is used to make flypapers, rodenticides, fungicides and sheep dips. Arsenic sulphides used for making yellow pigment for art.
- ***Case Examples:***
  - — Among the interesting cases of homicide by arsenic of the past is that of *Napoleon Bonaparte*, which was finally convinced by the scientific evidences recently.[1]
  - — In the Annual Report for the year 1941, the Chemical Examiner, Bengal, mentions case where he received from Magistrate of Sealdah, a cigarette box containing six 'Passing Show' cigarettes which was recovered from three old offenders loitering on the platform of the Shamnagar Railway Station. Four of the cigarettes were found to contain arsenic (about 240 mg in each) mixed with tobacco. Offenders planned to rob railway passengers by their novel method.[7]
  - — A case came under Modi's observation in 1930s, in which a man had committed suicide by taking orpiment. The stomach contained mucous in which were entangled particles of yellow sulphide of arsenic.[8]
  - — A woman died after swallowing a lump of white substance resembling sugar. The stomach and its contents revealed arsenic equivalent to about 0.57 gm of white arsenic.[9]
  - — Another woman, who took some white paste with betel leaves, mistaken for *chunam (lime)*, died after three hours. Arsenic, equivalent to about 0.5 gm of white arsenic was detected in her stomach content.[10]

MERCURY *(Quick Silver, Liquid Metal, Para, Padarasa)*

Mercury is a liquid metal and is a metallic inorganic irritant poison. It is available in inorganic, organic and metallic forms. Metallic mercury is a heavy, silvery liquid and is non poisonous. But it volatilises at room temperature and inhalation of vapours is toxic. Potential source of elemental mercury is at home, which includes mercury switches, mercury containing devices such as thermometers, thermostats, and barometers. Family members may also bring it home from laboratories, dental offices, and industrial sources, etc.[11]

Absorption

Absorption is possible through all routes.

Action

Pure metallic form is nontoxic. However, the mercurial compounds can act by inactivating sulphhydryl enzymes, which in turn interfere with cellular metabolism.

Toxic Compounds

Though the metallic form of mercury is non-toxic, its vapors as well as finely divided small particles of mercury can be toxic. There are several inorganic mercurial compounds, which are toxic (Table 33.6). Some of the organic mercurial preparations are presented in Table 33.7.

Fatal dose 100-400 mg of mercuric chloride.

Fatal period Few hours 1 to 2 weeks.

Toxicity rating 5 or 6 for most of the salts.

Signs and Symptoms

Mercurial poisoning manifests clinically in two forms. However, patterns and severity are dependent on the form of mercury and the route of exposure (Table 33.8).

Acute Poisoning

Symptoms commence about half an hour after swallowing mercuric chloride—

- Initially there will be acrid, metallic taste, feeling of constriction of throat, hoarse voice and difficulty in breathing. Tongue and mouth gets corroded followed by burning sensation extending down the abdomen. Vomiting of greyish slimy material with bloody streaks is then followed by blood stained diarrhoea and tenesmus. Oral consumption can lead to glossitis, ulcerative gingivitis, and necrosis of the jaw.
- Nephrotoxicity leading to albuminuria, cylindruria, uraemia, acidosis, etc. Urine will be scanty and contains blood and albumin. Toxic mercury compounds are considered as *nephrotoxic poisons* and cause renal tubular and glomerular necrosis.

Table 33.6: Inorganic mercurial compounds: synonyms, physical properties and uses

Chemical names	*Synonyms*	*Physical properties*	*Industrial uses or biologic occurrence*
Mercuric chloride	*Corrosive sublimate*, perchloride of mercury	Heavy colourless masses of prismatic crystals or white crystalline powder. Styptic, nauseous, metallic taste. Soluble in water. Alcohol, ether, glycerin, alkaline chloride solutions	Antiseptic property has made it useful in medicine and *taxidermy*
Mercuric oxide	*Sipichand*	*Brick red* crystalline *powder*, which forms *yellow powder* with caustic soda or potash. Insoluble in water	Used in BPC preparation *unguentum hydrargi oxidi rubric* red ointment. The yellow one is called *hydrargyri oxidum flavum*
Mercuric sulphide	*Cinnabar, hingul, ras sindhoor, cheenasindoor, singarf, vermilion* (see Fig. 33.2)	Occurs as chief ore of mercury. Is artificially prepared as a red, crystalline powder, and then known as *vermillion*. Is regarded non-poisonous but vapours poisonous	Used to prepare vermillion. Used to colour vulcanized rubber for artificial denture, resulted in chronic poisoning. Known for pruritus and nodular swellings when used for tattooing followed by exposure to sunlight[13]
Mercurous chloride*	*Raskapoor*, *calomel lotion*, subchloride of mercury.	It is a heavy amorphous white tasteless powder, insoluble in water, alcohol, ether. On exposure to light turns into toxic mercuric chloride	Used in laxative preparations, can result in chronic poisoning as laxative abuse.[14] Less toxic compared other mercuric salts above
Diethyl mercury and dimethyl mercury	Mercuric methide	Highly poisonous liquid, Inhaling its noxious vapours caused *insanity* and death	Used as fungicides
Mercury fulminate	—	Produce severe itching and ulcers on nailbeds and fingertips	Used in percussion cap and detonator industries

Table 33.7: Organic mercurial preparations and their uses

Chemical names	*Uses*
Neptal	Used as diuretics and has low toxicity when used in therapeutic doses
Thiomerin sodium (mercaptomerin sodium)	
Mecurophyllin	
Metaphen	Used as antiseptics for sterilizing skin and instruments
Merthiolate (thiomersalate)	

Table 33.8: Forms of mercury, absorption and toxicity

Form of mercury	***Absorption***		***Toxicity***		
	Inhalation	*Ingestion*	*Gastrointestinal*	*Renal*	*Neurological*
Elemental mercury (Liquid)	Not applicable	No	No	No	No
Elemental mercury (Vapor)	+++	Not applicable	No	Rare	Yes
Inorganic mercury	No	++	Yes	Yes	No
Organic mercury	++	+++	No	Rare	Yes

Courtesy: Alison LJ, Paul ID. Churchill's Pocket book of Toxicology[12]

- Inhalation of fumes of mercury can lead to metallic taste, salivation, gingivitis, and loosening of teeth with foetid teeth.
- Strong concentration may also cause ataxia, paresis, delirium, etc.
- Locally, mercury salts have corrosive action.
- Blood peripheral smear can show *leucocytosis*, while *leucopenia* occurs with organic mercurial poisoning.

Chronic Poisoning (Hydrargyrism/Mercurialism)

The synonym *Hydrargyrism* is after the Latin word *Hydragyrus*, which means mercury. Chronic poisoning occurs where the victim is exposed to mercury fumes in factories or excessive dose of mercurial compound are used for a prolonged period. Symptoms begin to appear at blood levels of 100 nanogram per cent of mercury. The victim manifests with:

- Excessive salivation (*ptyalism*/*sialorrhoea*), with swollen and painful salivary glands, metallic taste in the mouth, glossitis, ulcerative gingivitis, and necrosis of the jaw.
- A blue line on the gums called *Burtonian line* is a common clinical finding of chronic poisoning.
- Nausea, colicky pain, vomiting and diarrhoea are other gastrointestinal manifestations.
- Evidence of nephritis and uraemia may be seen.

- *Mercuria lentis* develops which is due to brownish deposit of mercury through the cornea on the anterior lens capsule and can be observed as a brown reflex on slit-lamp examination.
- *Mercurial tremors* can be detected in early stages with change in handwriting of the person as it first affects the muscles of the finger, followed by muscles of the tongue causing stammering and slurring speech, and finally affecting the muscles of the face, arms and legs. This was referred to as *Hatter's shake* in the past among the workers of *Hat industry*, where in mercury was used extensively in giving the peculiar kinking shape to the felt hats. *Other drugs and poisons which produce tremos are—Alcohol, phenothiazines, caffeine and theophylline, tricyclic antidepressants, carbon monoxide, and phosphorus.*
- *Mercurial erethism* comprises personality change resulting in an abnormally high degree of irritability or sensitivity or excitability, shyness, amnesia, insomnia, delusions, hallucination, leading to insanity.

Treatment

- Perform gastric lavage with 5 per cent solution of sodium formaldehyde sulfoxylate. About 100 ml of the same may be left in the stomach after the lavage.
- Administer demulcents like egg albumin.
- Administration of medicinal charcoal with magnesium sulphate is of great use.
- Specific antidotes are BAL (dimercaprol at a dose of 3-4 mg/kg body weight. every four hourly), or penicillamine at a dose of 250 mg to 2 gm orally, or sodium formaldehyde sulphoxylate as chemical antidote, etc.
- Symptomatic therapy.

Postmortem Changes

External – Nothing specific.

Internal – Mucous membrane of lips, mouth and pharynx show diffuse greyish white escharotic appearance. Stomach and intestine show severe irritation and corrosion with ulceration and softening. Intestines especially the caecum, colon and rectum are found to be inflamed, ulcerated or even gangrenous where the patient survives for some days. Kidney shows the findings of toxic nephritis. Liver shows fatty degeneration. Heart shows subendocardial haemorrhages and fatty degeneration.

Medicolegal Importance

- Mercury is an industrial poison. It is used in industries, connected with manufacture of thermometers, barometers, mercury vapour lamps, firecrackers, explosives, paints, etc. *Hatter's shake* or *glass blower's shake* which are moderately course tremors interspersed by jerky movements found in the workers of *glass blowing* and *hat industry* are some examples for chronic poisoning of industrial origin. Chronic mercury poisoning manifestations are prevalent among the gold miners and gold refining industry workers who are exposed to mercury used in the process.[15,16]
- The medical use of mercury as diuretics, vaginal douching purposes, as a dental restorative material in dental clinical,[23,24] etc. can also lead to accidental poisoning (by overdose)
- Incidences of suicide and homicide with mercury is though rare, accidental poisoning is quite frequent especially among the children, e.g. *Pharaoh's serpent (Diwali poison)*—is a black coloured tablet shaped fire cracker, which contains mercuric thiocyanate and on ignition yields a long black coloured tubular ash.

LEAD (SHISHA)

Lead is a metallic inorganic irritant poison. According to Health And Human Services Department USA, lead poisoning is the *most important environmental problem for the young children.*[17] Blood levels once thought to be safe have been shown to be associated with IQ deficits, behaviour disorders, slowed growth, and impaired hearing.[1] Studies in population blood-lead concentrations have shown a fall by up to 80 per cent in the last twenty years, cases of lead poisoning continue to occur.[12] Poisoning is more common from chronic occupational exposure among lead smelters, battery manufacturers, painters, decorators, etc. Chronic exposure may also occur at home from paint, pottery and contaminated drinking water by lead pipes used for city water supply.

Absorption

Absorption is possible through all routes.

Action

Pure metallic forms are nontoxic is a steel-grey metal. However, lead compounds can act by producing spasms of the capillaries and arterioles or by fixation of the poison in the tissues such as brain, bones, etc. It can also combine with *sulphydryl enzymes* and interfere with its action. Lead can decrease synthesis of heme leading to anaemia and can bring about hemolysis as well as release immature RBCs into circulation (reticulocytosis and basophilic stippling of RBCs). Lead can destroy nerve cells, myelin sheaths in CNS and also produce cerebral edema. It also exerts toxic effects on kidneys (nephritis) and reproductive system (infertility).

Toxic Compounds

The common toxic compounds, their synonyms and physical properties and uses are enumerated in Table 33.9.

- ***Fatal dose*** depends on toxic compound (20 gm of lead acetate).
- ***Fatal period*** 1 to 2 days.
- ***Toxicity rating*** 3 or 4 for most of lead salts.

Signs and Symptoms

Lead poisoning clinically manifests in two forms: acute and chronic forms.

Acute Poisoning

This usually occurs with high dosage of lead acetate, starts with burning and dryness in the throat, salivation and intense thirst. Vomiting occurs within 24 hours with colicky pain and tender abdomen. Constipation is a common feature. Urine is scanty. Finally there may be peripheral circulatory collapse, headache, insomnia, paresthesia, depression, convulsions, exhaustion and coma leading to death.

Subacute Poisoning

This type of poisoning occurs from repeated small doses of lead acetate. Blue line on the gums is seen with gastrointestinal symptoms. Urine is scanty and in deep red colour. In the later stages, nervous symptoms become prominent with numbness, cramps and flaccid paralysis of lower limbs. Death is rare but may be followed by convulsions and coma.

Table 33.9: Common toxic lead compounds, their synonyms, physical properties and uses

Chemical names	*Synonyms*	*Physical properties*	*Industrial uses or biologic occurrences*
Lead acetate	Sugar of lead, salt of saturn/ lotio plumbi	It occurs in white masses of acicular crystals, slightly efflorescent, a sweet astringent taste, and dissolves in water. Resembles loaf of sugar	It was used in the past in sweeten wine, and in medicine as an astringent and local analgesic for sprain
Lead carbonate	White lead, *safeda*	It is a white crystalline powder, insoluble in water, extensively used as a pigment in oil painting	Paint manufacturing. Children who suck and bite toys with white lead suffer from poisoning
Lead chromates	—	Is bright yellow, insoluble in water, known as chrome yellow used as pigment	Fatal cases reported with this when used to colour sweet meat with this[6]
Lead oleate	Diachylon		Plaster, abortifacient
Lead oxide	Litharge	—	Glazing of pottery and enamel ware
Lead sulphide (Fig. 33.3A)	*Surma*, galena*	Exists in the form of cubic crystals, but is sold as a powder form called surma in place of antimony sulphide, which was actually used as collyrium for eyes	*Collyrium* of the eyes among Muslims
Lead tetroxide (see Fig. 33.3B)	Red lead, *minium, sindhur, mitia sindhur* or *vermillion***	Is a scarlet, crystalline powder which varies in colour according to its mode of preparation and employed as a pigment. Insoluble in water	As sindhur/vermillion is used for coloring the front portion of parting of scalp hairs among the Hindu women, as a sign of being married
Tetraethyl lead	Lead tetraethyl *'Anti-knock'* for petrol	Readily absorbing, highly toxic, heavy, oily liquid, volatile at rook temperature. Soluble in alcohol, acetone and miscible with fats and oils	Added to petrol to prevent '*knocking*' of the engine, and called ethyl petrol or ethyl gasoline, is use as fuel in motor cars. Several accidental poisoning cases are reported by tetraethyl lead, among the petrol tank cleaners[6]

** Antimony trisulphide – Also called surma*

*** Mercuric sulphide – Also called vermillion*

Treatment of Acute and Subacute Poisoning

- Emetics
- Stomach wash with 1 per cent magnesium or sodium sulphate solution
- 25 gm of magnesium sulphate orally with demulcent drinks
- Calcium gluconate 1 gm to relieve colic
- Intravenous fluids
- Chelating agents like EDTA, BAL and penicillamine are helpful.

Chronic poisoning (Plumbism, Saturnism) Chronic poisoning with lead compounds manifests with a set of symptoms, which may be enumerated as:

- *Facial pallor:* Pallor seen especially around the mouth also known as *circum oral pallor* is due to the *vasospasm* of the capillaries and arterioles, around the mouth.
- *Anaemia: Hypochromic, microcytic anaemia* with *reticulocytosis* and *punctate basophilia* with presence of marked *basophilic stipplings* in the RBCs. Platelet count decreases. Anaemia is probably due to decreased survival time of RBCs and inhibition of haem synthesis by interference with the incorporation of iron into protoporphyrin.
- *Burtonian line (lead line):* It is a stippled blue line seen at the junction of the gums usually nearer to a tooth caries, especially in the upper jaw. This is due to the deposition of lead sulphide formed by the action of the combination of lead with hydrogen sulphide which had evolved from the decomposed food debris in the caries tooth.
- *Lead colic and constipation*: The victim will complain of severe colicky pain abdomen relieved by pressure and bowel irregularities. Abdominal muscles become tense and retracted.
- *Lead palsy:* There is a typical paralysis affecting the extensor muscles of the fingers and wrist causing '*wrist drop*' and '*claw shaped hand*'. Similarly paralysis may extend to the extensor muscles of the foot leading to foot drop.
- *Lead encephalopathy:* Mostly seen in infants presenting with severe ataxia, vomiting, lethargy, stupor, convulsion and coma. Cerebral psychic affection may be present.
- *Cardiorenal manifestations:* Elevated blood pressure and arteriosclerotic changes are observed. Urine contains albumin and abnormal quantity of lead, coproporphyrin III and delta-amino laevulinic acid. Interstitial nephritis may occur.

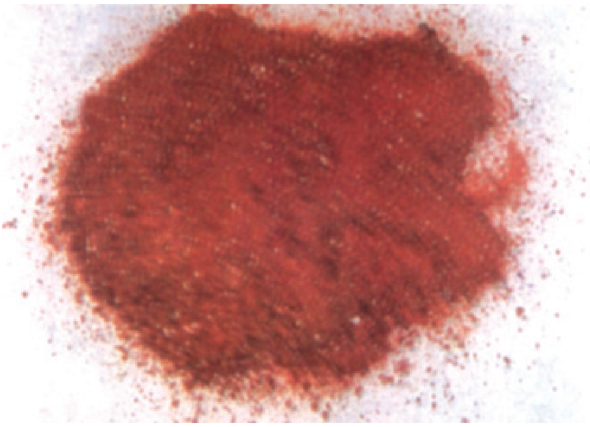

Fig. 33.2: Inorganic irritant: mercuric sulphide (vermilion)

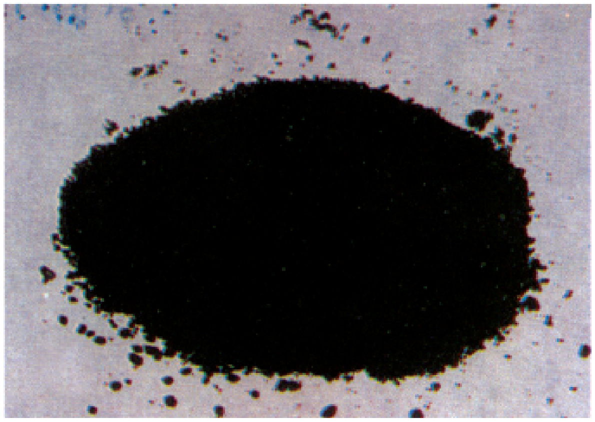

Fig. 33.3A: Inorganic irritant: lead sulphide

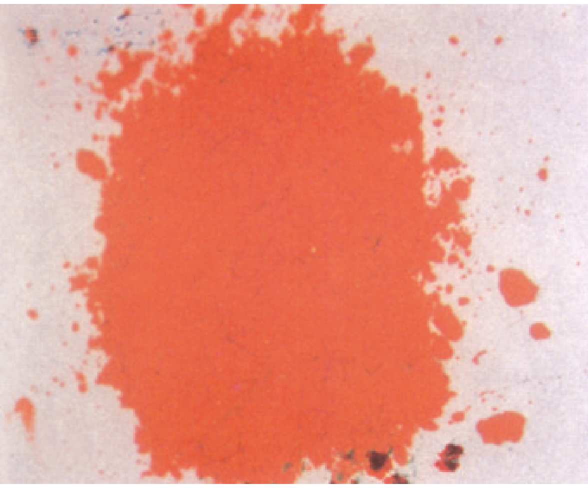

Fig. 33.3B: Inorganic irritant: lead tetroxide (red lead, vermilion)

- *Sterility/Infertility*.
- *General manifestations:* such as weakness, anorexia, metallic taste in the mouth, dyspepsia, foul breath, etc.

Laboratory Diagnosis of Chronic Lead Poisoning

- Urine lead levels of more than 0.08 mg per litre collected in 24 hours
- Blood lead level more than 0.8 mg per litre
- Increased coproporphyrin level in urine
- Increased urine and plasma delta-amino laevulinic acid
- X-ray evidence of *increased density* or *radio opaque bands* or *lines* at the metaphyseal ends of long bones in children. This is are also referred to as *lead lines*.[12]
- Presence of lead as *radio opaque material* on X-ray stomach and intestines may be seen in children particularly with history of *pica* (meaning abnormal craving for non-nutritive substances).

Treatment

- Potassium or sodium iodide for eliminating lead through the kidney.
- Large dose of sodium bicarbonate: 20 to 30 gm per day in divided doses increases the output of lead owing to the transformation of the insoluble tribasic lead phosphate into the soluble dibasic lead phosphate through the liberated carbonic acid.
- Calcium gluconate or calcium chloride to relieve colic
- Saline purgatives like magnesium sulphate or sodium sulphate to remove lead from the bowel
- Calcium disodium versanate as deleading agent

Postmortem Changes

Acute Poisoning

External: Nothing specific.

Internal: Stomach-gastric mucosa is congested, eroded and patchy in appearance with greyish white deposits. Large intestine may show black colored faecal matter. Evidence of renal tubular degeneration.

Chronic Poisoning

Blue line on the gums. Muscles are flaccid and show fatty degeneration. Intestines are contracted and thickened. Liver and kidneys are hard and contracted. Heart is hypertrophied. Renal tubular necrosis is usually noticed.

Medicolegal Importance

- Lead is an *industrial poison* presenting as an *occupational hazard*. It is commonly used in industries concerning manufacturing of battery cell, paints, crayon, hair dyes, toys, etc. Accidental poisoning by contamination of the drinking water occurs in places where *lead pipes* are used. Drinking fruit juices or water stored in improperly glazed ceramic wares can result in lead poisoning.
- Lead is *rarely* preferred to commit suicide or homicide.
- In cases of long standing gun shot bullet/s lodged and retained in the body, have reported of chronic lead poisoning due to absorption of lead particles from these bullets.[1] Gunshot wounds in adults and children has been reported of causing anorexia, abdominal pain, vomiting, anaemia, encephalopathy, seizers, etc.[17] The surface area of retained lead particles, location of retained lead particles (especially synovial fluid), length of time for which one is exposed to lead and type of activation (uncoated bullets-yielding greater surface area of lead for dissolution) are all factors that may lead to lead poisoning.[1]
- Long standing use of cosmetics containing lead salts (*surma, sindhur, vermillion,* etc) can result in chronic lead poisoning.

COPPER *(Thambe, Blue Vitriol)*

Copper, an inorganic metallic irritant, is not poisonous in metallic state, but some of its salts are poisonous, e.g. copper sulphate *(blue vitriol)* and copper subacetate *(virdigris)*.

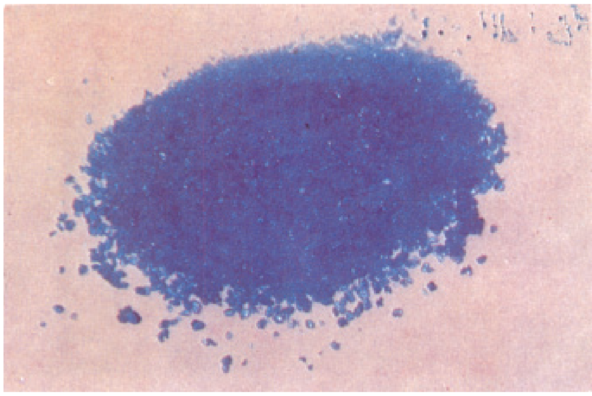

Fig. 33.4: Inorganic irritant: copper sulphate

Copper Sulphate

Copper sulphate is a crystalline salt with blue colour (Fig. 33.4) and has a metallic taste. In small dose of 0.5 gm it acts as an emetic, but in large doses it acts as an irritant poison. Poisoning is usually accidental or suicidal. Homicidal use is rare because of its metallic taste and striking blue colour.

Copper Subacetate or Verdigris

Copper subacetate is a bluish green salt. It is formed by the action of vegetable acids, while cooking in copper cooking utensils, which have been not properly tin lined. Thus, accidental *verdigris* poisoning from contamination of food cooked in such utensils, are often reported.

Signs and Symptoms

Acute Poisoning

- It is reported that renal failure and death may follow ingestion of as little as 1 gm of copper sulphate.[18] However, fatal poisoning by copper is very rare.[1] Symptoms of poisoning commence within fifteen to thirty minutes after swallowing the poison. There is a metallic taste in the mouth. Salivation and thirst are present. The mucosa of the mouth is discolored blue. There is pain in the mouth, oesophagus and stomach. Vomiting and diarrhoea occur. Vomitus is blue or green in colour. Stool is brownish or bloody. Oligurea, haematuria and uraemia may develop in some. There may also be low urinary output with casts and albumin in urine. Jaundice occurs in severe cases due to centrilobular necrosis and biliary stasis. Later muscular spasms cramps, coma and circulatory collapse precede death.
- A rare syndrome of *intravenous copper intoxication* with symptoms of nausea, vomiting, abdominal pain, diarrhea, anxiety and depression due to copper released from copper tubing during *haemodialysis* was noticed among patients undergoing haemodialysis.[19]
- ***Fatal dose*** Copper sulphate—30 gm
  Verdigris—15 gm
- ***Toxicity rating*** 4 for copper salts.

Treatment

- *Stomach washes* with warm water. Egg albumin acts as an antidote by forming an insoluble and innocuous copper albuminate. Stomach wash with potassium ferrocyanide 1 per cent solution in water also acts as an antidote by forming cupric ferrocyanide.
- *Calcium EDTA or BAL* is the recommended antidote.
- Maintain *electrolyte and fluid balance.*

Autopsy Finding

- Skin may be yellow due jaundice.
- The mucosa of the mouth, oesophagus and stomach are discolored greenish-blue and may show areas of corrosion and congestion.
- Colon and rectum may show large ulceration or perforations.
- Liver may be enlarged and show fatty degeneration.
- Copper is one of those poisons that can be detected by its characteristic colour.
- The kidneys are congested and may show focal necrosis of *proximal tubules*.[1]

Chronic Poisoning

Chronic poisoning is common among the industrial workers of copper and copper salts or its alloys owing to inhalation of copper dust or fumes. Copper welders may develop the *metal fume fever*.[6] Chronic copper poisoning is also observed among those who consume contaminated food with verdigris obtained from dirty copper vessels for a long period.

Airborne dusts of inorganic copper salts have been reported to produce low toxicity. *Hystiocytic granulomatous lung* and *liver disease* have been observed among individuals who had been exposed to copper sulphate spray for 2 to 15 years.[1]

Sign and Symptoms

- The symptom complex of chronic poisoning is called by several names: *hemochromatosis, bronzed diabetes* and *pigment cirrhosis*.[6]
- It presents with green or purple line on the gums, coppery taste in the mouth, nausea, headache, colicky pain, vomiting and diarrhoea, and anaemia. Atrophy of the muscles may be the other symptom observed.
- Skin is jaundiced.
- Urine and prespiration become green.

Treatment

- Remove the cause and prevent further exposure.
- Provide fresh air.
- Give massage and warm bath.
- Provide proper diet.
- Copper vessels if used for cooking, should be tinned and regularly kept scrupulously clean.

Autopsy Finding

No findings externally in acute poisoning cases. The mucosa of mouth and tongue may show bluish or greenish blue tinge. Internally the same tinge is observed with mucous membranes of oesophagus and stomach. Stomach mucosa is congested desquamated and hemorrhagic. Upper part of small intestine may also show mild to moderate irritation. The chief findings are fatty degeneration of liver and degeneration of the epithelial cells of the kidneys.

In chronic poisoning cases gums appear unhealthy with bluish lining. There is mucosal atrophy. Liver and kidneys show varying degree of degeneration. Poisoning due to Inhalation of vapours chronically can present with findings of chronic pneumonitis. Blood picture may show premature cells in the peripheral smear of the victim.[20]

Medicolegal Importance

- Copper coins when swallowed may remain in the stomach or in the intestine for days without producing any poisoning symptoms. However, when alloyed with other metals and reduced to a fine powdery state, copper may act as poison. All copper salts are poisonous.

- The colour and the strong metallic taste prevent it from being used for homicidal purposes. However cases of using copper sulphate mixed with powdered glass, sweetmeat or some other food is known in India.
- Fair number of suicidal cases are reported in India as it is used in leather industry and for white washing.
- Accidental poisoning by contaminated food due to verdigris is often reported.
- Copper sulphate is used often as a preservative or colouring agent to vegetables. It is added often to impart rich green colouration to tinned green peas, and mango pickles. Quantity added is usually small (<60 mg) and hence toxic effects are not produced. Conversion into harmless albuminate of copper in the stomach may be the other reason for resultant toxicity. However, constant consumptions of such food articles can lead to chronic poisoning manifestations.
- Copper is a normal and essential constituent of human body and is found in urine, feces, blood and other biological fluids and in liver (Normal serum level is 151.6 micrograms).[6]

IRON

Iron is an inorganic metallic irritant. Mills and Curry have comprehensively surveyed the current status of iron intoxication.[21] There has been an annual average of 22,000 reported exposures to medications containing iron over last 3 years. Most exposures involve children less than 6 years of age who have ingested paediatric multivitamin preparations. Most of these patients remain asymptomatic or develop minimal toxicity. Concentrated iron supplements overdoses more often results in serious poisoning and can present to emergency department at any stage. However, if the patient doesn't develop any symptoms within 6 hours of ingestion, it is unlikely that iron toxicity will develop.

Iron salts are used for treatment of prophylaxis from iron defeciency anaemia. There are several Indian iron preparations, containing different amounts of elemental iron (Table 33.10). Usually iron salts poisoning incidences are reported in children due to consumption of adult dose by mistake, or while giving intravenous injection. Ferrous sulphate and ferric chloride are some of the toxic compounds.

Action

The early features of iron poisoning are due to corrosive effects of iron, while later effects are largely due to the disruption of cellular process. Iron tablets may adhere to the stomach and duodenum causing irritation and in severe cases haemorrhagic necrosis and perforation.[12] Absorbed iron is rapidly cleared from extraellular spaces by uptake into parenchymal cells, particularly in the liver. It causes mitochondrial damage and cellular dysfunction resulting in metabolic acidosis and necrosis. Eventually widespread organ damage become apparent, hepatic failure with hypoglycaemia and coagulopathy may develop and this is often fatal.[12]

Signs and Symptoms

The clinical course of iron poisoning occurs in four phases:

Phase 1*:* In the first few hours (from 30 minutes to several hours) after ingestion, there is vomiting, abdominal pain and haemorrhagic gastroenteritis with black or grey vomitus and stool with metallic odour. In severe cases gastrointestinal haemorrhage can result with circulatory collapse and coma may supervene.

Phase 2*:* In the second stage, 6-24 hours after ingestion patient shows improvement and the clinical symptoms abate and the patient either recovers or moves on to next phase. In severe cases this may not appear or a latent phase occurs and is deceptively reassuring.[12]

Phase 3*:* Occurs in 12 to 48 hours after ingestion, which is characterized by severe lethargy, coma, convulsions, gastrointestinal haemorrhage, shock, cardiovascular collapse, metabolic acidosis, hepatic failure with hepatocellular necrosis, jaundice, hypoglycaemia, coagulopathy, pulmonary oedema and renal failure.[12]

Phase 4*:* This is a late phase of complication after 2-5 weeks with formation of gastric strictures and pyloric stenosis.

Diagnosis

- X-ray abdomen shows iron tablets
- Serum Iron level > 150 microgram per cent
- ***Fatal dose*** 20-40 gm of ferrous sulphate/> 150 mg of elemental iron

Table 33.10: Common Indian preparations and brand names with iron compounds in each one and elemental iron content of some of them*

Brand names	*Iron preparations*	*Elemental iron (mg)*
Aglow Fe, Anemidox, Astyfer, Autrin, Cheri, Duron-T, Elferi-Z, Hemplus, HiFi, Livogen, Pronutrin, Victofol, Ziferin-TR	Ferrous fumerate	65 mg
JP Tone, RB Tone	Ferrous gluconate	35 mg
—	Ferrous succinate	35 mg
Conviron-TR, Fefol, Ferrostan, Fesovit, Iberol, Ultiron-TR, Winfol, etc	Ferrous sulphate	60 mg
Inferon	Iron dextran	—
Dexoren plus, Globiron, Haem up, Hemfer, Hepp forte, Hi-Fi, Ruberplex	Ferric ammonia citrate	—
Incremin	Ferric pyrophosphate	—
Raricap	Ferrous calcium citrate	—
Toxic dose of elemental iron are: <30 mg/kg – Mild toxicity >30 mg/kg – moderate toxicity >60 mg/kg – Severe Toxicity >150 mg/kg – lethal		

* *Modified from Alison LJ, Paul ID. Churchill's Pocket Book of Toxicology. Churchill Livingstone, Edinburgh, London, 2001, Table 2.2, pp55.*

- ***Fatal period*** Uncertain
- ***Toxicity rating*** 3.

Treatment

- Gastric lavage, with dilute (2% solution) of sodium bicarbonate.
- Demulcent drinks like milk or egg albumin is useful.
- Whole bowel irrigation in acute poisoning is found to be safe and effective method. However, there is no report on controlled studies confirming this.[22]
- Electrolyte correction. Intravenous glucose.
- *Antidote*: Deferoxamine (Desferrioxamine) is the specific antidote. A solution of 2 gm in 1 litre of water can be used for gastric lavage; followed by 2 gm in 10 ml sterile water should be left in stomach. 2 gm of this is then given intramuscularly or by a slow IV infusion at the rate of 15 mg/kg body weight per hour, to a maximum of 80 mg/kg in 24 hours.

Medicolegal Importance

- Till recently iron was believed to be nontoxic. However, it is well known today that iron salts in excess can produce poisoning and acute toxicity often results in children due to over dose. Iron poisoning is usually, accidental.
- Suicidal or homicidal ingestions by iron are rarely reported.

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VEGETABLE AND ORGANIC ANIMAL IRRITANT POISONS

INTRODUCTION

An extended communication on all the Indian poisonous plants in a particular region is though beyond the scope of this section, some important plants commonly involved in poisonings, follows logically. It would be however relevant to mention here that a great deal of ignorance about these poisonous plants is a fact even among many of the clinicians routinely dealing with poisoning cases. The problem is made more intense by the fact that there is no accurate information available in India, since very few cases are reported or published in literature. It is also agreed that many of the experimental works are performed on laboratory animals and discussed in veterinary literature. The applicability of such studies in human beings is an open challenge. It is also true that many of plant poisoning cases the treatment is practically same, i.e. symptomatic measures and supportive therapy. Rarely these cases have any antidote therapy. There is virtually chaos in the areas of plant identification and nomenclature. Table 33.11 presents an idea on some of the Indian plant poisons.

ABRUS PRECATORIUS

Distribution—Grows all over India

Common name—Jequirity bean, rosary pea, Buddhist rosary bead, rosary bead, Indian bead, Indian liquorice, Seminole bead, prayer head, crab's eye, weather plant, lucky bean, *ojo de pajaro, gulagunchi, rati,* etc.[1,2]

Family—Leguminoceae.

Plant characteristics—It is a slender vine and climber, with compound leaves having 10-15 pairs of narrow leaves, small pinkish flowers with seedpods which split open when ripe exposing 4-6 seeds within. These seeds are bright red in color with black spot in one pole and weigh about 105 mg (Figs 33.5A and B).

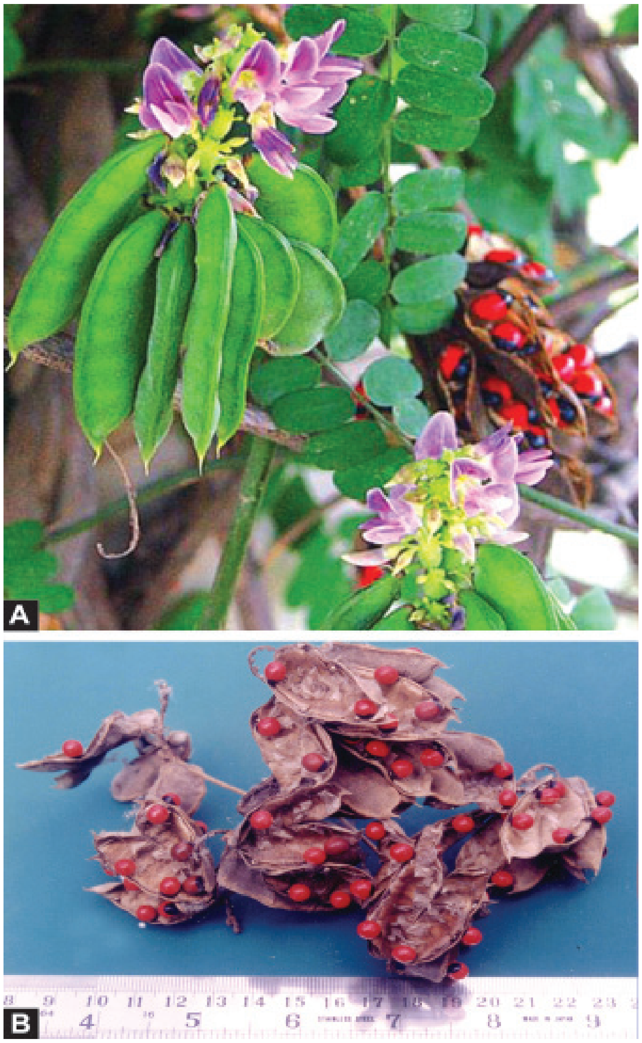
Toxic part of the plant—*Whole* plant is poisonous. However, seeds are more often used.

Toxic Principles[1,2]

- N-methyltryptophan
- Glycyrrhizin (lypolytic enzyme—the active principle of licorice)

Table 33.11: Enumerating some of the common Indian vegetable irritant poisons

Poisonous plants	*Common names*	*Main active principles*
Abrus precatorius	Jequirity, rosary bead, Indian Liquorice, gulagunchi, rati	Abrin
Ricinus communis	Castor, arandi, mole bean	Ricin
Croton tiglium	Croton, jamalgota	Crotin, crotonoside
Calatropis	Madar plant, calatropis gigantea, Calotropis procera	Uscharin, calotoxin, Calatropin gigantin
Semicarpus anacardium	Marking nut, bhilwa, bibva, Bhela, oriental cashew	Semicarpol, bhilawanol
Capsicum annum	Chillies, lal mirchi	Capsaicin, capsaicin
Ergot	Claviceps purpura	Ergotamine, ergotoxin and ergometrine
Eucalyptus globulus	Blue gum	Eucalyptol (cineole)
Azadirachta indica	Neem stearic acids, aflatoxins.	Linoleic, oleic, palmitic and
Colchicum autumnale	—	Colchicines, demecolcin



Figs 33.5A and B: Abrus precatorius: (A) Plant, leaves, flowers, pods and (B) Seeds

- Abrin (Toxalbumin*)
- Abrine (amino acid)
- Abralin (glucoside)
- Abric acid

Signs and Symptoms

Signs and symptoms manifest only if the seed is masticated and swallowed. It can act both locally as well as remotely.

Locally—can lead to dermatitis, conjunctivitis, rhinitis, asthma, etc. Oral ingestion can produce severe gastroenteritis, hemorrhagic gastritis with severe pain, copious vomiting, and diarrhoea that may become bloody, severe thirst and circulatory collapse. *Death* is reported to be due to persistent gastroenteritis.

Remotely – when implanted as ‘suis’ or the seed extract is injected parenterally, the person can develop cardiac manifestations like a *viperine snakebite*, with the site of injection turning oedematous and haemorrhagic. Victim (animal/human) then turns drowsy, unable to move, goes into coma, followed by convulsions and death. According to Seth, Lal et al abrin can lead to development of cardiac arrhythmias, convulsions and cerebral oedema.

Usual Fatal Dose—60-120 mg of abrin (1-2 crushed seeds)

Fatal Period—3-5 days

Toxicity Rating—5 to 6 *(Supertoxic)*

Treatment

All cases who report within 4 hours of ingestion should be treated by usual method of decontamination (lavage, charcoal, and cathartics). The presence of spontaneous diarrhea may obviate the need for cathartics. However following have also been found to be effective:

- Oral poisoning cases give: Acid hydrochloric pepsin mixture and 10 per cent sodium bicarbonate IV.
- Local injected cases: Dissect out the *Suis*.
- Symptomatic measures as required.

Postmortem Findings

- Findings show inflammatory changes and congestion of gastrointestinal tract.
- When injected, local signs of inflammation are seen.

Medicolegal Importance

- It is a commonly used cattle poison in Indian villages by injecting the seed extract into the animal in the form of certain fine needle-shaped structures known as *Sui* (meaning *needle* in *Hindi*). These are prepared by mixing the seed extract with opium, datura, and spirit/water and then blended into paste, shaped into fine needles and dried in the sun and used to kill cattle by driving it deep into the animal body by blowing through a hollow bamboo pipe.

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* Also known as ‘phytotoxin’ is described as a toxic protein, which resem—bles a bacterial toxin in action and causes agglutination of RBCs and haemolysis. It is antigenic in nature and is therefore, capable of producing antibodies when injected into the body.[2,3] Ricin, crotin and snake venom are some of the other examples for toxalbumin.

- *Suis* have been also used criminally and reported of *homicides* often in the Indian villages. It is kept in between the fingers of hand and slapped on the face of a victim, driving it deep into the skin, which releases toxic principle abrin and brings about its action.[3] An interesting case example is narrated below:

  A man was sleeping on a charpoy, when someone came and slapped him on his right cheek, developing a wound which showed presence of a foreign material. Several such foreign materials were detected in the charpoy, was confirmed to be pieces of Suis containing ground substance of abrus precatorius. Death occurred in two days after the symptoms of inflammation occurred in the chest, eyes, neck and mouth.
- Seeds are often used in rosary beads, necklace, etc. in rural India.
- Seeds reported of accidental poisoning among children, on account of their attractive colour and ingested orally, mistaken for a peppermint or just out of curiosity.
- Indian goldsmiths sometimes use seeds as a measure to weigh gold or precious stones.
- Seed are reported to have being used as birth control pills in the past in rural India.

RICINUS COMMUNIS *(Castor Oil Plant)*

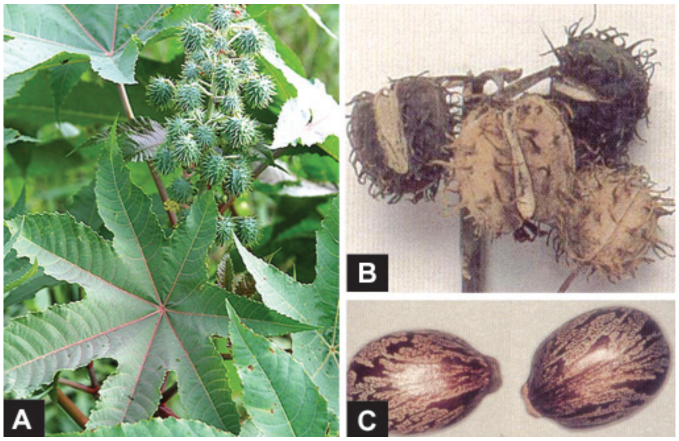
Distribution—Grows all over India, especially in waste lands.

Common name—Castor, arandi, mole bean.

Family—Euphorbiaceae.

Plant Characteristics—It is a large shrub with greenish-red leaves. Fruits are borne in clusters and are soft-spined greenish/ brownish capsules with seeds. Seeds are oval/round in shape and are of two types:[3] larger in size, red in colour with brown blotches (yields 40% oil) and second variety small in size, grey in colour with glossy bright, polished, brown mottling (yields 37% oil) (Figs 33.6A to C).

Toxic part—Seeds, especially the seed oil (*castor oil*) extract, which is pale yellow in colour and with faint odour and an acrid taste. Left over *cake* after the extraction of oil is also highly toxic.



Figs 33.6A to C: Castor plant: (A and B) Leaves, fruits and (C) Glossy brownish coloured seeds with mottling on it's surface

Active principle—The oil extract of the seeds has an acid called *ricinoleic acid* and the left over cake has the *toxalbumin* called *ricin*. Ricin is one of the most toxic *parenteral* substances in the plant kingdom. It contains two polypeptide chains held together by a single disulphide bond. Both these chains can bind with cell surface facilitating toxin entry into the cell and then disrupt the protein synthesis. Since the cell binding and protein disruption needs some time, its toxic effects are usually delayed but are widespread.[1] Ricin is more poisonous than cobra venom and is classified as *super toxic poison*.[4]

Signs and Symptoms

Locally—It can lead to dermatitis, conjunctivitis, rhinitis, asthma, etc. Castor bean dust is highly allergenic and may cause *anaphylaxis*.[5]

Orally—Seeds are effective orally only if *masticated* and swallowed.[2] It produces burning pain in the throat, followed by nausea, vomiting, colicky pain in the abdomen and bloody purging. Both can ultimately lead to dehydration, muscular cramps, etc.

Parenterally—It can produce same manifestation as on oral ingestion, but occurs more rapidly than oral route.

Fatal dose—1 mg/kg body weight or 6 mg of ricin (about 8-10 seeds).

Fatal period—Several days.

Toxicity rating—Ricin 6 (Super toxic), castor oil 2 (Slightly toxic).

Treatment

Every patient should receive the usual measures to prevent absorption (syrup ipecac, charcoal, cathartics) after taking usual precautions. Recommended treatment for asymptomatic patient who has chewed one or more seeds include emergency department evaluation, gastric decontamination, administration of activated charcoal, observation for 4 to 6 hours, and discharge with instruction to return if symptoms appear. However, all symptomatic patents need hospitalisation for treatment with IV fluids, supportive care, and monitoring for hypoglycaemia, haemolysis and complications of hypovolaemia. Most patient respond well to IV fluid therapy and recover without any permanent sequelae.[1]

Enumerated below is the line of management:

- Prevention of absorption by giving syrup ipecac inducing emesis/ activated charcoal/ cathartics.
- Gastric decontamination by stomach washes with water.
- Give plenty of demulcents.
- Rehydrate the victim by intravenous fluid and maintain electrolyte balance.
- Blood transfusion may be needed in some cases.[3]
- Other symptomatic measures as needed.

Postmortem Findings

Inflammatory changes and congestion of gastrointestinal tract. Liver, kidney and pancreas are considered as primary target organs clinically,[1] may show inflammatory changes and congestion on autopsy.

Microscopy: Microscopic examination, of stomach contents in a victim, revealing a prismatic appearance of the outer cells coat of the castor seeds (and also in *croton, abrus, and jatropa seeds*), has helped in identification of the poisoning.[3]

Medicolegal Importance

- Castor oil obtained from smaller variety seeds, is usually used in medicine purposes as a *purgative*. Oil which is obtained from bigger variety of seeds is used largely for illumination purposes or as an industrial lubricant.[3]
- Poisoning is usually accidental.
- *Georgi Markov Case* – This is an interesting case of homicidal ricin poisoning reported in London. Bulgarian traitor, Mr. Markov, who lived in London, was waiting on the bus stop, one evening, got stuck by a sharp jab in the back side of his right thigh. A by-stander had accidentally poked in his umbrella tip, apologized Mr. Markov and left. Mr. Markov turned seriously sick in the night with fever and gastrointestinal distress, was immediately admitted to a hospital, worsened steadily and died on the third day. As cause of death was not established, autopsy was performed, which revealed a *tiny pellet* in the thigh where the umbrella had poked. It had two small holes bored into its casing. On laboratory analysis, pellet revealed presence of *ricin* in it and the case was ruled as a case of homicidal poisoning by ricin, perhaps a political assassination.

Croton Tiglium

Distribution—Croton plant grows all over India, especially in wastelands. Grown in many varieties for their brightly coloured foliage; it is widely cultivated as a houseplant (Figs 33.7A to D).

Common name—Croton, *Jamalgota, Naepala.*[3]

Family—Euphorbiaceae.

Plant Characteristics—It is an evergreen tree with smooth ash-colored bark. The leaves of the tree are ovate-lanceolate. Flowers are small, and oblong. Fruits are three lobed containing oval, dark brown seeds, with brownish black colour and longitudinal striations (Fig. 33.7D). Seeds though resemble castor seeds; the longitudinal striations mark the difference from castor seeds, which has mottling (see Fig. 33.6C).

Toxic part—Seed and oil extracted from the seeds is extremely toxic. Seed oil is commented to have tumour promoting *phorbol diesters*.[1]

Active principles—There are two active principles:[2]

- Crotin (toxalbumin)
- Crotonoside (glycoside)

Signs and Symptoms

Resembles *Ricinus communis* (castor) poisoning manifestation

Fatal dose—1 to 2 ml of oil or 4 to 6 crushed seeds

Fatal period—4 to 6 hours to 3-6 days

Toxicity rating—5 (croton oil)

Treatment

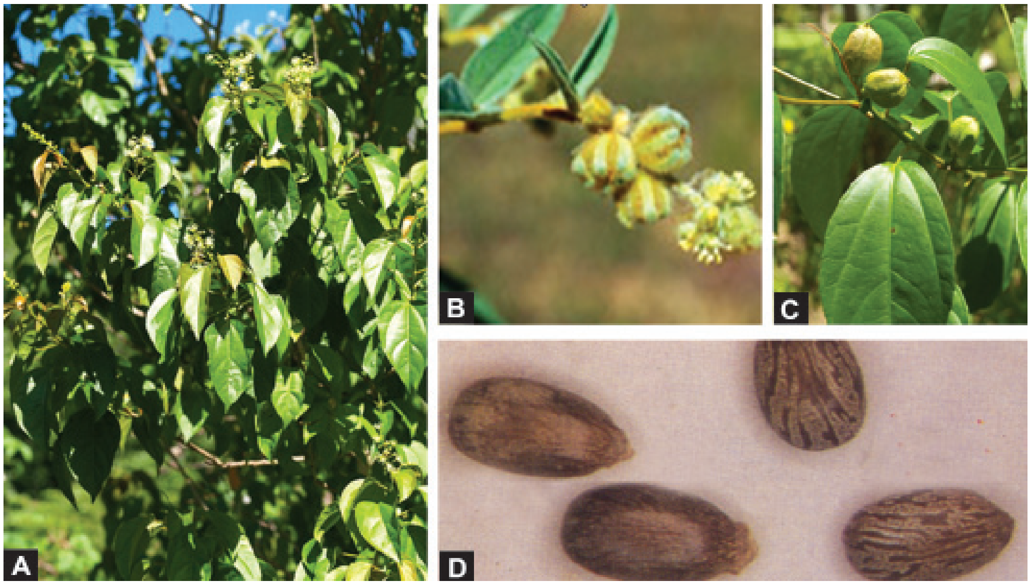
Same as for *Ricinus communis* (castor).[2]

Postmortem Findings

Findings show inflammatory changes and congestion of gastrointestinal tract.

Medicolegal Importance

- Mistaken for castor oil or castor seed, resulting in *accidental* poisoning. Some of the typical accidental poisoning cases reported are enumerated below:
  Case of wedding party ice-cream—at a wedding party in Delhi, after the dinner ice cream was served to every one. All those who consumed it developed stomach disorder and vomiting. Later on it was found to have been accidentally mixed with croton oil.[6]
- Cases of deliberate homicide are also reported with Croton oil. Madras Chemical examiners report mentions one such case in 1936.
  Case of poisoning by croton seed—Two brothers had stolen bales of yarn and Rs. 200 cash. They were invited by the owner of yarn for a *pooja* in which they received some white pill like materials at the end of the pooja in which they performed by a *quack*. The quack gave some black pills to others who assembled. One of the brother swallowed the entire pill while the other who was little suspicious took only a portion of the pill and rest he preserved. The first one who swallowed entire pill developed severe purging, vomiting blood and died within 24 hours, while the other who consumed partly, suffered similar but not very severe



Figs 33.7A to D: Croton tiglium: (A) Plant with leaves and flowers; (B) and (C) Fruits (D) Seeds glossy brownish black colour with longitudinal lines

complaints and survived after being given treatment at the hospital. Chemical examiner reported material resembling the outer covering of croton seeds in the intestines of the deceased. Similar material was detected in the pill remnants with second victim. The police also recoverd a grinding stone showing similar material like outer covering of croton seed. The quack who confessed everything was later arrested. He was sentenced to rigorous imprisonment for two years.[7]

- Rarely the croton oil is used as an *abortifacient* in rural India.

CALOTROPIS

Distribution—Grows all over India, especially in wasteland and deserts.

Common name—Madar has two species.[2]

- *Calotropis gigantea, which* is a purple flowered plant.
- *Calotropis procera, which* is a white flowered plant

Family—Asclepiadaceae.

Plant characteristics—It is a tall shrub with yellowish-white bark, and oblong thick leaves and purplish or white flowers (Fig. 33.8). When the stem, branches and leaves are cut, crushed or incised, it yields milky white latex, which is an acrid juice called *madar juice*.

Toxic part—Stem, branches, leaves and the milky white latex *(madar juice)*.

Active principles—They are four:[2]

- Uscharin
- Calotoxin
- Calotropin
- Gigantin

Signs and Symptoms

Locally—It can give rise to lesions resembling bruises on skin (called *fabricated injuries*), which at times can lead to pustule formation and vescication. Juice when instilled into the eyes or coming in contact with eyes can result in severe *conjunctivitis*.

Orally—Bitter in taste. Produces burning pain in the throat, salivation, nausea, vomiting, etc. followed by diarrhea, pain abdomen, mydriasis, tetanic convulsions, delirium, collapse and death.

Fatal dose—Uncertain.

Fatal period—12 hours.

Treatment

- Gastric lavage with warm water or potassium permanganate ($KMnO_4$)
- Give demulcent drinks
- Symptomatic measures
- Washing with soap and water can treat skin lesions.
- Cases of conjunctivitis can be managed by saline irrigations.

Postmortem Findings

Froth at nostrils, stomatitis and inflammatory changes of gastrointestinal tract with ulceration, stomach may show perforation. All viscera including brain usually shows congestion.

Medicolegal Importance

- Parts of the plant or the juice extract are used as folk medicine in rural India.
- Accidental poisoning is common due to quackery.
- The criminal use of the juice includes criminal abortion, infanticide, cattle poisoning, and creating conjunctivitis and *artificial bruises*.

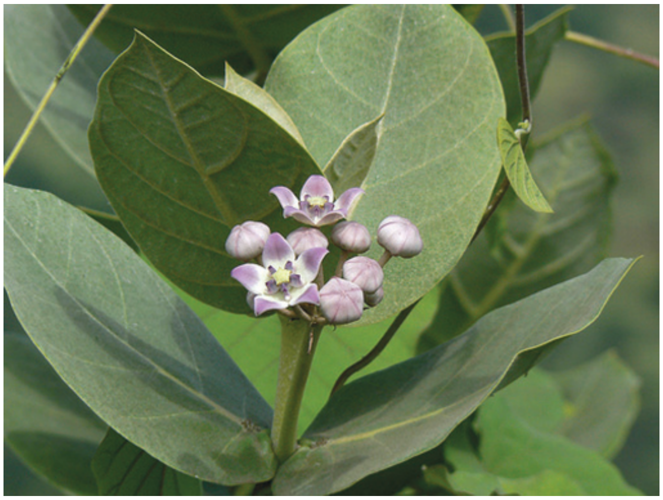


Fig. 33.8: Calotropis (Madar) plant. Note the leaves and flowers

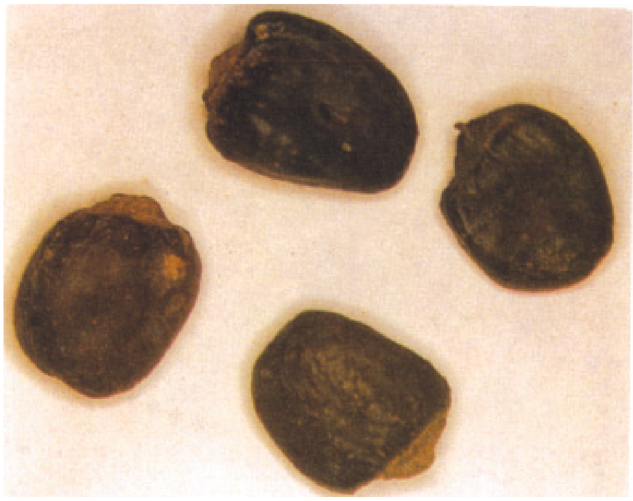


Fig. 33.9: Semicarpus anacardium. Note brownish black and heart-shaped seeds

SEMICARPUS ANACARDIUM

Distribution—Grows all over India.

Common name—Marking nut, Bhilwan, Bibva, Bhela, and Oriental Cashew.[2]

Family—Anacardiaceae.

Plant Characteristics—It is a small tree of East Indian origin. Its flower is dull/greenish yellow in colour. Fruit is black, heart-shaped with hard rind (Fig. 33.9) within which is a thick fleshy pericarp which yields brown oily resinous fluid. This turns black on exposure to air. This fluid is often used as 'marking ink', on linen and cotton clothes by the washer men (*Dhobis*).

Active principles Two active principles are isolated in the fluid extracted from the pericarp:[12]

- Semicarpol (monohydroxy phenol compound).
- Bhilawanol (alkaloid).

Signs and Symptoms

Locally—On skin produces bruise like lesions which are actually raised blackish blisters or vesicular ecchymatous eruptions, which are itchy and scratching of which can cause similar lesions on the tips of fingers, on the nailbeds, below the nail tips. These can lead to pain, fever and stranguria with excretion of brownish urine.

Orally—Large dose can produce blisters in mouth and throat, with gastroenteritis. Can also produce dyspnoea, cyanosis, tachycardia, coma and death.

Fatal dose—Uncertain.

Fatal period—12 to 24 hours.

Treatment

- For skin lesions, wash with water and apply liniments.
- For oral ingestion cases perform gastric lavage and give demulcents
- Symptomatic measures as needed.

Postmortem Findings

Inflammation of gastrointestinal tract and congestion of viscera and skin showing black vesicles with acrid serum.

Medicolegal Importance

- Common criminal use of the juice is by malingerers' (those who pretend *injury* or *illness* with some hidden motives) to fabricate wounds (usually a *bruise*) by external application over the skin. However, unlike the true bruise the lesion produced by the fluid will result in blister formation with irritation/itching. A dilute solution of the fluid is also at times instilled in the *eyes* by malingerers' to induce abrupt *conjunctivitis* or *ophthalmia* as conjunctivae turn red due to local irritation effect.
- Accidental poisoning is also common when used as *a medicine* by *quacks*.
  - A case of applying an oily preparation containing marking nut juice by a *hakim* to paralyzed limbs of twelve years old child, as apart of treatment resulted in death has been reported by the Chemical Examiner Mumbai.[3]
- Criminally it has been used to pour over geniatalia or introduced into the vagina for infidelity:
  - A man had an affair with a neighbouring woman during absence of his wife. The lady was annoyed when her lover's wife arrived. When the man was not home, she got into the house of the couple stealthily and poured marking nut juice mixed with oil over the genitalia of her lover's wife when she was asleep out of frustration for breaking her love affair. The woman was charged of voluntarily causing *grievous hurt* by means of poison under section 320 IPC.[3,13]
- Juice if often used as an *Illegal abortifacient* to induce *criminal abortion* by its local application through the *abortion stick* with a cloth piece soaked in it, wrapped over its tip and thrust into the uterus.[2,3]
- Modi has reported cases of homicide and infanticide by marking nut juice:[3]
  - A wife after a quarrel with her husband gave him a drink mixed with ground marking nut. Man developed severe gastrointestinal irritation and died within twelve hours.
  - A Hindu female administered ground marking nut to a seven month infant with her finger. Child became sick, developed vomiting and diarrhea followed by blisters on tongue and chest, and died with in twenty four hours.
- The fruit extract poured on the body (especially *genitalia*) of the prisoners for extortion of truth or confession of guilt.
- Occasionally used to commit *vitriolage*.

CAPSICUM ANNUM

Common name—Chillies, *Lal mirchi, Red pepper, cayenne pepper.*[2,3]

Family—Solanaceae.

Plant characteristics – It is a small herb bearing somewhat long, tapering fruits, which become red when ripe, and possess a pungent odor and taste. The fruit (chilly) contains a number of small, flat, yellowish seeds (Figs 33.10A and B), which bear a superficial resemblance to datura seeds. Table 33.12 presents with the differences.

Toxic part—Fruit and the seeds

Active principles—Capsicin (crystalline) and capsaicin are both acrid, volatile, alkaloid substances.[3]

Signs and Symptom

Locally—It can produce irritation resulting in burning and redness of skin; and burning, redness, and lacrimation of the *eyes*.

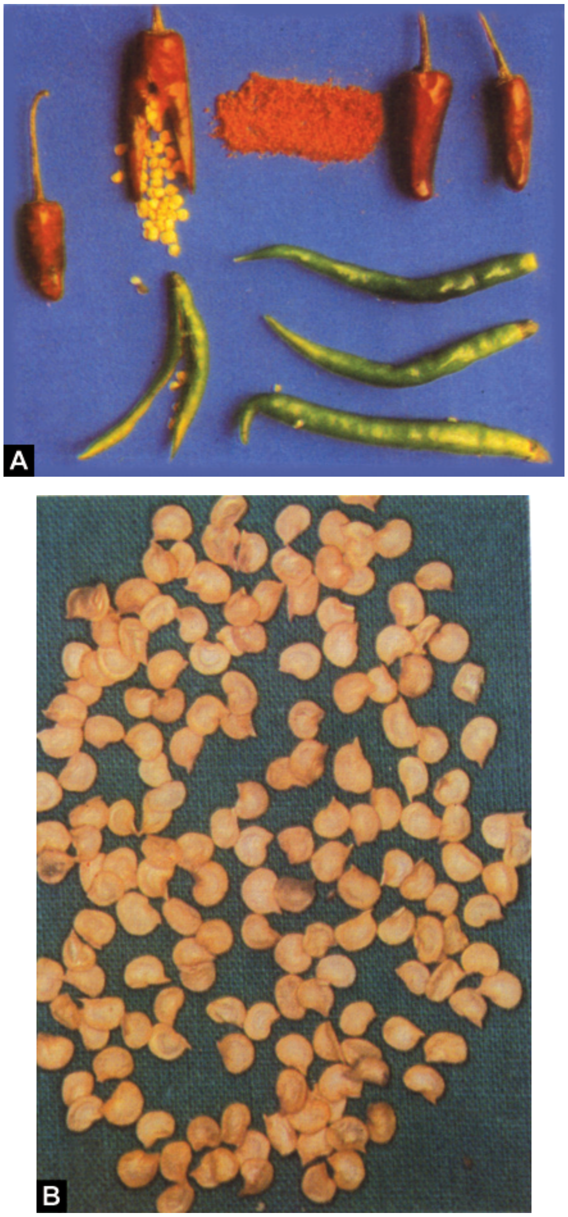


Fig. 33.10A and B: (A) Capsicum annum (Chilly). Note red and green variety intact and cut opened showing the seeds and red chilly powder; (B) Chilly seeds

Table 33.12: Differences between chilly and datura seeds

Characteristics	*Chilly*	*Datura*
Size	Small	Large
Colour	Yellowish	Brownish
Shape	Rounded	Reniform (kidney shaped)
Surface	Smooth	Pitted
Odour	Pungent	Odorless
Taste	Acrid	Bitter
Cut section	Embryo curves inwards towards the hilum	Curves outwards
Diagrammatic representation of the cut section	(Fig. 33.10C)	

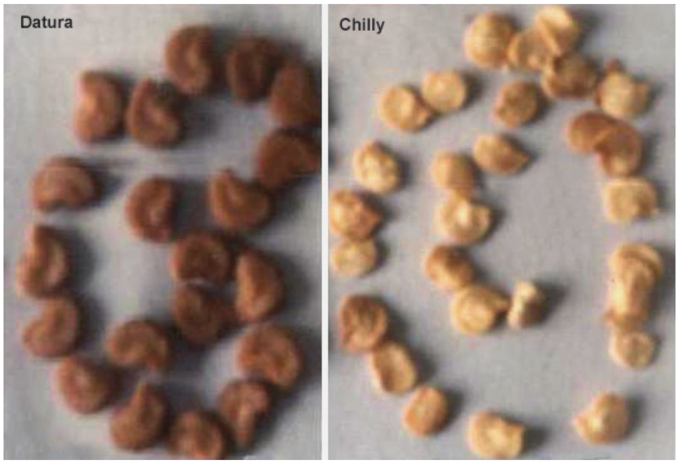


Fig. 33.10C: Dathura and chilly seeds embryo compared *(Courtesy:* Dr SC Mestri, Professor and HOD, Forensic Medicine, JSS Medical College, Mysore, Karnataka)

Orally—Large quantity can produces burning and fiery hot sensation in the mouth, salivation, excessive perspiration, abdominal pain, vomiting and diarrhea. Urine may also turn dark.

Usual Fatal Dose—Can cause serious toxicity.

Fatal Period—Fatality unlikely.

Treatment

- *In case of oral ingestion do the following:* [2,8]
  - Stomach wash with warm water
  - Blunt scraping of the tongue
  - Sucking of ice
  - Sips of ice-cold water.
- *In case of local skin contamination,*
  - Wash the area with copious amount of water.
  - According to Jones et al, affected skin may be kept immersed in *vinegar* (5% acetic acid).

Medicolegal Importance

- Other than irritation fatality due to the chilly is never reported. It constitutes one of the most common and popular condiments in Indian cooking to enhance flavour or enjoyment. Also they are used in preparation of variety of pickles and sauces.[2] As a carminative or an appetizer in a dilute form it is often used as a household remedy. In modern medicine it has been also used as a countre irritant in the form of an ointment or as an adhesive plasters to relieve muscular sprain and such other conditions.
- Dermatitis and burning of hands and fingers known as *'Hunan hand'* is common among the pickle industry workers, who use their hands for handling chilly paste or powder for prolonged period.[14]
- The chilly in the form of powder or paste has also been reported to have been used for the purpose of felonious act of extorting truth, by thrusting into the urethra, vagina, rectum, rubbing over breasts in females.[3]
- *Datura* seeds may be consumed *mistaken* for chilly seeds (Figs 33.10B and 33.11A and B), resulting in grave datura poisoning.
- Criminals often use chilly powder to facilitate act of felony, robbery, rape, etc. by putting the victim in sudden agonized or helpless condition by throwing it into their eyes prior to the criminal act.

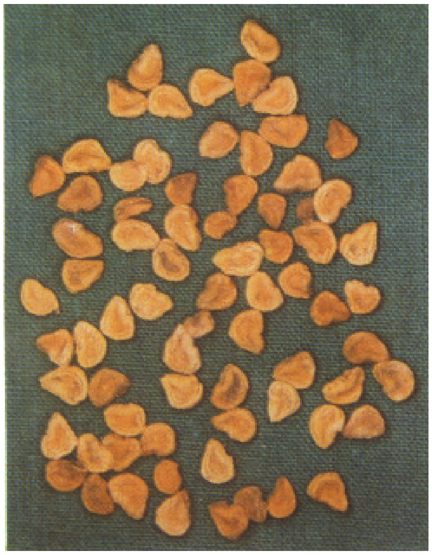


Fig. 33.11A: *Datura* seeds

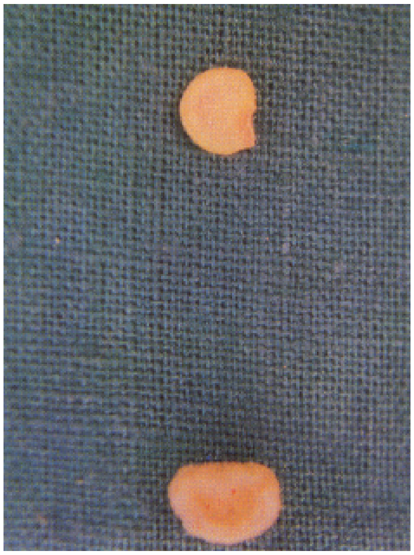


Fig. 33.11B: Capsicum (upper) seed in comparison with *Datura* seeds (lower) (Refer Table 33.12)

- Some of the interesting cases involving criminal use of chilly powder in India are narrated below:
  - *Case of Robbery With Chilly powder*— An office peon at Kolkata, after encashing a cheque for thousand rupees was passing through Dalhousie square. A man threw some chilly powder in his eyes, which blindfolded him for a moment, and flicked the cash from his pocket and while was making effort to run away, was detained and handed over to police.[3,9]
  - *Case of Prisoners Escape Using Chilly*—Five undertrial prisoners at Mumbai were being taken in a motor truck to Esplanade Police Court under police escort. One of the prisoners flicked the cap of constable on the way. Truck was stopped to enable the constable recover his cap, when, seizing the opportunity, other prisoners in the truck flung chilly powder into the eyes of their escorts, blinding them, jumped out of the truck and escaped in a car awaiting outside.[3,10,11]

EUCALYPTUS GLOBUS

Distribution—Grows in South India, especially in hills of Nilgiris, Tamil Nadu.

Common name—Eucalyptus, blue gum.

Plant characteristics—It is a tall tree with smooth bark, long curved leaves, and large flowers. Eucalyptus oil obtained by steam distillation of the extract derived from the leaves.[15]

Active principles—Eucalyptol (cineole).

Signs and Symptoms

- Burning pain in the mouth, nausea, vomiting, diarrhea, abdominal pain.
- Bronchospasm, tachypnoea, chemical pneumonitis, respiratory depression.
- Headache, vertigo, drowsiness, slurred speech, ataxia, convulsions, and coma.
- Breath and urine may smells of eucalyptus oil.

Usual fatal dose—5 to 10 ml can cause serious toxicity.

Fatal period—Fatality is unlikely.

Treatment

- Stomach wash
- Symptomatic measures as needed.

Postmortem Findings – Nothing specific

Medicolegal Importance

- Eucalyptus oil is a *house holds remedy* [15] for common ailments like common cold and pain.
- Most of the poisoning cases are due to the accidental consumption, by mistake or due to over dose. However, death due to eucalyptus oil is rare.

AZADIRACHTA INDICA

Distribution—Grows all over India. It is grown for its medicinal use.

Common name—Neem

Family—Meliaceae

Plant Characteristics—It is a tree. Seed grown in the tree yields a yellowish oil (*margosa oil*), which has a disagreeable odour and bitter taste.[15]

Active principles—The oil contains active principles enumerated below:

- Azadirachtin
- Meliantriol
- Salanmin
- Nimbin
- Nimbidin
- Unrefined oil may also contain *aflatoxins,* which are injurious to health.

Signs and Symptoms

Hepatotoxicity characterized by vomiting, dehydration, drowsiness, encephalopathy and metabolic acidosis.

Usual fatal dose—Can cause serious toxicity.

Fatal period—Fatality unlikely.

Treatment

- Rehydration
- Treat cerebral oedema
- Correct the metabolic, acidosis
- Symptomatic measures.

Postmortem Findings—Not reported

Medicolegal Importance

- For centuries neem leaves has been in use in ayurvedic medicine.[16] Neem is a medicinal plant whose oils, seeds, and leaves contain organic antibiotics, organic pesticides, and organic fungicides. Neem tree products are used in natural cosmetics and organic skin-care products. Margosa oil is used in treating skin diseases, cough, common cold, helminthiasis, etc.
- Resin tapped from the bark provides a gum commonly used as glue.
- Neem timber has been shown to be decay and insect-resistant.
- The Neem tree's wide strong branches produces excellent firewood
- Daily, millions of people brush their teeth with Neem twigs. Dentists confirm that this practice guards them against periodontal disease.
- A paste made from the leaves has been found to successfully treat skin lesions. Also small portions of leaves mixed with regular feed seem to affect intestinal parasites in livestock.
- The neem tree can produce up to 50 kg of olive-like fruit per year from which an antiseptic soap can be made.
- The seed has an active ingredient which acts as a pesticide, insecticide and even a fungicide.

COLCHICUM AUTUMNALE[17]

Common Name—Autumn crocus, meadow saffron, naked ladies

- ***Distribution:*** Eurasia, Africa.
- ***Family:*** Liliaceae
- ***Genus:*** Colchicum
- ***Species:*** Autumnale

Plant Description

Perennial herb (*Category:* Bulbs) Height: 15-30 cm, with basal, slender leaves; and long, tubular, 6-parted, flowers which are pink, violet/ lavender or white in colour.

- ***Poisonous part***—All parts of plant are highly poisonous, and may be fatal if eaten.

- ***Active principle***—Alkaloid colchicines and demecolcin.
- ***Mode of poisoning***—Oral ingestion.

Signs and Symptoms

- *Gastrointestinal system*—Presents with vomiting, diarrhoea, abdominal pain, cramping and hepatic dysfunction.
- *Cardiovascular system*—It can bring about increased blood pressure. Rarely, it can produce disseminated intravascular coagulation and bone marrow failure.
- *Respiratory system*—Rarely it can produce respiratory failure.
- *Urinary system*—It may also cause signs and symptoms of renal dysfunction.
- *Hairs*—It can produce alopecia.

Usual fatal dose—Can cause serious toxicity.

Fatal period—Fatality unlikely.

Treatment—In oral poisoning cases, perform gastric lavage. Treat with antihypertensives for increased blood pressure. Renal failure is to be treated by dialysis.

Postmortem Findings

Medicolegal Importance

Not reported.

ERGOT

Common name—Mother of rye

Characteristics

Ergot is an alkaloid. It is the sclerotium (mycelium) of a fungus *Claviceps purpura*, which grows on many cereals like *rye*, barley, wheat, oat, etc. fungus gradually replaces the whole grain to a dark purple mass, which on drying yields ergot.[2,18,19]

Active principles—are three and all are *ecbolics*, which can contract gravid human uterus in late pregnancy and they are:

- *Ergotamine*
- *Ergotoxin*
- *Ergometrine*
- They also contract arterioles which can lead to gangrene of part supplied.

Signs and Symptoms

Acute poisoning—Very rare.

GI. Tract: Irritation of throat, dryness, severe thirst, nausea and vomiting, diarrhea, pain in abdomen, tingling in hands and feet, cramps in muscles (all due to smooth muscle contraction), dizziness, feeling of coldness, etc. It might also present with symptoms of hypoglycemia, anuria, abortion and hemorrhage in a pregnant woman. Death is usually slow after a week of poisoning.

Chronic poisoning – is called *Ergotism* and is quite common. It appears in two forms:

Convulsive form—presents with painful toxic contraction of voluntary muscles followed by drowsiness, headache, giddiness, madness, etc. Victim may complain of feeling of itching/ numbness and ant crawling sensation under the skin.

Gangrenous form—Begins as pustules and swelling of limbs and feet, followed by intense hot feeling, severe pain, numbness, etc. and results ultimately into gangrenous changes (resemble Raynaud's disease). Recovery is possible, if ergot is withheld.

Fatal dose and period—both uncertain.

Toxicity rating—4 to 5.

Treatment

- Stomach wash with tannic acid and magnesium sulfate
- Amyl nitrate inhalation
- Sodium nicotinate 140 mg intravenously
- Withdrawal of ergot contaminated substance in chronic cases
- Symptomatic measures as needed.

Medicolegal Importance

- Ergotamine may be abused by patients with migraine headaches. If it is used for long period, the development of a rebound headache constitutes a major clinical problem which is alleviated only by continued use of the drug.[1]
- Used as an abortifacient, inducing *criminal* abortion.
- Accidental poisoning, usually by food contamination
- Used as medicine.

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ORGANIC ANIMAL IRRITANT POISONS

Organic animal irritant poisons include effects of bites/stings of poisonous snakes and insects. Envenomation that can occur with these bites and stings lead to toxic condition which at times be serious enough to cause even the death of the victim. The following discussion orbits around the more common of these toxicological syndrome complexes.

SNAKES AND SNAKE BITES

Snakes are ectothermic (*cold-blooded*) limbless vertebrates of the Class *Reptilia*, which also include lizards, crocodiles and alligators, tortoises, turtles, etc. There are at least 3,000 species of snakes, but only 400 are poisonous, which means most of them is non-venomous. Some snakes have evolved specialized glands, which produce venom, mostly derived from salivary glands. Venom may have several functions for the snake such as rapid *immobilisation* and *predigestion* of prey.[1-3] According to Burton, *Cobra venom* is a potential source of medicines also, including *anticancer drugs* and *painkillers.*[4]

Epidemiology of Snake Bite[2,3]

It is estimated that the true incidence of snake bite/envenomation could exceed 5 million per year. About 100,000 of these develop severe sequelae. Around 30-40 thousand people die every year due to snake bite all over the world. Around 10,000-15,000 deaths are reported in India annually.[5] The global disparity in the epidemiological data reflects variations in health reporting accuracy as well as the diversity of economic and ecological conditions. Hospital records fall far short of the actual number owing to dependence on traditional healers and practitioners of witchcraft, etc. It has been reported that in most developing countries, up to 80 per cent of individuals bitten by snakes first consult traditional practitioners before visiting a medical centre. Owing to the delay, several victims die during transit to the hospital.

Feature of Snakes in General[1-5]

- Body is elongated and covered with horny epidermal scales, which are shed/ moulted off several times a year.
- Snakes usually move on the tips of their ribs
- Eyelids are fused so appear to be absent
- There are no visible external ears, and hence there is a great controversy on whether snakes can hear sound
- Skull bones are movably articulated
- Tongue is forked at the tip and serves as a sense organ and can be protruded out even when the mouth is closed through a gap in the upper jaw.

  Jacobson's organ—The snakes have nostrils and olfactory (smell) organs connected with the smell centre of the brains, they also have an organ called *Jacobson's organ*. It is a cavity in the roof of the mouth with olfactory cells in which the snake inserts the tips of the forked tongue. The snake waves its tongue, searching for scent molecules, which it then transfers to this organ, where it is analyzed and the information is transferred to the brain.[2-4]
- They have got a paired copulatory organ and the cloacal aperture is transverse
- *Epidermal scales*[8]—Scales on the head are plate like and are called *shields*, on the back are small arranged in midline almost quadrangular and are called *vertebrals*, on the sides of the trunk are called *costals*, on ventral aspect (belly) are transversely elongated and are called *ventral shield*, on lower surface of ventrum of tail are in single/double rows and are called *subcaudals*.

Classification of Snakes

- Venomous/poisonous snakes
- Non-venomous/non-poisonous snakes.

VENOMOUS SNAKES

Based on their morphological characteristics including arrangement of scales, dentition, osteology, myology, sensory organs, etc. snakes are categorized into several families. Venomous species are usually confined to *5 families - Colubridae, Elapidae, Viperidae, Hydrophiidae,* and *Atractaspidae.*[2-4,7-12]

Colubridae

Only a few of these can cause significant injury to humans. There are also several species of Colubridae that have modified salivary glands producing toxins, but *without* true fangs (Fig. 33.12A). They rely instead on the trauma caused by other teeth to provide an entry track for their "venom". A few of these may cause some effects in humans, though are not expected to be lethal.

Elapidae

Include "*cobra*" type snakes, common in Asia and Africa. They have small to moderate sized fangs at the front of the mouth, possibly the "*true fangs*" (Fig. 33.12B). Cobra venoms are quite toxic and they are a major cause of human envenoming morbidity and mortality. Examples are *cobras, kraits, coral snakes* and *mambas.*

Viperidae

Vipers constitute this family. All species have *well-developed, longer fangs* (Fig. 33.12C) on hinged maxillae, allowing *rotation* (elevation) when biting, than in any other groups of venomous snakes. Venom glands are also typically *larger* than in other groups. They have heat-sensing pitorgans at the front of the head, giving some degree of infrared or heat sensitive "vision". Vipers are a major cause of snakebite in the Americas, Africa, Europe and Asia. Examples are *vipers, pit vipers,* and *rattle snakes.*

Hydrophiidae

These are *sea snakes* and are closely related to the *cobras*, with similar fang structure, but live most or all of their lives in an aquatic, usually *marine* environment. They are a significant cause of envenoming amongst fisherman in the Indian and Pacific oceans. Example: *Stoke's sea snake.*

Atractaspidae

These are side fanged *viper like* snakes confined to Africa and the Middle East. They have unusual fang structure and venoms, which contain *endothelin* like compounds called *sarafotoxins*, causing potent smooth muscle contraction.

The Major Families and Species of Venomous Snakes in India

There are about 52 poisonous species of snakes in India and they belong to *three* families:[1-3]

- *Elapidae*, which includes *common cobra, king cobra and krait*

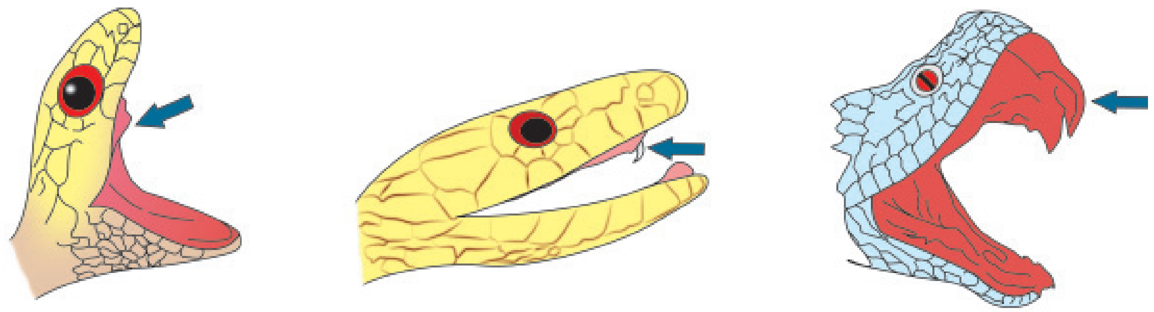


Fig. 33.12: Fangs in different snake families

- *Viperidae*, which includes *Russell's viper, pit viper and saw-scaled viper* and
- *Hydrophidae* (the sea snakes).

However, among these, majority of bites and consequent mortality in India is attributable to only five species and they include King Cobra (*Ophiophagus hannah*), Common Cobra (Naja Naja), Russell's Viper (*Daboia rusellii*), Krait (*Bungarus caeruleus*) and Saw-scaled Viper (*Echis carinatae*).

Features of Venomous and Non-venomous Snakes

Venomous snakes have certain features commonly observed in all of them and certain other features, which are unique to each of them. Table 33.13 enumerates these features.[4-6]

POISON APPARATUS

It is a modified salivary (*Parotid*) gland (Fig. 33.13A) consisting of gland, duct and fangs.

Gland—It lies just below and behind the eyes, one on either side and is somewhat almond shaped.

Duct—This arises from the gland to carry the poisonous, venom from gland to the fangs.

Fangs—These are two in number, one on either side of upper jaw (could be more than 2 and kept in reserve), are hollow hypodermic needle like. It could be grooved as in *cobra*, *krait* or could be tubular as in *vipers*.

SNAKE VENOM

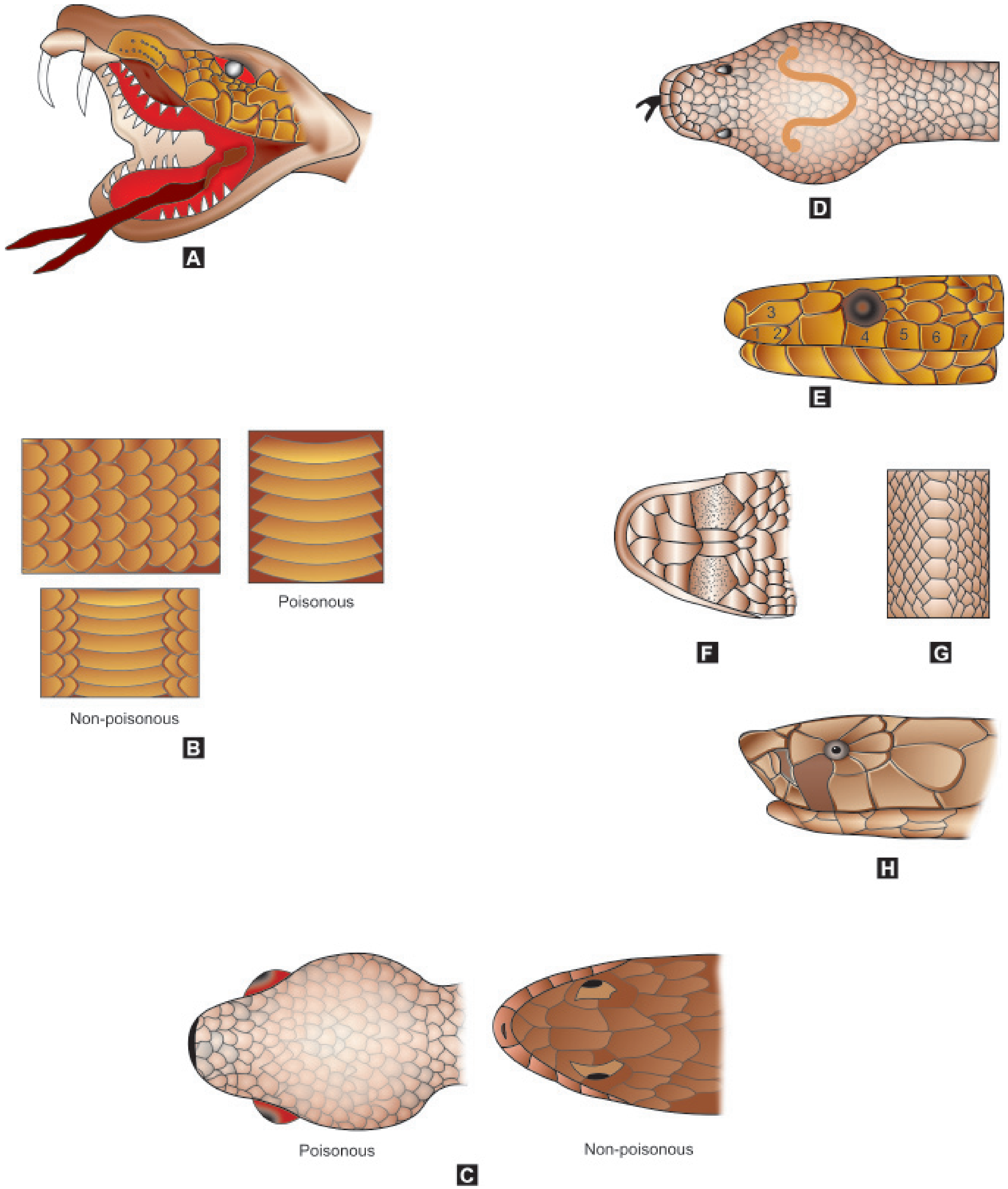
Snake venom is the poisonous secretion (*saliva*), ejected from the poison apparatus (modified *parotid gland*) of a poisonous snake, during the act of biting. Snake venoms are usually clear and amber coloured fluid when fresh. On drying, its potency will be the same as that of liquid state.[5-9] Snake venoms are poisonous only when injected by a needle or by bite of a snake.[1-3,6,8,9] However, the venom is non-poisonous when taken orally.[9]

Toxic Principles in Snake Venom

Venoms of the different species of poisonous snakes are usually a mixture of *toxic substances* or *toxins, enzymes* and *toxalbumins*

Table 33.13: Comparative account of poisonous and non-poisonous snakes

Features	*Venomous/poisonous snakes*	*Non-venomous/ non-poisonous snakes*
Poison apparatus (Fig. 33.13A)	Present and is in the salivary gland.	Absent
Saliva	Contains toxic peptides and enzymes	Non-toxic
Tail	Compressed	Rounded
Ventral (belly) scales	Broad and always extends across the entire width of the belly (Fig. 33.13B)	Small/ moderately large, never extends across the belly.
Vertebral (back) scales	Enlarged as in Krait (Fig. 33.13G)	Not so
Head scales	Usually smaller (Fig. 33.13C). It could be larger also and when larger, will possess special features such as: • A pit between eye and nose as in pit viper (Fig. 33.13H) • A third supra-labial scale touching the eye and nasal shield, as in *cobra, king cobra* (Fig. 33.13E) • A large fourth Infralabial shield, as in *krait* (Fig. 33.13F)	Usually larger and without any special features
Teeth	Upper jaw has a pair of teeth modified into fangs that are grooved (Cobra) (Fig. 33.13D) or channelised (viper)	All teeth are uniform and small in size and there are no fangs. Usually there are 4 longitudinal rows of teeth in upper jaw and 2 rows in lower jaw
Bite marks	Usually two (fang marks)	More than two
Nocturnal habit	Usually nocturnal	Not nocturnal



Figs 33.13A to H: Important features of poisonous and non-poisonous snakes: (A) Poison apparatus and fangs (canalised and grooved), (B) Ventral shields (belly scales) and vertebrals, (C) Head scales, (D) Cobra (note the hood and spectacle mark) (E) Cobra – Third supralabial touching eye and nasal shield, (F) Krait — four infralabials (note the large fourth one), (G) Krait — enlarged vertebrals on the back, and (H) Pit viper — a pit between eye and nostril

in varying proportion.[1,6-9] Table 33.14 enumerates the different types of toxic principles in the snake venom.

Types of Snake Venoms

Basically snake venoms are of three types, namely *neurotoxic, haemotoxic* and *myotoxic* venoms.

Table 33.14: Different types of toxic principles in the snake venom

Toxins	*Enzymes*	*Toxalbumin*
Low molecular Weight peptides and proteins	Cholinesterarse Anticholinesterase Hyaluronidase Lecithinase Phosphatidases Phospholipase-A Proteases Proteinases Ribonuclease	Cardiotoxin Neurotoxin Cytolysin Haemorrhagin Haemolysin Fibrinolysin Proteolysin Thromboplastin

Neurotoxic Venom

Origin—Common in Elapidae snakes, e.g. krait, cobra, etc.

Action—Acts like Curare, mainly on the *motor nerve cells* and results in *muscular paralysis*, the muscles are affected in following order:

- Firstly—Muscles of the mouth
- Secondly—Muscles of the throat
- Finally—Muscles of respiration

Symptoms at bite site—Local manifestations are least with neurotoxic venom snake bite.

Other symptoms—Convulsions may be seen with Cobra venom (Krait venom produces only paralysis).

Haemotoxic Venom

Origin—Common in Viperidae snakes, e.g. *Pit viper* (*Crotalidae*); Pit-less viper (*Russell's viper, Saw scaled viper/Phoorsa/Echis/ Echis Carinata*), and Bamboo snake (*Common green pit viper*).

Action—Acts by cytolysis of endothelium of blood vessels, lysis of red cells and other tissue cells and coagulation disorders. All these can lead to:

- Severe swelling with oozing of blood and spreading cellulitis at bite site. Blood from such patients fails to clot even on adding thrombin, because of very low level of fibrin.
- Necrosis of renal tubules, and
- Functional disturbances like convulsions, due to intracerebral haemorrhage.

Myotoxic Venom

Origin—Common in hydrophidae or sea snakes

Action—Produces generalized muscular pain, followed by:

- Myoglobinuria within 3 to 5 hours.
- Death usually occurs due to respiratory failure.

NONVENOMOUS SNAKES

Important Features

- They have no poison apparatus
- They possess 4 longitudinal rows of teeth in upper jaw and 2 rows in lower jaw
- Tail is not compressed
- Ventral shields are small/moderately large
- Head scales are usually larger and without any special features
- Fangs are short and solid
- These are not nocturnal
- The bite marks show more than two teeth markings.

Comparative accounts of venomous and non-venomous snakes in general are discussed in Table 33.13 above. Among the poisonous species, snakes belonging to family *Elapidae* and *Viperidae* would be dealt with in more detail, as they are responsible for the most of the snakebite fatality in India and other parts of the globe. Each of the colubrine and viperine snakes is discussed in detail in Table 33.16. Common features of elapid and vipenine snakes are enumereted in Table 33.15.

VENOMOUS SNAKEBITE (OPHITOXAEMIA/ENVENOMATION)

Incidence

Snakebites are usually accidental. Rarely it can also be homicidal or suicidal.

Entry of Venom on Snake Bite

Venom is inoculated into the body. Snakes like cobra can inject/ emit the venom by spitting.

Degree of Toxicity

Degree of toxicity depends on three factors:

- Toxic principle in the venom
- Quantity of venom injected
- Type of fang (see Fig. 33.12)
  - Channeled (viperine) fangs—complete transfer of venom
  - Grooved (elapid) fangs—less transfer of venom.

Signs and Symptomatology

Instantaneous death due to shock due to *fear* of snakebite is observed more often than the actual toxicity of the venom. Signs and symptoms of snakebite depend on the type of venom and are discussed under local effects (at the bite site) and its systemic effects.

1. Neurotoxic Venomous Snake Bite

Local action—Severe burning at bite site, rapid edema and inflammatory changes followed by oozing of serum.

Table 33.15: Elapid and viperine snakes—common features

Features	*Elapid snakes*	*Viperine snakes*
Body	Long and cylindrical	Fat and short
Head and neck	Same size and covered by large scales having special features	Head is triangular and wider than neck and usually covered with smaller scales having no special features
Pupils	Rounded	Vertical
Fangs	Situated anteriorly, grooved, short and fine. It cannot bite through the clothing.	Situated posteriorly, are canalized, long and strong and can bite through the clothing
Venom	Neurotoxic	Hemotoxic
Young ones	Hatch out from the eggs (*oviparous*)	Born (*viviparous*)
Examples	*Cobra, King cobra, Common krait, Banded krait*	*Pit viper, Russel's viper* and *Saw scaled viper*

Table 33.16: Elapid and viperine snakes—specific features

ELAPID SNAKES			*VIPERINE SNAKES*		
Types	*Features*		*Types*	*Features*	
Cobra *(Common cobra, Naja naja)* (Figs 33.14A and B, and Figs 33.13D and E).	*Length*	1.5 to 2 m	***Pit Viper*** (Fig. 33.13H)	*Length*	30-100 cm
	Colour	Black/yellow/brown/green		*Colour*	Vivid green/ yellow/ brown
	Head	—	***Habitat*** Hilly regions, throughout India	*Head*	Triangular with a pit between eye and nostrils
	Neck	Expandable on enragement called *hood*, bears a '*spectacle*' or a '*monocle*'		Neck	—
Habitat -Usually in thickly populated areas, throughout India	*Body*	Long and cylindrical		*Body*	Fat and broad
	Flanks	-		*Flanks*	Has yellowish white lines
	Tail	Has divided shields		*Tail*	Has divided scales.
King Cobra *(Ophiophagus hannah)*	*Length*	2.5 to 4.5 metres.	***Russel's Viper*** *(Vipera russelli)*	*Length*	1.5 m
	Colour	Young one, jet black; Adults, Yellow/ green/ black with white/ yellow cross bars on the body.	Makes terrific hissing noise when it is about to bite.	*Colour*	Brown/buff
Habitat - Thick jungles/ forests	*Head*	—	***Habitat*** - Throughout India	*Head*	Flat and triangular with a distinct "V" mark and small scales. *Nostrils* - are big
	Neck	Hood without spectacle mark		Neck	—
	Body	—		*Body*	Stout with 3 rows of diamond shaped chains (Figs 33.16A and B). *Belly* - Is white and comprised of broad plates.
	Flanks	—		*Flanks*	—
	Tail	Has entire shield		*Tail*	Narrow, short with divided shields
Common Krait *(Bungarus caeruleus)**	*Length*	1.25 to 1.50 m (Figs 33.13F and G)	***Saw Scaled Viper*** *(Echis carinata)*	*Length*	50 to 100 cm.
	Colour	Glistening black	Makes *rustling noise* while moving due to the rough scales	*Colour*	Brown/brownish grey/green
	Head	Covered with large shields		*Head*	Triangular + white Bird's footprint/ arrow like mark
	Neck	—		*Neck*	—
	Body	Shows single/double arches with central row of hexagonal scales and a creamy white *belly*		*Body*	Has serrated scales like a saw on the back and broad plates on the belly. *Special features*
	Flanks	—		*Flanks*	Has a wavy line and diamond shaped marks between upper curves of wavy line.
	Tail	Covered with larger shields		*Tail*	Have undivided shields
	Habitat	Very close to human dwelling		*Habitat*	Throughout India

**Banded Krait (Bungarus fasciatus)* *is about 2 m long, resembles common krait, except that it has alternate black and yellowish bands across the back (Fig. 33.15) and its habitat is Assam, Bengal and South India*

Systemic action—Found within 15-30 minutes or 2 hours of biting. Flow chart below presents the neurotoxic effects of colubrine snakebite. Giddiness, weakness, lethargy, muscle weakness etc, are followed by:

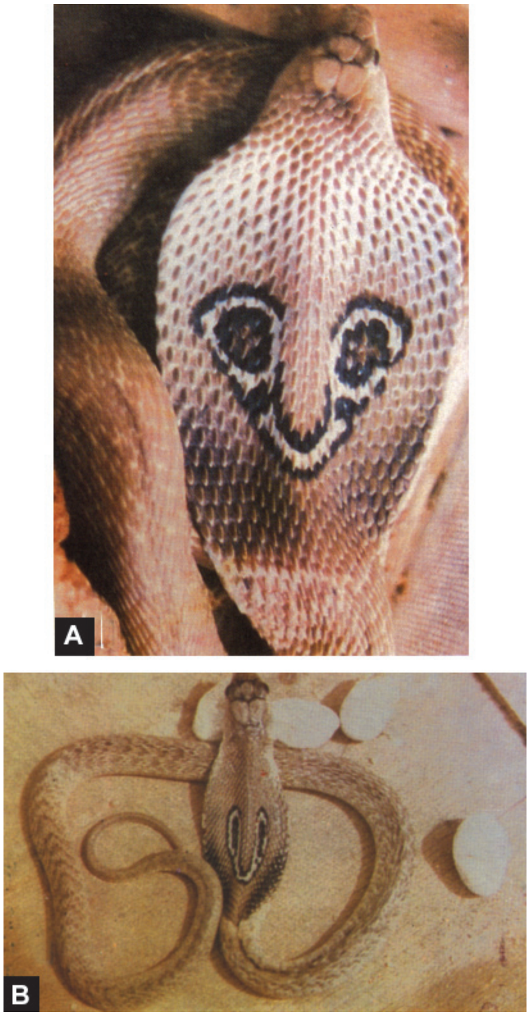
A spreading paralytic symptom
↓
Coma
↓
Bulbar or central paralysis
↓
Respiratory paralysis
↓
Death

However, recovery is *complete* from paralysis if patient survives, e.g. cobra bite.

2. Haemotoxic Venomous Snake Bite

Local actions—Severe pain at bite site, followed by swelling, ecchymosis, cellulitis and severe haemorrhage (Figs 33.17A and B).

Systemic action—It is due to hemolytic effect on heart and blood vessels resulting in cardiovascular collapse and death. If the patient survives suppuration, sloughing with infection at the site of bite, haemorrhage from the mucosa of rectum, other natural orifice, etc. and gangrene of the parts involved can occur.



Figs 33.14A and B: Poisonous snakes: (A) Cobra with expanded hood showing spectacle mark, (B) Cobra with its eggs (note—oviparous nature)

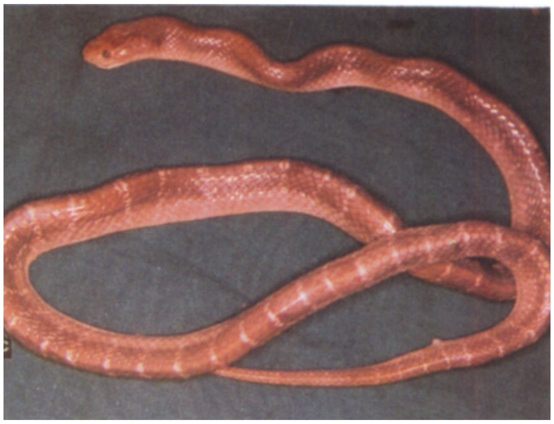


Fig. 33.15: Poisonous snakes: Banded krait

3. Myotoxic Venomous Snake Bite

Local actions—Minimal swelling and pain

Systemic action—Myalgia, muscle stiffness, myoglobinuria, renal tubular necrosis.

Table 33.17 summarises the symptomatology of snakebite briefly. Snake venoms and fatal dose of venom, amount of venom injected at a bite site and fatal period of venom are presented in tabular form in Tables 33.18 to 33.20 respectively.

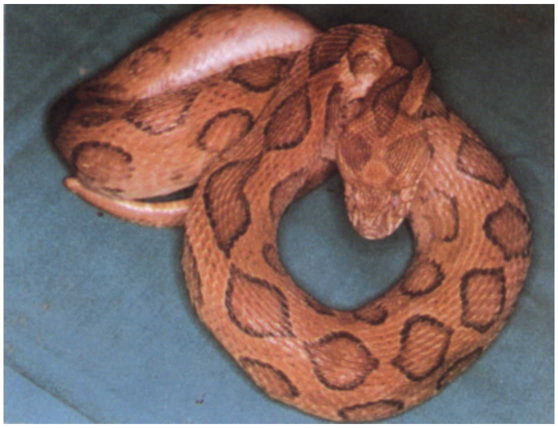


Fig. 33.16A: Poisonous snakes: Russel's viper (note—triangular head with distinct 'V', narrow neck and stout body with three rows of diamond shaped chain

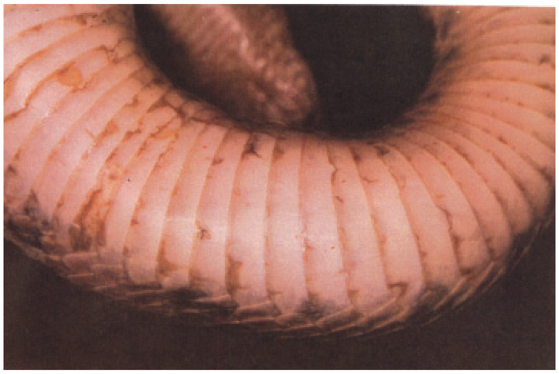


Fig. 33.16B: Poisonous snakes: Russel's viper—wide belly scale

TREATMENT OF SNAKE BITE

Principle[8,9]

1. To allay the anxiety and fear
2. Prevention of the spread of venom (first aid measures)
3. Antivenom treatment
4. General measures

1. Allaying of the Anxiety and Fear

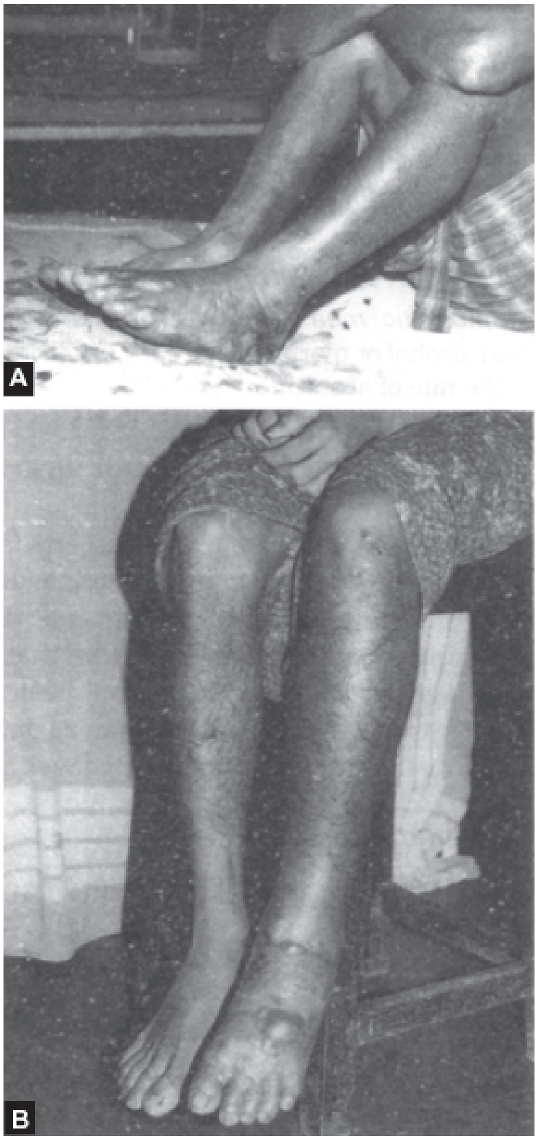
Convince and reassure the patient that all snakes are not poisonous, and even if poisonous need not be fully charged with poison, even if fully charged, the quantity that it has injected at the bite site, need not be lethal to kill.

2. Prevention of the Spread of Venom

The spread of venom is usually by lymphatic. Following first aid may be useful.

First Aid

- Reassurance of the victim.
- Do not tamper with the bite wound, except wipe with a damp cloth to remove the venom lying on the skin surface.
- *Immobilisation* of the bitten limb.
- Transport the patient to a medical facility immediately.
- Identify the snake if possible but not necessary. Better to take along the dead snake for identification. Be sure it is



Figs 33.17A and B: Poisonous snakes: (A) Viperine snake bite of foot (Note—bitemark on outer aspect of the ankle with, cellulitis bleeding, and (B) Viperine snake bite—diffuse cellulitis of entire left leg (*Courtesy:* Dr Rajaram Nayak, Former Tutor Forensic Medicine, KMC, Mangalore)

dead. Severed snake heads, both fresh and preserved, have inflicted severe and even fatal bites.[13]

- Avoid potentially harmful traditional first aid measures such as cauterisation, incision, excision, or amputation of bite site; suction by mouth, vacuum pump, or syringe; combined incision and suction by 'Venomex' apparatus; injection or instillation of compounds such as potassium permanganate, phenol (carbolic soap), and trypsin; application of ice (cryotherapy) or electric shocks; herbal, folk, and ayurvedic remedies such as emetic plant products and parts of snake; multiple incisions and tattooing; insufflations of oily substances into the trachea; and application of irritants in conjunctivae, etc.[13,14]
- Do not apply tourniquets, ligatures, or constricting bands unless the snake is a neurotoxic envenomating, *i.e. snakes of species: elapids—Indian Cobras, Kraits; Australian elapids (genera: Acanthopis, Micropechis, Oxyuranus, Pseudechis, and Pseudonaja) and sea snakes,*[14] However avoid this for long periods because local area will develop depletion of fresh blood. Similarly opening of the tourniquet will cause the rush of blood to the site and hence the rapid spread of venom, therefore, the antivenom treatment should be applied before releasing the tourniquet.

Dangers of Tourniquets, Compression Bandages and Other Occlusive Methods[13]

- Ischaemia and gangrene
- Damage to superficial peripheral nerves, especially the lateral poplitial (common peronial) nerve at the neck of fibula.
- Increased fibrinolytic activity in the occluded limb.
- Congestion, swelling, and increased bleeding from the occluded limb.
- Shock on releasing a tight tourniquet.
- Intensification of local effects of venom in the occluded limb.

A firmly applied crepe bandages exerting a compression of approximately 55 mm Hg may be used after bites by neurotoxic elapids and sea snakes and may left in place for several hours. Tourniquets tight enough to obliterate the arterial pulse are painful and must be released for about one minute after an hour. If reapplied they can finally be removed after two hours in a hospital or dispensary after an intravenous infusion of antivenom is begun and drugs and resuscitation equipment is ready for immediate use.

Table 33.17: Symptoms of common Indian snake bites

Cobra and krait bite	*Viper bite*
• In case of *Cobra* and *Krait* poisoning, ***constitutional symptoms*** are more prominent than local pain and swelling. • General intoxication is soon followed by a *sense of creeping paralysis* beginning in the legs and ascending to the head by way of trunk • Paralysis of the muscles of the eyelids, staggering gait, in-coordination of speech, paralysis of the limbs, drooping of the head and complete paralysis of all voluntary muscles develop • Nausea and vomiting frequently occur • Breathing gets more and more difficult and finally stops • In the case of *Krait* poisoning, in addition there are convulsions and violent abdominal pains due to *internal hemorrhages* • The venoms of *Cobra* and *Krait* act very rapidly if a large amount of venom is absorbed into circulation	• In case of *Russell's* and *Saw-scaled Viper* poisoning, the ***local symptoms*** are prominent and severe • There is great and persistent pain and intensive swelling at the site of the bite • The venom of the *viper* contains several enzymes, which may possibly act synergistically to produce shock, consumption coagulopathy, spontaneous hemorrhages in the organs and tissues, acute necrosis and death • There is constant and incessant oozing of blood from the punctures • Sloughing occurs permitting other infections • ***Constitutional symptoms*** are characterized by hemorrhages; both external and internal hemorrhages in the abdomen are responsible for pain, tenderness and vomiting • *Death* is due to heart failure, there is no paralysis

Table 33.18: Venoms and fatal doses of venom

Venoms	*Fatal doses*
Cobra	15 mg (dried form)
Viper	40 mg (dried form)
Krait	06 mg (dried form)
Echis carinata	08 mg (dried form)

Table 33.19: Venoms and amounts of venom injected at a bite site

Venoms	*Amounts of injected at a bite*
Cobra	200-350 mg (dried form)
Viper	150-200 mg (dried form)
Krait	22 mg (dried form)
Echis carinata	4.5 mg (dried form)

Table 33.20: Venoms and fatal periods of venoms

Venoms	*Fatal periods*
Cobra	Few minutes to hours
Viper	Few hours to days

However, sudden death due to fright and shock is usual

3. Antivenom Treatment[13]

Do not use antivenom treatment routinely and indiscriminately because of reasons:

- All commercial antivenoms carry a risk of potentially serous serum reaction.
- Antivenom is not always necessary; many patients are bitten by nonvenomous snakes, and a large portion of those bitten by venomous snakes are not envenomated.
- Antivenoms have a range of specific and paraspecific neutralizing activity and are useless for venoms outside that range. Specific antivenoms are not available for treatment of envenomation by some species (e.g. *Bungarus candidus* in Southeast Asia)
- Antivenom is expensive, always in short supply, and has a limited shelf life.
- Some of the antivenoms available in India are discussed below.

Indications for Antivenom

A. Systemic envenomation
  1. Haemostatic disturbances; spontaneous systemic bleeding (*e.g. gums, epistaxis*), coagulopathy (*e.g. incoagulable blood, prolonged clotting time,* thrombocytopenia, etc.)
  2. Cardiovascular abnormalities: shock, hypotension, abnormal electrocardiogram, arrhythmia, cardiac failure, pulmonary oedema.
  3. Neurotoxicity.
  4. Generalized rhabdomyolysis.
  5. Impaired consciousness of any cause.
  6. in patients with definite signs of *local envenomation*, the following indicate significant systemic envenomation:
     a. Neutrophil leucocytosis
     b. Elevated creatine phoshokinase and aminotransferases,
     c. Haemoconcentration, uraemia, hypercreatininaemia, oliguria, hypoxaemia, acidosis and vomiting.

B. Severe local envenoming

Local swelling involving more than half of the bitten limb, or associated with extensive blistering or bruising, especially in patients bitten by species whose venoms are known to cause local neurosis (*e.g. viperidae, cobras*). Bites on digits carry a high risk of necrosis.

Contraindications to Antivenom[13]

There is no absolute contraindication to antivenom in patients with life-threatening systemic envenomation. However, patients with an atopic history (asthma, hay fever, vernal conjunctivitis, eczema, and food and drug allergies) and those who had reaction to equine antiserum on previous occasions have an increased risk of severe reactions.

In case of pretreatment with subcutaneous adrenaline and intravenous antihistamine and corticosteroids may prevent or diminish the reaction.

Rapid desensitisation is not recommended.

Timing of Antivenom

Give as soon as signs of systemic or severe local envenomation are evidenced. Average time between bite and death:[13]

- *Cobras* – 8 hours (12 minutes to 120 minutes)
- *Bungarus caeruleus* – 18 hours (3 to 63 hours)
- *Vipera russelli* – 3 days (5 minutes to 264 hours)
- *Echis carinatus* – 5 days (25 hours to 41 days)

It is almost never too late to try antivenom treatment: it has been effective up to 2 days after sea snake bites and 10 days or more after *Echis carinatus* bites.

Antivenom Specificity

Optimal treatment consists of *monospecific/monovalent antivenom.* If no dead snake is brought in for identification, polyspecific/*polyvalent antivenom* may be useful.

Administration

Preferably give antivenom intravenously: 5 ml/minute, or dilute in isotonic fluid, infused over 30 to 60 minutes. Dress venipuncture sites with a pressure bandage. Injection of antivenom into the fang marks is probably ineffective and painful.[13]

Dosage

Children must be given the same dose of antivenom as adults.

Response to Antivenom: Time to Possible Response

- Neurotoxicity – slowly
- Cardiovascular effects –(hypotension, bradycardia) – 10 to 20 minutes
- Stopping spontaneous systemic bleeding – 15 to 30 minutes
- Blood coagulability restored – 1 to 6 hours
- Repeat the initial dose of antivenom if severe cardiovascular or neurotoxic symptoms persist for more than 30 minutes and incoagulable blood persists for more than 6 hours after first dose.

Antivenom Reactions

1. *Early reactions*: 10 to 60 minutes after starting intravenous antivenom, cough, tachycardia, itching (especially of scalp), urticaria, fever, palpitation, nausea, vomiting, headache, etc. may develop. Over 5 per cent with early reactions develop manifestations of severe systemic anaphylaxis: hypotension, bronchospasm, angioedema, etc. and a few may die.

Treatment: Adrenaline (epinephrine) subcutaneously: 0.5 to 1.0 ml 0.1 per cent (1 in 1000) for adults; 0.01 mg/kg for children. In severe cases, give same dose by intramuscular injections, or during cardiac resuscitation, by slow IV or even intracardiac injection. Follow with an antihistamine, e.g. chlopheniramine maleate – 10 mg (adults); 0.2 mg/kg (children).

2. *Pyrogenic reactions*: develop in 1 to 2 hours after treatment and include—chills, cutaneous vasoconstriction, goose flesh, shivering, drop in temperature, sweating, vomiting, diarrhoea, etc.

   Treatment: Lie flat; reduce temperature by fanning, tepid sponging, hypothermia blankets, or antipyretic drugs such as acetaminophen (5 mg/kg by mouth, suppository, or via nasogastric tube).

3. *Late reactions (serum sickness type)*: About 7 days after treatment (5 to 24 hours).

   Treatment: Antihistamines may restrain a milder attack. Steroids may be useful in more severe cases.

Ancillary Treatment

Local Envenomation

1. *Secondary infection:* Prevention with penicillin or erythromycin and booster dose of tetanus toxoid.
2. Clean wound with antiseptic.
3. Bullae can be aspirated to dryness with a fine sterile needle.
4. Nurse limbs in most comfortable position.
5. Examine the wound frequently for evidence of necrosis.

Polyspecific/Polyvalent Antivenom

- *Haffkin's Institute, Mumbai and Central Research Institute Kasauli, Himachal Pradesh, King's Institute, Chennai, Serum Institute, Pune* prepares this. It is available as a *lyophilized powder* in an ampoule, with potency for nearly 10 years.
- It can neutralize the venom of *cobra*, *common krait*, *Russel's viper* and *Echis carinata*.
- It is effective when given within *four* hours of biting.
- It is to be dissolved in distilled water or normal saline before use.
- Use only if the solution prepared is *clear*, if it is opaque, it is considered as not potent.

Mode of Administration

Always perform *serum sensitivity test* (*test dose*) before giving it.

- ***The skin test procedure***—Inject 0.02 to 0.03 ml of the antivenin in 1:10 dilution, intradermally. If there is a hypersensitivity reaction (urticarial wheal with erythema within 15 minutes) the patient should be *desensitized*.
- ***The desensitisation procedure***—Inject 0.1, 0.2 and 0.5 ml of the antivenin in 1:100 dilutions at an interval of 15 minutes. Subsequently a 1:10 dilution is given in the same manner, followed by undiluted antivenin. If no severe reaction occurs, the usual dose is given intravenously.
- ***According to another view*** —Skin testing is *not* necessary, but *adrenaline* should be injected subcutaneously as premedication with a dose of a systemic corticosteroid.[13]

Dose Schedule

Inject 40 to 60 ml as *follows:*[8]

- One third of total dose is given subcutaneously at bite site
- Next one-third is given intramuscularly, and
- Final one-third is given intravenously.
- This schedule can be repeated every sixth hour till the symptoms disappears.
- *According to another view*—antivenom serum is to be given intravenously at the rate of 15 drops per minute. After finishing the first dose, which is completed in an hour, one may increase the rate. If the patient's response is not favourable, further infusion may be given.

Specific Antivenom Serum

- In India, *only polyvalent antivenin* is available, which is effective against common cobra, common krait, Russell's viper and sea-scaled viper.
- Is preferred when identity of biting snake is known
- Dosage and schedule of administration is same as for polyvalent antisnake venom serum.
- *Adrenaline* may be useful in paralytical cases.
- *Artificial respiration* is often required
- *Cortisone* 50 to 100 mg intramuscularly can combat the shock
- *Antihistamines* may also be helpful
- Table 33.21 provides the summary of general guidelines of specific serum treatment for a venomous snakebite in a hospital.

General Measures

Symptomatic measures as required, but, avoid *alcohol* or *morphine*, for these can increase the rate of absorption of venom. However, pethidine may be used instead of morphine.

Postmortem Findings

Bite mark—Two in number, 1 cm deep for colubrine bite and 2.5 cm deep for viperine bite. At the bite site there may be a little oedema, cellulitis, bleeding, (Figs 33.18A and B) etc for viperine bites.

- For colubrine bite deaths, changes will be that of *asphyxia*.
- For viperine bite deaths, changes will be that of haemorrhage in lungs, pleura and pericardium. Kidneys will show renal tubular necrosis, desquamation and cloudy swelling.

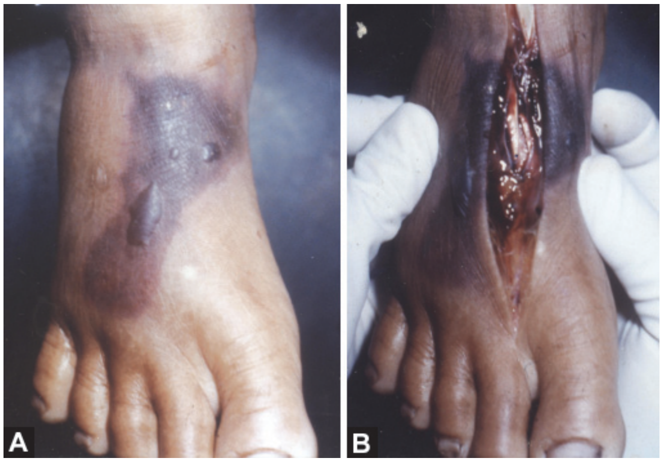
Medicolegal Aspects

- Snakebites are usually accidental, in warm and moist climates.[6] Rarely bites could be suicidal as well (Cleopetra, Queen of Egypt, committed suicide by snake bite).[8] However, homicidal cases though rare, where death is alleged to be from snake bite, the presence of snake venom has to be established.[9]
- *Cattle poisoning* To obtain the hide from cattle, the skin workers in India adopted a very peculiar method, wherein, a cobra and a ripe banana were placed in an earthen pot and the pot was warmed up to enrage the cobra which would then bite the banana. The cobra was then released; banana was then smeared on a rag and thrust into the rectum of the animal by a bamboo stick, leading to the death of the animal.[8]
- To identify the venom at the bite site the following steps may be considered:[8]
  - This is done by injecting the washings into the fowls or rabbits with specific antivenom sera injected previously
  - Detection of cholinesterase activity- *colubrine* snake bite is confirmed
  - Detection of thromboplastin activity- *viperine* snakebite is confirmed.

Table 33.21: General guidelines of specific serum treatment in the hospital

1. Installation of the intravenous drip infusion solution of physiological saline with or without vasopressor drugs until hypotension is overcome.
2. Sensitivity test to horse serum.
3. Alleviation of pain with small amount of pethidine but not morphine.
4. Intravenous administration of polyvalent antivenom serum in adequate amount.
5. Infiltration of the site of bite with a small amount of the antiserum
6. Tetanus toxoid if required.
7. Monitoring of blood pressure, blood count, coagulation, and observation of oedema.
8. Blood transfusion, if anaemia develops.
9. Surgery if necrotic area appears.
10. For detailed treatment, the hospital doctors may decide on the individual case to case basis.

Note: The specific treatment, may vary from patient to patient, and hence a medical doctor should be consulted in all cases of snakebites



Figs 33.18A and B: **Autopsy in a case viperine snake bite site:** (A) Note the oedema, cellulitis and ecchymosis. (B) Incising the area confirms the same

INSECTS

Cantharides *(Spanish Fly, Blister Beetle, Lytta)*

It is a winged insect, which has a body of: length 2 cm and breadth 0.6 cm and greenish black colour with shiny wings of same colour (Fig. 33.19). The insect as such or the powder of dried body has the toxic (active) principle.

Active principle: Cantharidin.

Route of absorption: Skin and all other muscosa.

Signs and Symptoms

Within 2 to 3 hours coming in contact with the skin, the poison produces burning pain, red-ness and vesication. Taken orally, symptoms manifest within 30 minutes to 2 hours as depicted in (Fig. 33.20).

Fatal dose—15 to 30 mg of cantharidin 1.5 gm of powdered cantharides.

Toxicity rating—For cantharidin 6.

Treatment Stomach wash, demulcents, symptomatic.

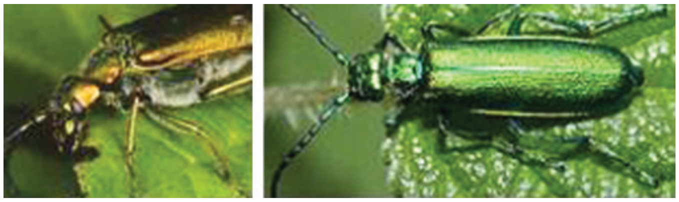


Fig. 33.19: Cantharides/Spanish fly/blister beetle

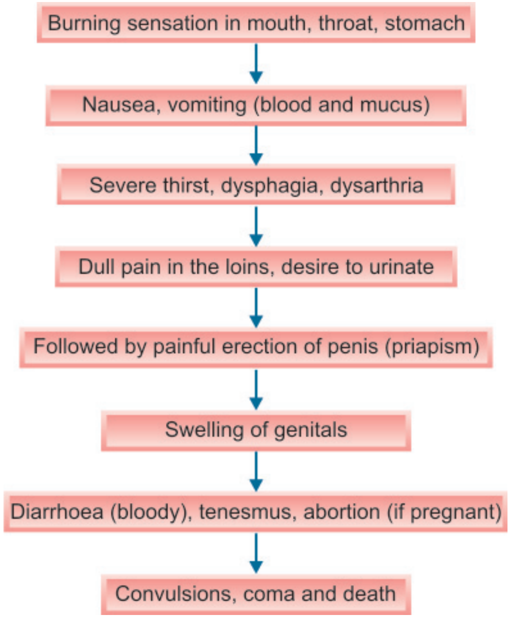

Burning sensation in mouth, throat, stomach
Nausea, vomiting (blood and mucus)
Severe thirst, dysphagia, dysarthria
Dull pain in the loins, desire to urinate
Followed by painful erection of penis (priapism)
Swelling of genitals
Diarrhoea (bloody), tenesmus, abortion (if pregnant)
Convulsions, coma and death

Fig. 33.20: Cantharides poisoning symptoms

Postmortem Findings

- Mouth, stomach and intestines may show inflammation and vesication. Particles of insect may be found in the stomach contents.
- Heart, lungs, kidneys are also found to be inflamed and haemorrhagic.

Medicolegal Importance

- Used often as an aphrodisiac
- Poisoning is reported very rarely (accidental/homicidal)
- Used also as stimulant for scalp hair growth (hair oil).

CENTIPEDES

Centipedes belong to Myriapoda (under class Arthropods) and are organic animal irritants. They have a long segmented dark to brownish black colored body with a pairs of legs in each segment (Fig. 33.21).

Signs and Symptoms

Usually centipedes can inflict painful bites with erythema, oedema, and local lymphangitis.

Treatment is by washing the bite site with soap and water and administering analgesics.

SCORPIONS

Scorpion is a poisonous insect (Arthropod) with a crab like body with eight legs and a segmented tail having a bulbous expansion and a sting in the last segment (Fig. 33.22) which has a clear,

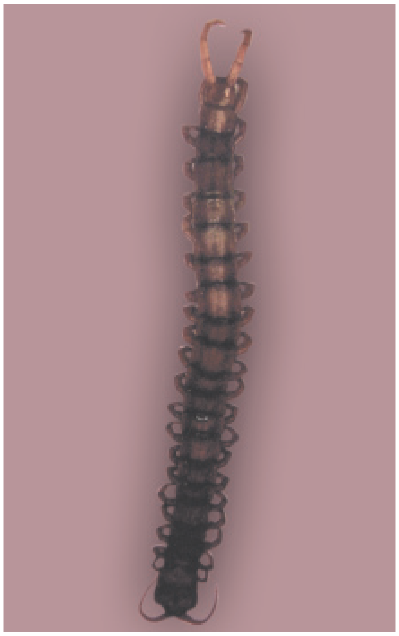


Fig. 33.21: Poisonous insect: centiped

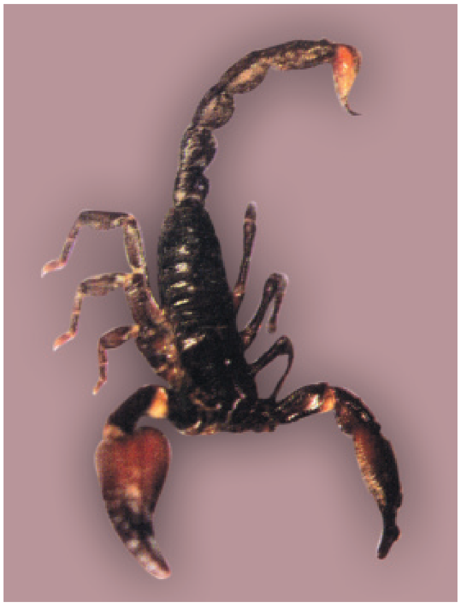


Fig. 33.22: Poisons insects: scorpion

colourless *venom* (toxalbumin) having two components, a *hemolytic* and a *neurotoxic* fractions. Fatality is rare as dose in sting is not lethal.

Signs and Symptoms

- Haemolytic factors can mimic viperine snakebite. Diagnosis is by locating only one deep punctured wound (snake bite—fang marks are always two in such wound) with red surrounding area (inflammation), oedema, severe burning pain, etc.
- Neurotoxic factor can mimic strychnine poisoning. Victim presents with nausea, vomiting, restlessness, fever followed by convulsions, paralysis, coma and death (due to respiratory paralysis).

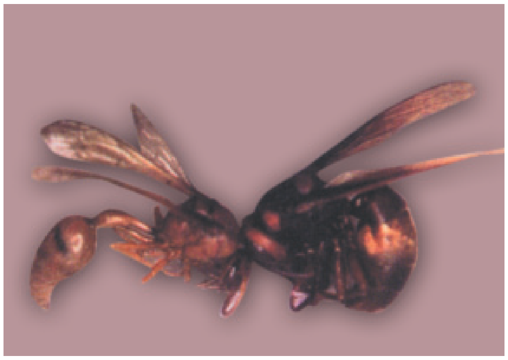


Fig. 33.23: Poisonous insect: wasp

Treatment

- Measures to reduce the rate of absorption of the venom by:
  - Tourniquet above the level of stinging
  - Ice packing, incision and suction (first aid)
  - Wash with solution of ammonia.
- Local anaesthetics can also be helpful to reduce the pain
- Intravenous administration of calcium gluconate may reduce swelling.
- Giving barbiturate can alleviate anxiety
- Atropine may be given to reduce the pulmonary edema (do not use morphine).

BEES, WASPS, HORNETS, ANT

Venom of bees, wasps (Fig. 33.23), hornets, ants, etc. is a complex mixture of biomedical compounds ranging from simple amines to complicated proteins or enzymes.

Action

Action is usually local. It may be rarely fatal if venom is histamine (especially when bites on neck, face, etc.). However, it can result in laryngeal edema leading to asphyxia and deat, when not treated immediately. When there are multiple stings, it can lead to severe systemic reactions, resulting in gastrointestinal disturbances, shock, unconsciousness and death.

Treatment

- Measures to reduce the rate of absorption of venom by:
  - Tourniquet above the level of stinging or incision and suction
  - Adrenaline is also useful.
- Apply tincture iodine/antihistaminic (to reduce inflammation)
- Adrenocorticotrophic hormone (ACTH) 25 mg in 1000 ml normal saline given as an intravenous drip can help in prevention of severe allergic reactions
- Calcium gluconate given intravenously can reduce edema/rash.

MECHANICAL IRRITANT POISONS

This poison includes glass powder, hair, diamond dust, etc. (Fig. 33.24). All these are nonpoisonous materials, but they may be labeled as poison as they can act as an irritant on gastrointestinal tract mucosa by mechanical action leading to death.

Powdered Glass

Symptoms

Burning in the stomach, nausea, vomiting with blood stains, sense of apprehension, tenderness all over the intestines (when the sharp edges come across them), may lead to death.

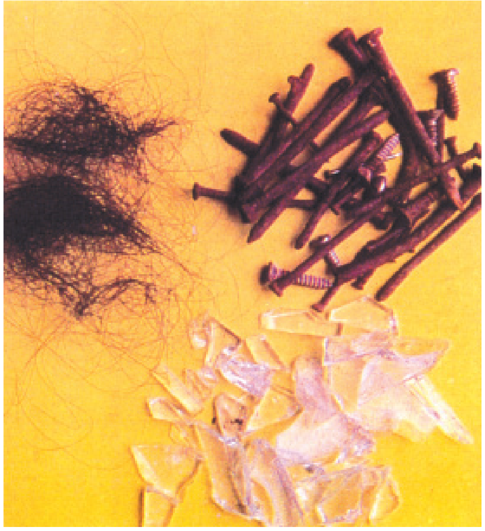


Fig. 33.24: Mechanical irritant poisons (hairs, nails and glass pieces)

Diagnosis

Radiograph—Glass shadow seen clearly.

Treatment

Bulky food like ripe banana, rice, custard, etc.

Medicolegal Importance

Accidental usually. Could be for homicidal or suicidal purposes.

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34

Chapter

Neurotoxics

Neurotoxics are the poisons acting on the nervous system. Depending on site of action on the nervous system neurotoxics are classified into three groups: *cerebral, spinal* and *peripheral neurotoxics*.[1-5]

CEREBRAL NEUROTOXICS

Somniferous Poisons (Narcotics)

Somniferous poisons are the ones, which produce *analgesia and sleep*, and are often used therapeutically, e.g. *opium* and its *derivatives*, *pethidine*, etc.

Inebriant Poisons

These are poisons which produce excitement and narcosis:

- **Classical inebriants—**Alcohols (*ethyl alcohol, methyl alcohol, ethylene glycol*, etc.)
- **Anaesthetics—***Chloroform, ether*, etc.
- **Sedatives and hypnotic poisons—***Chloral hydrate, barbiturates*, etc.
- **Benzodiazepines—***diazepam, flurazepam*, etc..
- **Hydrocarbons—**Aliphatic (e.g. *diesel oil, petrol, kerosene*, etc.), aromatic (e.g. *benzene*) and halogenated (e.g. *carbon tetrachloride*)
- **Insecticides—***Organophosphorous compounds, Carbamates and Organochloro compounds.*

Deliriant Poisons

Deliriant poisons are poisons which produce well marked deliriant or confusional stage, e.g. *Datura, Belladonna, Hyoscyamus, Cannabis, Cocaine*, etc.

SPINAL NEUROTOXICS

Spinal neurotoxics are poisons, which act especially on the spinal cord, e.g. *Nux-vomica, and its alkaloids, gelsemium.*

PERIPHERAL NEUROTOXICS

Peripheral neurotoxics are poisons, which act especially on the end plates of the motor nerve terminals, e.g. *hemlock, curare, conium*, etc.

OPIUM (*AFIM*) DERIVATIVES

Opium is a grey mass with bitter taste, obtained on drying the milky latex of unripe seed capsule of poppy plant, *Papaver somniferum*.[1-5] The opium is usually collected after all the flower petals have fallen off from the capsule, by making slits along its circumference, allowing the milky latex to ooze out and harden. After the plastic gummy opium is removed, it can be refined into heroin, morphine, and codeine.[6]

Classification

Somniferous (meaning inducing *sleep*) type of cerebral poison.

Distribution

Worldwide. 70 to 80 per cent of worldwide need of opium for therapeutic purposes is supplied by India. Opium in India is cultivated in UP, Rajasthan and MP by the government license and the government purchases the whole of the produces. Clandestine cultivation of poppy is very much rampant lately in India, and Nepal. Other countries namely Myanmar, Thailand, and Laos, which form the *Golden Triangle* and Afghanistan, Pakistan, and Iran forming the *Golden Crescent* also, undertake such illegal cultivation of opium and manufacture of heroin.[1,2]

Botanical Name

Papaver somniferum.

Common Name

White poppy plant, opium (*afim*) plant.

Plant Description[1,6]

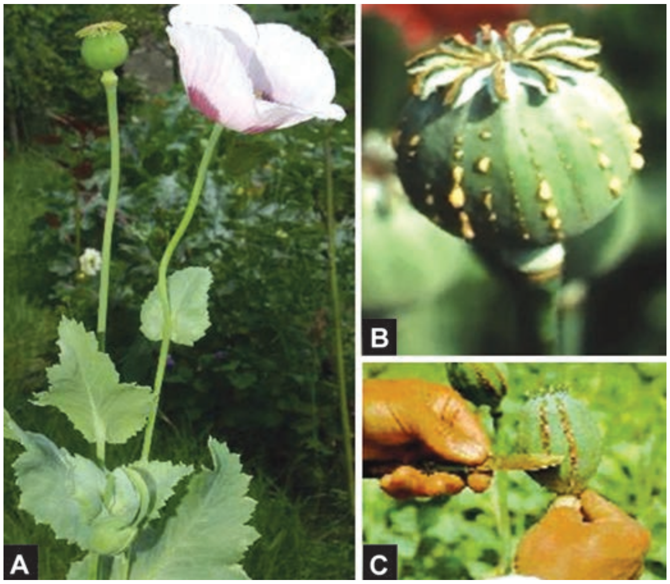
It is an herb growing up to 1 meter in height. The leaves are oblong, irregularly toothed, and slightly lobed. Flowers are large and may be bluish white, purple, or white in colour. Each plant bears 5-10 capsules (Figs 34.1A and D).

Toxic Part

Unripe fruit capsule (Fig. 34.1B), which yields the latex, juice on incision. The opium is collected after all the flower petals have fallen off. Slits are made along the circumference of the seed capsule (Fig. 34.1C) allowing the milky latex to ooze out and harden. After the plastic gummy opium is removed, it can be refined into heroin, morphine, and codeine. Opium is used extensively as a sedative and painkiller. The various derivatives are also habit-forming narcotics.[6] Seeds inside are nonpoisonous and called *khaskhas* (Fig. 34.1D) which constitute a condiment in Indian cooking.

Active Principle

Capsule latex juice has *opium alkaloids*. An alkaloid is a complex substance with nitrogenous base and behaves like an alkali and unites with acid forming salts. The crude opium has about 25 alkaloids, which belongs to *two groups,* namely *Phenanthrine derivatives* and *Benzyl isoquinoline derivatives*.[1-9] Phenanthrine derivatives generally have the alkaloids with *sedative* and *analgesic* properties, while the benzyl isoquinoline derivatives



Figs 34.1A to C: (A) Opium (Poppy) plant with leaves, flower and an unripe fruit capsule. (B) Poppy unripe fruit capsule. (C) Incising the capsule which exudes the latex which has the crude opium in it

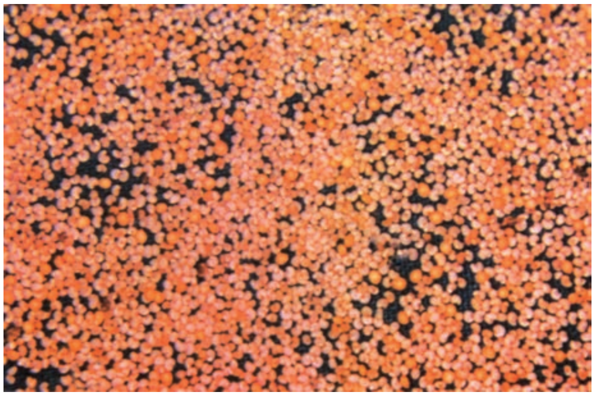


Fig. 34.1D: Poppy seeds (Khaskhas)

have the alkaloids with *antitussive* and *smooth muscle relaxant* effects.

Phenanthrine Derivatives

- Morphine (10%) – in its pure form is a white crystalline powder, having bitter taste, freely soluble in alcohol and water. Morphine has a mode of action of depressing cerebral cortex, most of the centres of medulla, including *respiratory centre* and stimulates *vagal nucleus* and *vomiting centre*.
- Codeine (0.5%) – It is a white, crystalline powder, with bitter taste, is chemically CH_3 morphine and is soluble in water. Its principle action is depression of the cough centre.
- Thebaine (0.3%) – It is a convulsant poison that can produce tetanic convulsions, resembling tetanus/strychnine.

  Semisynthetic and synthetic preparations of opium:
- Heroin *(Diacetyl morphine)* or *brown sugar*– previously used as a sedative, but for its narcotic and addiction forming effect its production has been banned. It is considered as health hazard for younger generation, all over the world.
- Dionin *(Ethyl morphine)* – It is being used as a cough sedative in intractable cough, as it depresses the cough centre.

Benzyl-isoquinoline Derivatives

- Papaverine (1%) – It has no narcotic effect, but has a smooth muscle relaxant effect.
- Narcotine (Noscapine) (6%)

All these are natural derivatives of opium and are called "*opiates*". However, there are certain other drugs, which have a similar action but are not derived from natural opium and are called as "*opioids*", e.g. *pethidine (meperidine), methadone, pentazocine,* etc. are all synthetic derivatives. Table 34.1 enumerates the commonly encountered *opiates* and *opioids*.

Action

Opiates act by binding with four major classes of opiate receptors with differing affinity[8-12] and produce major toxic effects upon the central nervous system, cardiovascular system and gastrointestinal tract.[10] Table 34.2A and B highlight these aspects respectively.

Signs and Symptoms

The signs and symptoms comprise of *direct effect* of opiates on central nervous system and *maladaptive behavior* associated with the *regular use* of opiates, constituting *acute* and *chronic poisoning* respectively.[8]

ACUTE POISONING

While the traditional account is presented in detail below, the contemporary explanations encompassing the *medical syndromes* such as opioid intoxication and opioid withdrawal[8,13] are discussed in detail and presented in Table 34.2C. However, the *acute overdose* is usually a medical emergency and is

Table 34.1: Commonly encountered opiates and opioids

Opium—Natural derivatives	*Semi-synthetic analogues*	*Synthetic analogues*
Phenanthrine derivatives	• Heroin	• Alphaprodein (*Nisentil*)
• Morphine	• Hydromorphine (*Dilaudid*)	• Anileridine (*Laritine*)
• Codeine	• Oxycodone (*in Percodone*)	• Butarphanol (*Stadol*)
• Thebaine	• Oxymorphone (*Numorphan*)	• Dextromethorphan
Benzyl-isoquinoline derivatives		• Diphenoxylate (*Lomotil*)
• Papaverine		• Fentanyl (*in Sublimaze*)
• Narcotine (*Noscapine*)		• Levorphanol (*Levo-Dromoran*)
		• Meperidine (*Pethidine, Demerol*)
		• Methadone (*Dolophine*)
		• Nulbuphine (*Nubain*)
		• Pentazocine (*Talwin*)
		• Propoxyphene (*Darvon, Darvocet*)

Table 34.2A: Opiate/opioid receptors and their action[8-12]

Receptors	*Locations*	*Actions*	*Drugs*	*Comments*
Mu receptors with subtypes of Mu_1, and Mu_2.	Cerebral cortex (lamina IV), thalamus, and periaqueductal grey area.	*Mu_1* – accountable for supraspinal analgesia, and euphoria. *Mu_2* – accountable for physical dependence, miosis, spinal analgesia, respiratory depression, gastrointestinal dysmotility, etc.	Majority of the therapeutically used opiates have the affinity for these receptors Morphine, fentanyl, codeine, Naloxone	Classic effects of opioids act at this receptor, Mu_1 present in low concentrations at birth
κ (Kappa) receptors with three subtypes κ_1, κ_2 and κ_3	Spinal cord	κ_1 – accountable for miosis, spinal analgesia κ_2 – linked with *psychotomimesis* and dysphoria κ_3 – accountable for supraspinal analgesia	Dynorphine, mixed antagonists (e.g. butorphanol, pentazocine, nalbuphine)	Ceiling effect, may cause withdrawal
δ (Delta) receptors	Frontal cortex, limbic system, olfactory tubercle	This type is accountable for spinal and supraspinal analgesia	Enkephalin, endogenous opioid peptides	Functional significance unclear
σ (Sigma)	—	• *psychotomimetic:* hallucination and dysphoria • Tachycardia, hypertension, respiratory and vasomotor stimulation	Phencyclidine, ketamine, pentazocine	

Table 34.2B: Physiologic effects of opioids by organ system[8,10]

Central Nervous System
- Analgesia
- Sedation
- Nausea and vomiting
- Miosis
- Antitussive
- Dysphoria

Respiratory System
- CO_2 response
- Minute ventilation, rate, tidal volume

Cardiovascular System
- Bradycardia (fentanyl, morphine)
- Tachycardia (meperidine)
- Histamine release (morphine)

Gastrointestinal System
- Decreased motility and peristalsis
- Increased sphincter tone: Oddi, ileocolic

Table 31.2C: Medical syndromes of either opioid intoxication or opioid withdrawal[8,13]

Syndrome (onset and duration)	*Characteristics*
Opiate intoxication	Conscious, sedated, '*nodding*'; mood normal to euphoric; pinpoint pupils; history of recent opiate use.
Acute overdose	Unconscious; pinpoint pupils; slow, shallow respirations.
Opiate withdrawal	• *Anticipatory* behaviour # *(3-4 hours after last 'fix')*—Fear of withdrawal; anxiety; drug craving; drug-seeking. • *Early (8-10 hours after last 'fix')*—Anxiety, restlessness; yawning; nausea, sweating; nasal stuffiness, rhinorrhoea; variation, dilated pupils; stomach cramps; drug-seeking behaviour. • *Fully developed (1-3 days after last 'fix')*—Severe anxiety; tremor; restlessness; piloerection (*cold turkey*); vomiting, diarrhoea, muscle spasms (*kicking habit*); muscle pain; increased blood pressure, tachycardia; fever, chills; impulse-driven drug-seeking behaviour. • *Protracted abstinence (may last up to 6 months)*—Hypotension, bradycardia; insomnia; loss of energy, appetite; stimulus driven opiate craving.

Note: Time given above refer to heroin. Withdrawal will develop more slowly with long acting opiates such as methadone.# Anticipatory symptoms begins as acute effects subside

considered as a complication of *opioid intoxication*. Table 34.2D presents the manifestations of acute opiate poisoning in general.

Opioid Intoxication

On the basis of its action on central nervous system the signs and symptoms are traditionally divided into three stages: It acts by combination of stimulation and depression of central nervous system producing a *stage of excitement*, followed by *stage of depression* or *stupor*, to merge into a third and final stage of *narcosis*.[1-5] Signs and symptoms manifest with 30 to 60 gm of oral ingestion.

Table 34.2D: Acute opiate overdose manifestation in an alphabetical order

- Bradycardia
- Coma
- Convulsions
- Cyanosis
- Hypotension
- Hypothermia
- Non-cardiogenic pulmonary oedema
- Pinpoint pupils (dilated if hypoxia supervenes)
- Urinary retention

Signs and Symptoms of Central Nervous System

Stage of Excitement

This stage chiefly due to the initial stimulation of CNS and includes sense of euphoria, a pleasurable feeling, hallucination and convulsions, especially in children.

Stage of Depression (Stupor)

In this stage the victim presents with weariness, weakness, headache, giddiness, heaviness in limbs, urge to sleep, itching of skin and constricted pupils (*pinpoint pupils*).

Stage of Narcosis

As the time passes, the victim from the stage of stupor goes into the stage of narcosis/deep sleep, which leads to further manifestations, as represented in Figure 34.2.

Signs and Symptoms of Cardiovascular System

Patient can present with *orthostatic hypotension* and syncope when trying to assume supine to sitting position.

Signs and Symptoms of Gastrointestinal Tract

Opiates in toxic dose can produce decrease in gastric motility and increases antral muscle tone and proximal duodenal muscle tone. Increase in segmental contractibility and decreased longitudinal propulsive peristaltic movement of small intestine and colon may create spasm. Increase in ileocecal valve and anal sphincter tone can result in antidiarrhoeal action, and discomforting side effects of opiates, such as intestinal cramping, atony, faecal impaction, etc. Nausea and vomiting may be due to the stimulation of chemoreceptor trigger zone in the area postroma of the medulla.

Fatal Dose

- *Crude opium*—200 to 900 mg is fatal, in a non-addicted adult
- *Morphine*—180 to 480 mg (addiction is known with morphine).

Fatal Period

45 minutes—9 to 12 hours minimum and 2 to 3 days maximum.

Toxicity Rating

- Crude opium—5
- Morphine—6

Treatment

- Emetics may be useful only in cases brought immediately after taking the poison, as later on the vomiting centre also gets depressed

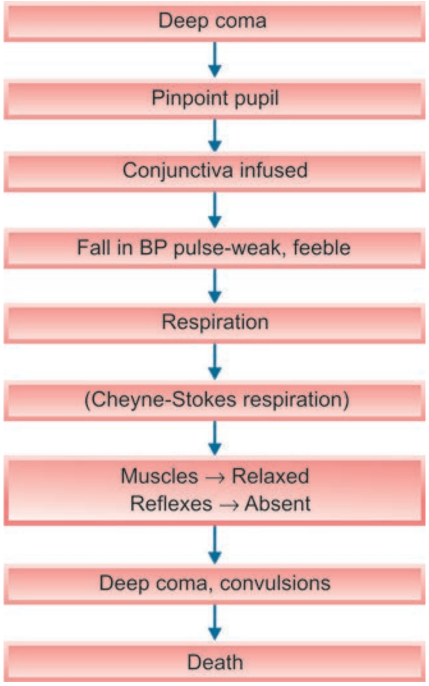


Fig. 34.2: Manifestations in stage of narcosis

- Gastric lavage with 1:1000 $KMnO_4$ (useful for even parenteral poisoning cases as the morphine absorbed is resecreted by the gastric glands into the stomach)
- Strong coffee
- Maintain body warmth
- Specific antidotes like—Oxymorphone derivatives, *Naloxone*, *Naltrexone* and *Nelmefene* are pure antagonist, are presently recommended and approved.[17-20]
  - — *Naloxone* (Indian brand name *Narcotan, Troikaa 2 ml ampoules of 20 mcg/ml and 1 ml ampoule of 400 mcg/ml)* is a drug of choice today and administered intravenously in a dose of 1.2 mg for an adult and 0.4 mg for a child. Single dose is usually short-lived and hence repeated every 15 minutes. Table 34.3 enumerates the intravenous *Naloxone regimens*.
  - — *Naltrexone* (Indian brand name: *Naltima, Intas* 50 mg tablets) is a long acting opiate antagonist, which can be administered orally. It is usually used for treating opiate addiction. It is administered in a dose of 50 mg/day orally, for several weeks or months. However it is not recommended for opiate dependent patient.
  - — *Nalmefene* is a naltrexone derivative with a longer duration of effect compared to naloxine in acute poisoning, given intravenously. Usually begin with 0.1 mg, followed by 0.5 mg if there is no withdrawal reaction and one more dose of 1 mg is administered within 2-5 minutes, only if necessary.

Differential Diagnosis

Opium poisoning may resemble:

- Cerebrovascular accidents, especially in elderly, onset is abrupt, there is hemiplegia and pupils unequal, with high blood pressure. In pontine haemorrhage, the pupils are pinpoint and the body temperature is high.

Table 34.3: Intravenous nalaxone regimens

Regimen – A	Adult: 0.4-0.8 mg; repeat × 2 Child: 0.01 mg/kg; repeat × 2	Recommended for opiate toxicity caused by *heroin, morphine* and *codeine*
Regimen – B	Adult: 1.2 mg; 1.2 mg × 1 Child: 0.03 mg/kg; 0.03 mg/kg × 1	Incorporates the *three* repetitive doses of Regimen – A and is recommended for routine use in non-addict to quickly reverse opiate effects.
Regimen – C	Adult: 0.4 mg; 4.0 mg × 1 Child: 0.01 mg/kg; 0.1 mg/kg × 1	Recommended for poisoning due to high doses of long duration opiates (e.g. *Propoxyphene, methadone, and diphenoxylate*)

Note: Reproduced from Clinical Management of Poisoning and Drug Overdose, by Haddad Winchester, WB Saunders Co, 1983;429

- Uraemia or diabetic coma—smell of urine/ketone bodies, history of kidney disease and diabetes is available.
- Alcohol intoxication and poisoning—breath smells of alcohol
- Carbolic acid poisoning—phenolic odour present.
- Organophosphorus poisoning—kerosene like smell and froth around mouth and nostrils.
- Other comatose conditions such as epileptic coma, hysterical coma, barbiturates poisoning coma, etc. are to be ruled out in a case.

Acute Poisoning/Opiate Overdose

Circumstances of Poisoning

- It could be due to therapeutic overdose, accidental overdose (in addicts), or deliberate overdose (suicidal).
- Receiving '*Hotshots*' in an illicit opiate user, implying taking concentrated form of opiate (i.e. dose > the tolerance the addict has developed)
- Accidental paediatric over dosage in the therapeutic clinical setting.
- Deliberate self-poisoning in the drug-experimenting adolescent and suicide attempt patient.
- Mixed substance abuse such as with – methanol, ethanol, barbiturates, and antianxiety or antidepressant medication.

Signs and Symptoms

- Severe overdosage—Following rapid intravenous opiate administration, is characterized by apnoea, circulatory collapse, convulsions, cardiopulmonary arrest and death.
- Less severe overdosage—Less severely poisoned patients classically present with *triad* of: *CNS depression, meiosis and respiratory depression*.
- The additional finding of needle marks or '*tracks*' or *abscess* strongly suggest chronic parentral opiate use. The important clinical manifestations of acute opiate poisoning/overdose are summarized in Table 34.4.

Table 34.4: Clinical manifestations of acute opiate poisoning/overdose

Severe over-dosage	*Less severe over-dosage*
• *Apnoea* • *Circulatory collapse* • *Convulsions* • *Cardiopulmonary arrest* • *Death*	*CNS depression:* • Coma • Convulsions • Urinary retention *Miosis:* • Pinpoint pupils • may be dilated when hypoxia is severe *Respiratory depression:* • Bradypnoea • Cyanosis • Non-cardiogenic pulmonary oedema *Cardiovascular depression:* • Hypotension

Acute Complication of Overdose

The major cardiopulmonary toxic effect of opiate poisoning, present in virtually all fatalities is pulmonary oedema. Onset of symptoms is generally rapid, occurring immediately following injection of heroin, but it may be delayed for several hours. In the comatose patient it may be delaying upto 24 hours after recovery from coma following administration of a narcotic antagonist. Patient will present with bilateral rales, sinus tachycardia, respiratory distress, hypoxia, and respiratory acidosis.

Opioid Withdrawal *(Withdrawal Syndrome, Abstinence Syndrome, Cold Turkey)*

Abrupt cessation of opiate intake can cause withdrawal syndrome. Generally, withdrawal syndrome symptoms develop at the time the next dose would ordinarily be administered. The intensity and character of withdrawal symptoms are directly related to the daily amount of opiate used and the state of increased excitability of the bodily functions that have been depressed by the opiate.[21] Tables 34.2C and 34.5 summarize the time related and general manifestations of withdrawal syndrome respectively.

The withdrawal symptoms gradually increase in intensity, reaching a maximum at 36 to 72 hours for *heroin* and *morphine*, and subside gradually over 5-10 days. Withdrawal precipitated by narcotic antagonist administration is manifested by the development of symptoms within a few minutes and maximum intensity within 30 minutes.

CHRONIC POISONING *(Synonym: Opium/Opiate Addiction, Morphinomania, Morphinism, Opioid Dependence, Opioid Abuse)*

The major complications of chronic opiate administration in the setting of clinical and illicit use are development of *tolerance, psychological dependence* and *physical dependence*. Thus opium is a drug of addiction. Regular use of opium for painful conditions results in addiction. The condition is also called as *morphinomania* or *morphinism*. The cost of drug sometimes makes an addict antisocial activities by stealing money, ornaments, etc. to get the drug. Person who is an addict indulge in usually displays a common syndrome, consisting of *dysphoria*, problems of sexual identification, and disturbances of interpersonal relationships, dermal scars (from intravenous abuse), confusion, and occasional hallucinations. For him, life is tolerable with drug only. Apart from this there are several *behavioural characteristics* and *medical complications* observed in an addict.

Behavioural Characteristics

- Abrupt changes in quality of work, work output, attendance,
- If the addict is a student, he may present with poor attendance, grades and discipline.
- Unusual flare-ups or outbreaks of temper.
- Withdrawal from responsibility.
- General changes in overall attitude.
- Deterioration of physical appearance and grooming.
- Wearing of sunglasses at inappropriate times.
- Continual wearing of long-sleeved garments particularly in hot weather or reluctance to wear short-sleeved attire.
- Association with known substance abusers.
- Unusual borrowing of money from friends, co-workers or parents.
- Stealing small items from employer, home or school.
- Secretive behavior regarding actions and possessions; poorly concealed attempts to avoid attention and suspicion such as frequent trips to storage rooms, restroom, basement, etc.

Diagnostic Criteria or Opioid Dependence and Severity of Opioid Dependence

There are about eight diagnostic criteria for opioid dependence and five grades of severity of opioid dependence as put forth by the American Psychiatric Association. Considering the criteria, it states that at least three criteria must be present:[8,13]

1. Opioids are taken in larger amounts or over a longer period than the person intended.
2. A desire for the drug persists, or the patient has made one or more unsuccessful efforts to cut down or control opioid use.
3. A great deal of time is spent in activities necessary to obtain opioids (such as theft), taking the drug, or recovering from its effects.
4. The patient is frequently intoxicated or has withdrawal symptoms when expected to fulfil major role obligations at work, school, or home (e.g. does not go to work, goes to school or work in “high,” is intoxicated while taking care of his or her children) or when opioid use is physically hazardous (such as driving under the influence).
5. Important social, occupational, or recreational activities are given up and reduced.
6. Marked tolerance; needs greatly increased amounts of the drug at least 50 per cent increase to achieve the desired effect, or notably diminished effect occurs with continued use of the same amount.
7. Has characteristic withdrawal symptoms (see opioid withdrawal syndrome Table 34.5)
8. Opiates are often taken to relive or avoid withdrawal symptoms. In addition, some symptoms of disturbance have persisted for at least a month or have occurred repeatedly over a longer period.

Severity of Opioid Dependence

Severity is assessed into five grades as presented below:[8,13]

Mild – few, if any symptoms are present in excess of those required to make diagnosis, and the symptoms result in no more than mild impairment in occupational functioning or in usual social activities or relationships with others.

Moderate – functional impairment of symptoms is between mild and severe.

Severe – many symptoms are present in excess of those required to make the diagnosis, and the symptoms greatly interfere with occupational functioning or usual social activities or relationships with others.

Partial remission – during the past six months, there has been some use of substance and some symptoms of dependence.

Full remission – during the past six months, either there has been no use of opioids, or opioids have been used and there were no symptoms of dependence.

Medical Complications

Medical complications of opiate addict are not due to opiate. Malnutrition, unattended diseases, infection, infection and injury from the irritant properties of diluents in the street source of opiates cause morbidity among the users. Non-sterile substances like lactose, sucrose, manniitol, starch, quinine, baking soda, talc, etc are the common diluents used. Such diluents usually contain pathogenic microorganisms.

In addition the addict frequently ignores sterile precautions during injection, which, through tissue injury, facilitates the development of infectious diseases. The drug for injection is commonly placed in a unsterile spoon and a small quantity of tap, rain or toilet bowl water is added to dissolve the drug. The solution process is hastened by heating the spoon over a candle or lighted match. A piece of fabric is added to the solution to trap larger particles in the solution. The solution is drawn up through a needle into syringe or nose dropper or to injection; the needle is frequently *licked* or *rubbed on the scalp* so as to penetrate skin smoothly and easily. The needle is *freely shared* among addicts, will not be cleaned unless the clotted blood occludes the needle lumen. The other medical complications are shown in Table 34.6.

Table 34.5: Signs and symptoms of opiate withdrawal syndrome*

Early	*Intermediate*	*Late*
Yawning	Mydriasis	Involuntary muscle spasm
Lacrimation	Piloerection	Fever
Rhinorrhoea	Flushing	Nausea
Sweating	Diaphoresis	Intestinal discomfort
	Tachycardia	Diarrhoea
	Twitching	Vomiting
	Tremors	Spontaneous ejaculation or orgasm
	Restlessness	Increased blood sugar
	Irritability	
	Anorexia	

* *Reproduced from Clinical Management of Poisoning and Drug Overdose, by Haddad Winchester, WB Saunders Co, 1983;429*

Table 34.6: Medical complications of opiate use

Infection	*Endocarditis – Bacterial (Staphylococcal)/fungal (Candidal), Soft tissue infections – Abscess, Nodules, Ulcers; Tetanus, Malaria, Tuberculosis, Bacteraemia, Sepsis, Hepatitis B; HIV*
Pulmonary	*Pneumonia, Pulmonary, Oedema, Embolism, Atelectasis, Fibrosis, Glaucoma*
Dermatological	*Tracks and scars, Cellulitis, Lymphangiitis, Phlebitis, Gangrene of the skin.*
Neurological	*Cerebral oedema, Transverse myelitis, Horner's syndrome, Postanoxia encephalitis, Polyneuritis, Crush injury.*
Hepatic	*Cirrhosis, Alcoholic hepatic disease.*
Renal	*Nephropathy, Glomerulonephritis, Immune complex nephritis*
Musculoskeletal	*Infectious spondylitis, Sacroiliitis, Lumbar vertebral osteomyelitis, Myositis ossificance, Camptodactylia*
Haematological	*Thrombocytopenia, Leucopenia, Anaemia*

Treatment of Chronic Poisoning

1. Gradual withdrawal of opiate.
2. Substitute therapy with methadone begun at 30-40 mg/day and then gradually tapered off.
3. A beta adrenergic blocker like propanolol (80 mg) is said to be quite effective in relieving the anxiety and craving associated with opiate addiction, but has no effect on physical symptoms. Clonidine can be used alternatively,[18,14,15] which ameliorates symptoms of opioid withdrawal syndrome via noradrenergic pathways such as watery eyes, runny nose, sweating, diarrhoea, chills, and goose flesh. Bone and muscle pain, insomnia, and craving for euphoric effects of opioids are not relieved by clonidine.
4. Nitrous oxide is found to show promising results; however this is not recommended currently as study results are awaited for final approval in clinical practice.[8]
5. Antispasmodics may help abdominal cramps, vomiting, diarrhoea, etc.
6. Sedation by tranquilizers at bedtime may be necessary.
7. Psychiatric counselling is mandatory ultimately.

Postmortem Findings

Postmortem findings are those of *coma* or *comatose asphyxia*.[1,2]

External

- Body may emit smell of opium.
- Deceased may present with severe emaciation and also untidy looks.
- Injection marks may be seen in the anticubital fossae, forearms, back of the hands, neck, groin, and ankles. A true addict may try to conceal puncture mark/scars by tattooing designs around the injection site. Dermal abscesses, scarring may also give clue to the drug abuse.
- Face/body—bluish/deeply cyanosed/blackish
- Postmortem lividity—purple/blackish
- Froth at nostril
- Pupils' constricted/dilated.

Internal

All organs are congested, trachea contains frothy secretions and the blood is dark and fluid. Lungs may present with gross pulmonary oedema and froth exuding from mouth and nostrils, is a common feature among the sudden *heroin* related deaths. Stomach may show the presence of small, soft brownish lumps of opium, and the smell of the drug may be perceived. It disappears with onset of putrefaction.

In opium poisoning, preserve the following items for chemical examination: blood, bile and brain along with other viscera collected routinely.

PETHIDINE (MEPERIDINE)

Classification—Pethidine is an opioid, narcotic analgesic drug, which is a synthetic derivative of opium.

Properties—It is colourless, crystalline powder with bitter taste.

Action

Action of pethidine resembles that of morphine and produces a degree of euphoria and therefore is not free from the risk of addiction. It is administered mainly by I/M or I/V routes for analgesic, antispasmodic and sedative effects, with a therapeutic dose of 50 to 150 mg.

Signs and Symptoms

An overdose on account of cerebral excitation can produce flushing of face, dilated pupils, disturbances of vision, dry mouth, tachycardia, raised temperature, vomiting, excitement, tremors, convulsions, etc. These may be followed by drowsiness, coma, and death from respiratory depression.

Fatal dose—1 to 2 gm with *monoamino oxidase* (MAO) inhibitor or phenothiazine, even a much smaller dose can be fatal.

Fatal period—24 hours.

Toxicity rating—5.

Treatment

- Gastric lavage
- Intravenous administration of coramine
- General measures
- Symptomatic line of treatment.

Postmortem Appearances

Postmortem appearances are those of asphyxia.

Pethidine Addiction

It is quite severe, difficult to treat and has high mortality rate. Pethidine is often used for its analgesic, sedative and tranquilising effects, establishing tolerance rapidly. Common victims of addiction are doctors, nurses or other paramedical professionals to whom the drug is easily available. Continued use can lead to addiction, characterized by:

- Euphoria
- Dulling of intelligence
- Impairment of memory
- Withdrawal symptoms when drug is withheld or when more dose is required
- Larger doses can result in confusional state with hallucinations, illusions and personality changes rapidly than with morphine.
- Accidental poisoning with therapeutic doses is also reported.

Medicolegal Importance[1-5,16]

- Opium preparations are used therapeutically to reduce pain and induce sleep.
- In countries which have legalized *euthanasia*, morphine is used as a drug of choice, since opiate is a reputed drug to cause painless death.
- Opiates are common among the drugs of abuse in India. In cities like Mumbai, Delhi, etc. Heroin (*Brown Sugar*) is the commonest drug of abuse. Codeine, which is easily available as an antitussive is increasingly becoming popular among the college youth as drug of addiction.
- Morphine, pentazocine, pethidine are therapeutic preparations commonly abused among the medical and paramedical workers.
- It is a poison of choice to commit suicide (*ideal suicidal agent*) among the medical and paramedical professionals.
- Homicide by giving opium is rare because of bitter taste and characteristic odour.
- Infanticide by breastfeeding the infant by the woman who had smeared her nipple with tincture opium, is easy.
- Accidental fatal poisoning by overdose is not infrequent, and reported among
  - Intravenous heroin abusers (*death on the needle*)
  - Criminals who take opium to *build courage* before committing crime, e.g. homicide
  - It is said to increase *libido* and hence often used as an *aphrodisiac*.

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ALCOHOL

CLASSIFICATION

Alcohol is an *inebriant* cerebral neurotoxic poison. Alcohol is also classified as *sedative and hypnotic*.

INTRODUCTION

Alcohol in general refers to an aqueous solution containing 95 per cent ethyl alcohol. Alcohol is one of the oldest intoxicants ever known to man. Alcoholic beverages consumption is increasing in every part of the world. Emergency physicians and toxicologists are paying increasing attention to the acute and chronic effects of alcohol, alone or in conjunction with other intoxicants.[1,2]

Many countries including India, have tried *prohibition* but with little success. Illegal imports, illicit manufacture, consumption of dangerous substitutes like methylated spirit, production and consumption of cheap and dangerous imitations and forgery of liquor permits are some of the consequent problems of prohibition. Recently it has been observed that the crimes involving alcohol directly or indirectly are also increasing significantly.[3,4]

The purpose of this chapter is to understand the mechanics of alcohol, its nature, effects, detection, etc. which is essential for a medicolegal expert, police and judiciary, in order to deal effectively with crime involving alcohol.

Alcohols are *hydroxyl derivatives* of *aliphatic hydrocarbons*. Routinely there are *three* categories of alcohols, *monohydroxy alcohols, dihydroxy alcohols,* and *trihydroxy alcohols*.[5]

- The *monohydroxy alcohols* have only *one* hydroxyl (OH) group and include – *ethyl alcohol* (C_2H_5OH), *methyl alcohol* (CH_3OH), *isopropyl alcohol, isobutyl alcohol, amyl alcohol,* etc as examples. Among these *methyl alcohol* is much more toxic than *ethyl alcohol*. *Isopropyl alcohol* is three times more toxic than *methyl alcohol*, whereas *isobutyl alcohol* is one and half times as toxic as *isopropyl alcohol* and *amyl alcohol* is one and half times as toxic as *isobutyl alcohol*.

- The *dihydroxy alcohols* have *two hydroxyl (OH) groups* and are often called as *glycols*. They include *ethylene glycol*, *propylene glycol*, etc. as examples.
- *Trihydroxy alcohols* – are not really alcohols, but their derivatives, such as the *propane derivative, glycerol* or *glycerine.*

ETHYL ALCOHOL *(Synonyms: Alcohol, Ethanol, Grain Alcohol)*

Physical Properties

In a pure form ethyl alcohol is transparent, colourless, volatile liquid, having a spirituous odour and burning taste. It is both water-soluble and lipid soluble.

Production

Ethanol is obtained by fermentation of sugars, molasses, grains, fruit juices and starch. It is a complex enzymic process. The enzymes are obtained from yeast. It is carried out under controlled conditions. The final fermented mass contains about 10 per cent alcohol. It is then purified by *distillation*. All strong alcoholic beverages are distilled products after fermentation.

It could be available as following types:[1-4]

- ***Absolute alcohol*** or *alcohol dehydratum* (contains 99.95% ethyl alcohol by volume)
- ***Rectified spirit*** (contains 90% ethyl alcohol by volume)
- ***Industrial methylated spirit*** or denaured alcohol (contains 95% ethyl alcohol and 5% wood naphtha by volume)
- ***Surgical spirit*** refers to methylated spirit which contains in addition small amounts of castor oil and oil of Wintergreen (methyl salicylate).
- ***Alcoholic beverages*** (Fig. 34.3) – The active ingredient of all beverages/ liquors is alcohol. Alcoholic beverages are mixtures of *absolute ethyl alcohol*, *water* and *congeners* (which are organic compounds, esters and acids) formed during fermentation process as in most of the alcoholic beverages; exception being in wine and brandy wherein the congener is methyl alcohol.
- ***Proof spirit:*** It refers to a standard mixture alcohol and water of relative density. It means that strength of absolute alcohol which when poured onto gunpowder gets it burnt completely (this strength is 57.10% by volume and 49.28% by weight). Thus proof strength of alcohol in a beverage can be calculated by dividing the alcohol per cent (volume strength) in it by 0.571. Consequently, a particular whisky/ brandy containing 43 per cent alcohol have a *proof strength* of 43/0.571 = 75° (expressed in degree).[1,2,4-7] Table 31.7 presents the approximate percentage concentration of absolute alcohol (by weight, by volume and proof strength) in some of the common alco-holic beverages in use.
- Alcohol beverages, various brands, their concentrations and uses of alcohol are presented in Tables 34.7 and 34.8 respectively.

Director's Special Whisky
MANSION HOUSE
ROYAL STAG
Officer's Choice

Fig. 34.3: Various brands of alcoholic beverages

Absorption of Ethyl Alcohol

Alcohol because of its smaller molecular size is rapidly absorbed in gastrointestinal tract. Absorption of ethyl alcohol is mainly from *stomach (20%)* and *intestine (80%)*. It commences almost immediately after ingestion within 5 minutes and completed within one to two and half an hour of ingestion. Almost 60 per cent of alcohol is absorbed in first 60 minutes. However, certain factors that can interfere with absorption are:[1,7]

- *Food*—Presence of food, especially fats and proteins in the stomach delays absorption. Absorption is faster in an empty stomach.
- *Concentration/strength* of alcoholic beverages taken—Higher the strength, rapid will be the rate of absorption.
- *Tolerance*—Body tissues of certain people absorb alcohol more easily than those of others. This has been though not understood fully, is suspected to be due to the fat deposition in the body tissues.

Table 34.7: Alcoholic beverages, their brands and concentration percentage of absolute alcohol in it

Beverage types	*Concentrations*		
	By weight (%)	By volume (%) [6]	Proof (°) [6]
Rum	50-60	42.8	75.0°
Whisky, Brandy, Gin, Arrack (Country Liquor)	40-50	42.8	75.0°
Fortified Wines: like port, sherry[#]	18-22	15.5	27.0°
Natural Wines: Burgundy, Hock, Claret*	10-15	08.0	14.0°
Natural Wines: Champagne*	10-13	08.0	14.0°
Strong beers*	02-06	10.0	17.5°
Ordinary beers*	02-05	02.0	03.5°
Ginger beers*	01-03	02.0	03.5°

[*] *Beers (commonly derived from barely) and wines are undistilled liquors*[3]

[#] *Fortified wines are fermented wines fortified by the addition of alcohol*[3]

Table 34.8: Uses of alcohol

As a beverage	*As a solvent*	*Medicinal*
• Arrack, Toddy, Feni (all these in India), Tequila (Mexico), Sake (Japan), *Eau de vie* fruit brandy (France), Beer, Wine, Whisky, Gin, Brandy, Rum, Vodka, etc.	• In a concentration of 15-80 per cent alcohol is used as solvent in most of the after-shave lotions, colognes, mouthwashes and variety of perfumes	• Alcohol in concentration of 2-25 per cent used in cough syrups antihistaminic, decongestant, multivitamin, etc. • As an antiseptic, e.g. surgical spirit, is a combination of ethanol and methanol (90-95% and 10-5% respectively) • As a preservative: Rectified spirit is the preservative for viscera for chemical analysis • As a fuel is often in use especially in laboratory • Ethanol sponging is an effective remedy for fever/ hyperthermia conditions. • Injecting dehydrated alcohol in close proximity of nerves/ sympathetic ganglion is a successful remedy for incurable chronic pain conditions—*trigeminal neuralgia*. • In methanol poisoning ethanol is a life-saving measure.

- *Habituation*—Habituation can suppress rate of absorption.
- *Drugs*—Insulin, prostigmine, etc increase absorption, while atropine delays
- *Chronic gastritis*—retards absorption.
- *Carbonic acid (soda water) and Chilling*— Presence of soda water can accelerate the absorption, while chilling retards absorption.[7]

Distribution

Once absorbed, alcohol gets fairly and evenly distributed in all organs except bone, adipose tissues, and possibly skin too, attaining equilibrium with a constant blood alcohol concentration and concentration of alcohol in other body fluids, in the ratio of:

- Blood: Urine = 1:1.31
- Blood: Exhaled air = 1:2100
- Blood: Saliva = 1: 12

Blood Alcohol Concentration (BAC)

Alcohol can be detected in blood within 2-3 minutes of a few sips of beer or whisky. After single dose, usually within half to one hour, the blood-alcohol concentration reaches the maximum and gets proportional to the amount consumed.[1,7,11] Accordingly, by estimating the amount of alcohol in the blood, it may be possible not only to calculate the approximate total amount of alcohol in the body at the material time but also the minimum amount ingested.

Calculating Alcohol Level in Blood/Tissue

Concentration of alcohol in blood is expressed as mg% (w/v) or as percentage as follows:

50 mg% (w/v) = 0.05% (w/v), i.e. 50 mg alcohol per 100 ml blood.

It is also expressed as mg% (w/w) in solid tissues like viscera sent for chemical analysis, i.e. so many mg alcohol per 100 gm tissue.

Widmark's Formula to Estimate Alcohol in Blood [6,12]

According to this:

i. Formula for estimating alcohol in blood is
   a = prc,
   Where in
   'a' is the total amount (weight) of alcohol absorbed (in gm) in the body
   'p' is the weight of person (in kg)
   'r' is a Widmark's constant, 0.68 for men and 0.5 for women.
   'c' is the concentration of alcohol in blood (in mg per kg)

ii. Formula for estimating alcohol in urine is:
   a = ¾ prq,
   Where in
   'p' is the weight of person (in kg)
   'r' is a Widmark's constant, 0.68 for men and 0.5 for women.
   'q' is concentration of alcohol in urine (in mg per kg).

Example

70 kg male has consumed 120 ml (around 4 fluid ounces) of 75 per cent proof liquor. His blood alcohol (peak) level will be:

$$= \frac{120 \times 0.428 \times 0.8 \times 1000}{70 \times 10 \times 0.68} \text{ mg \% w/v}$$

$$= 86 \text{ mg\%}$$

List of various concentration and equivalent intake of 75° proof spirit in 70 kg man is presented in Table 34.9. Whisky, gin and brandy are generally 75° proof.

Metabolism and Excretion

Elimination is chiefly through oxidation.[7] Ninety per cent (90%) of ethyl alcohol is metabolized in liver *at the rate of 09 to 15 ml/hour (i.e. 15% from blood/hour),* while the lungs, kidneys and skin mainly excrete the remaining 10 per cent. In the liver, alcohol is subjected to three pathways during its metabolism.

Firstly—In the cell/ cytosol pathway. This is the main pathway and here the *alcohol dehydrogenase* (ADH) *enzyme*, its *coenzyme*, and *nicotinamide adenine-dinucleotide* (NAD) oxidises the alcohol (Fig. 34.4) forming acetaldehyde. Acetaldehyde that is formed is then converted to acetic acid by *aldehyde dehydrogenase*, which in turn is converted to *acetyl coenzyme—A*, and enters the *Citric acid (Krebs') Cycle,* where it is metabolized to *carbon dioxide* and *water*.

Table 34.9: List of concentration and equivalent intake of 75° proof spirit in 70 kg man

Level of alcohol in mg/100 ml of blood	*Minimum consumed volume of 75 per cent proof spirit*	
	In fluid ounces	*In ml*
50	2.45	69.5
100	4.9	139.0
200	9.8	278.0
300	14.7	417.0
400	19.6	417.0
500	24.5	695.0

Calculated presuming Widmark's factor for male = 0.68; and 1 fluid ounce = 28.4 ml

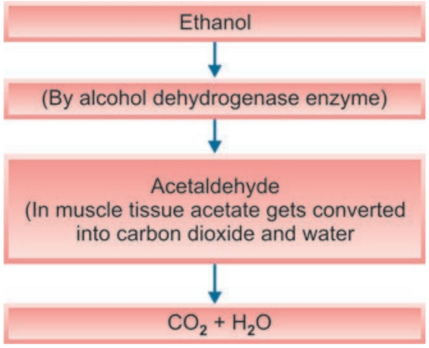
Ethanol
(By alcohol dehydrogenase enzyme)
Acetaldehyde
(In muscle tissue acetate gets converted into carbon dioxide and water
$CO_2 + H_2O$

Fig. 34.4: Illustration of detoxification of ethyl alcohol in liver

Secondly—The pathway through the *microsomal ethanol oxidising system* (MEOS), located on the endoplasmic reticulum).

Thirdly—Peroxidase catalase system (in the hepatic peroxisomes) pathway is the least important pathway. A part of it may be *deposited* in tissues as neutral fat and cholesterol.

Excretion of alcohol is mainly by the *kidneys, lungs* and *skin* through urine, breath and sweat respectively. It is also secreted in *saliva* and *milk* in dilute concentrations. More than 90 per cent being metabolized in the body only 5-10 per cent is excreted unchanged by kidney, lungs and sweat. In lungs, ratio between the concentration of ethanol in alveolar air and blood is 1:2100 constant (*Henry's law*), forms the basis for reliability to estimating blood alcohol concentration by breath using *alcometer*.

Action

Ethyl alcohol mainly acts on CNS. It acts chiefly as a depressant of brain centres. It acts on neural cells in a way similar to *hypoxia* and reduces their activity. Research reports in the past postulated that ethanol depresses the CNS by dissolving in the cell's lipid membrane and causing disorganisation of lipid matrix (membrane fluidisation). Recently, this concept has been disputed by two newer postulations.

The *first postulate* by *Charness, Simon, and Greenberg*, states that the ethanol acts by enhancing *GABA-nergic* function through interaction with *$GABA_A$ receptors* and associated *chloride ion channels*.[8]

The *second postulate* by *Hu and Ticku*, which is more acceptable, deals with N-methyldaspartate (NMDA) ligandgated, glutamate receptors. NMDA receptor mediates neurotoxicity by increasing permeability to *calcium*, and *regulates neuronal long-term potentiation*. Studies make obvious that in acute use of ethanol, NMDA receptor function is inhibited, while in chronic use of ethanol, NMDA receptors get up-regulated.[9]

However, it is well reported that, the initial apparent stimulation seen in early stage is due to the depression of the *higher inhibitory/evolutional centres* (such as centres regulating *conduct, judgement* and *self criticism*), which normally control the human behavioral attitudes, resulting in *behavioral changes* followed by depression of the *vital centres of medulla*, producing *cardiorespiratory failure*, *alcoholic coma* followed by *death*.[1-4,6-7]

Table 34.10 and Fig. 34.5, present various behavioural changes that occur with increasing levels of ethyl alcohol in blood

Table 34.10: Seven stages or *7 D's* of acute alcoholic intoxication based on BAC level

Blood alcohol concentration (BAC) (in mg/100 ml)	*Stages of alcoholic influence*		*Signs and symptoms of intoxication*
	Stages	*Behaviour*	
0-50	I Dry/decent	Sobriety	• No influence apparently—Behaviour almost normal
50-100	II Delighted/ devilish	Euphoria	• Sense of wellbeing, sociability, talkativeness • Greater self-confidence, decreased inhibitions • Diminution of attention, judgment, and control • Loss of efficiency in finer performance tests
100-150	III Delirious/ dizzy	Excitement	• Emotional instability, decreased inhibitions • Loss of critical judgment • Impairment of memory and comprehension • Decreased sensory response, increased reaction time • Some muscular in-coordination
150-200	IV Dazed	Confusion	• Disorientation, mental confusion, dizziness • Exaggerated emotional state, such as fear and anger • Disturbance of sensation (diplopia, etc.) and of perception of colour, form, motion dimensions • Decreased pain sense • Impaired balance, muscular in-coordination, staggering gait, slurred speech
200-300	V Dejected	Stupor	• Apathy, general inertia, approaching paralysis • Markedly decreased response to stimuli • Marked muscular in-coordination, inability to stand or walk • Vomiting, incontinence of urine and faeces • Impaired consciousness, sleep or stupor
300-500	VI Dead drunk	Coma	• Complete unconsciousness, coma, anaesthesia • Depressed or abolished reflexes • Subnormal temperature • Incontinence of urine and faeces • Embarrassment of circulation and respiration • Possible death
500+	VII Dead	Death	• Death from respiratory paralysis

7 D's of Acute Alcoholic Intoxication—Dry/ Decent, Delighted/Devilish, Delirious/ Dizzy, Dazed, Dejected, Dead Drunk, Dead

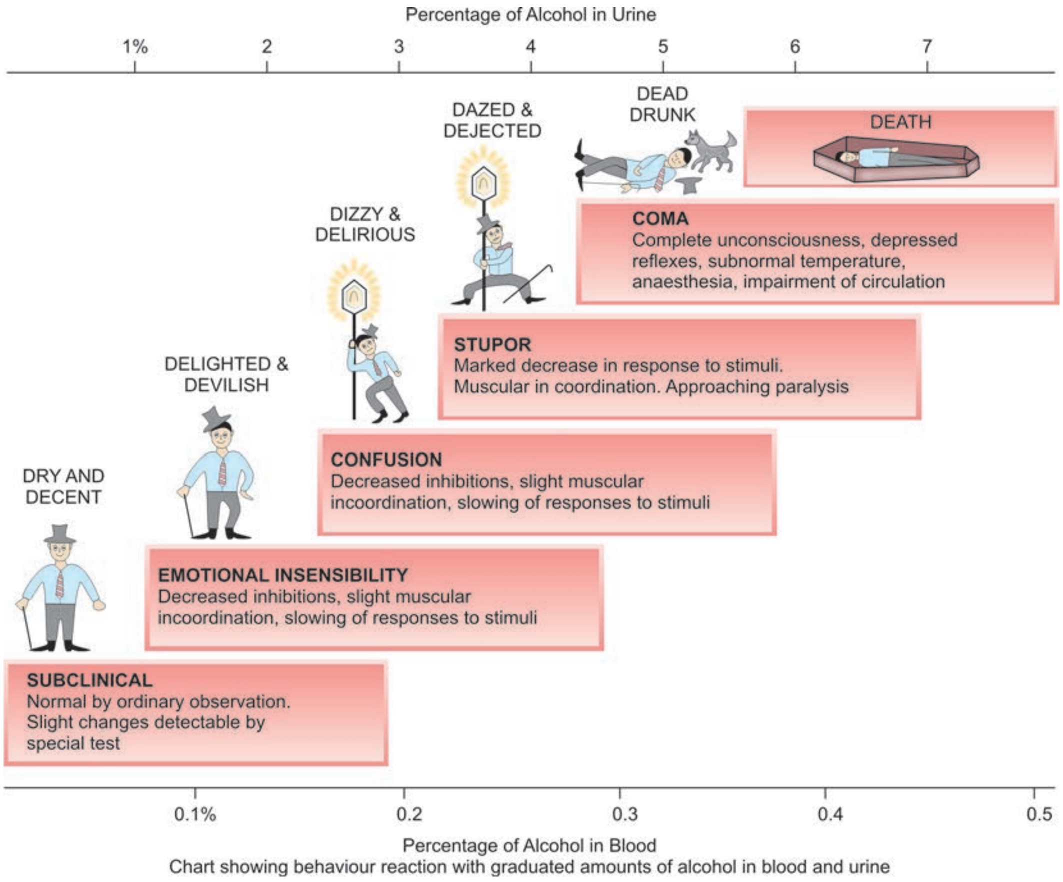

Fig. 34.5: *Seven D's* or *Seven stages* of acute alcohol intoxication
(*Concept from* an old chart hung in the Museum of Forensic Medicine, KMC, Mangalore, Karnataka, India)

and urine. It also acts as *hypnotic*, *diaphoretic* and in small doses acts as an *appetizer*. A little brandy has a carminative effect. Diuresis with alcohol intake is usually due to the inhibition of release of *antidiuretic hormone* from the posterior pituitary. It is though believed to be an *aphrodisiac*, is not so actually. At best it provokes the desire but takes away the performance.[7] Mixing of alcoholic beverages is said to produce greater intoxication, than would be expected from the amount consumed.

Causes of Death

Death is usually caused by direct *depressive effects* upon the *brain stem* or mediated via respiratory centre or due to aspiration of the vomit. Alcoholics may bleed to death from trivial minor trauma. In individuals who sustain head injury, with subdural or epidural haemorrhages, and survive for hours to days, the peripheral blood may be negative for alcohol. Analysis of brain blood clot, which is outside the circulating detoxifying cycle, may reveal the initial blood alcohol levels at the time of injury. Intoxicated person may kill himself by falling, exposure to heat or cold, overdosage with drugs, being burnt by fire, trying swallowing too large bolus of food (*cafe coronary*), drowning, aspiration, etc. Death may also occur from complications. Excessive drinking may produce irreversible brain and liver damage that can prove fatal. Alcoholics may also die from poisoning by various toxic substances present in cheap industrial/ illicit alcoholic concoctions. Table 34.11 highlights these facts. However, it is the experience of the author that deaths due to chronic effects of alcohol surpass the deaths due to overdose of alcohol.

Table 34.11: Causes of death due to alcohol

- Direct depressive effect of brainstem
- Direct depressive effect of respiratory centre
- Aspiration of the vomit, choking *(Café coronary)*, drowning, etc
- Severe bleeding from trivial trauma, esp. subdural/ epidural intracranial haemorrhages
- Irreversible complications of brain and liver damage
- Poisoning by various toxic substances present in cheap industrial/ illicit alcoholic

ALCOHOL POISONING

Clinically alcohol poisoning presents in two forms, *acute* and *chronic*, which are more often referred to as *alcohol intoxication* and *alcohol addiction* respectively.

Acute Alcohol Poisoning

(Alcoholic Intoxication/Alcohol Overdose)

Consumption of any preparation containing alcohol either in small doses at short intervals, or in one big dose beyond his/ her capacity with the blood alcohol concentration exceeding 150 mg/100 ml, constitutes *acute alcohol poisoning*.

Causes

Deliberate heavy drinking is undoubtedly the cause of acute poisoning. However, rarely it can also occur accidentally due to continuous inhalation of alcoholic vapours.

Signs and Symptoms

Alcohol intoxication is mainly because of its depressing action on CNS (both higher and vital centres). Depending on group of signs and symptoms observed, acute alcohol poisoning manifestations are described under three different stages, namely: *stage of excitement, stage of incoordination,* and *stage of coma*.

Stage of excitement: This stage develops with blood alcohol concentration (BAC) of 0.05 to 0.1 per cent, i.e. 50-150 mg/100 ml of blood. In a non-addict person, there is a sense of well-being or *euphoria* and is due to the depression of inhibition controlling capacity of higher evolutionary centres; there will also be changes in behaviour of the person and he becomes:

- Free in action, speech and emotions
- He may perform dancing, thrilling shows, etc. carelessly and fearlessly
- He might disclose secrets
- The person might show increase in confidence, but lack of self-control
- There may be easy sexual excitation and desire but cannot perform the act due to impotency (thus, alcohol is not an aphrodisiac)
- There will be lowering of visual acuity
- There will be reduction of mental concentration power, leading to impairment of judgement.

Stage of incoordination: This stage develops with a blood alcohol concentration (BAC) of 0.15 to 0.25 per cent, i.e.150-250 mg/100 ml of blood. This is due to further depression of higher centre. The individual may present with:

- *Morose/gay/irritable/excitable/quarrelsome/sleepy,* etc behavior depending on the dominant impulses released. Memories of recent events are impaired.
- Soon when the centres of perception and skilled movements are involved, the victim presents with *incoordination, inabilities to perform skilled movements, alteration in speech, etc*. In addition to this he will have:
  - Face flushed
  - Breath with alcoholic smell
  - Eye changes such as:
    - Increase in reaction time.
    - The pupils are dilated and sluggishly react to light (this is a dangerous sign), and accommodation.
    - Positional nystagmus may be observed (i.e. jerky movements of the eyeball in direction of gaze independent of the movement of head and is called as alcohol gaze nystagmus, with blood level at 0.03-0.05%).
  - There will be excessive sweating, loss of body heat, resulting in fall of body temperature (subnormal temperature). This may be also due to the depression of temperature regulation centre.

Stage of Coma

Blood alcohol concentration (BAC) required to develop stage of coma is > 0.25 per cent, i.e. >250 mg/100 ml of blood. This occurs when both motor and sensory cells are deeply affected, resulting in:

- Speech—thick, slurred
- The victim may become giddy, stagger and may fall as coordination is markedly affected.
- Ultimately, he may go into the stage of coma with stertorous breathing, rapid thready pulse, subnormal temperature, etc.
- Pupils are constricted, but it reacts to external painful stimuli (like pinching or slapping on the face, etc.) by dilatation followed by constriction again. This is called as *McEvan's* sign and if this is positive, it is suggestive of alcoholic coma.

However, for an easy rememberance, *acute alcoholism* manifestation may also be discussed under as *seven stages* (or *Seven D's*) *of Acute Alcoholic Intoxication* (see Table 34.10 and Fig. 34.5).

Recovery—Unless a large quantity of alcohol is consumed in short time, recovery is the rule. With recovery, coma gradually lightens into deep sleep. Victim will wake up in 8 to 10 hours with acute depression of mood and complain of nausea and severe headache, etc. called as *alcohol hangover*.

Alcohol hangover—It comprises of throbbing headache, irritability, lethargy, nausea, and abdominal discomfort. While the abdominal discomfort is due to alcoholic gastritis, the other symptoms are due to the effect of *congeners* and other *impurities* present in the alcoholic beverages consumed, which causes mild *cerebral oedema*. The hangover manifestations may also be partly due *alcoholinduced hypogylcemia*.

Death—If the victim does not recover from coma within 5 hours, the prognosis is worse and may result in fatality, due to asphyxia or shock. Thus it is appropriate to mention here that stage VII (Death) is extremely rare with ingestion of pure ethanol. However, alcohol, resembling cocaine, amphetamine, toluene, etc. predisposes cardiac arrhythmia by direct action on myocardium, especially after/during struggle/fight when *catecholamines* released may bring about sudden death. This may occur after violent struggle in intoxicated state, struggling for breath leading to resultant hypoxia.

Table 34.10 provides the signs of alcohol consumption and the degree of intoxication based on blood alcohol concentrations (BAC). This chart reflects the testimony of experts used by prosecutors (government official who conduct criminal prosecutions on behalf of the state) and shows where "*legal intoxication*" begins. Since most of the offences associated with drinking are committed during the *III (Excitement) and IV (Confusion) stages of intoxication*, these stages have become important medicolegally and considered as beginning of '*legal intoxication*'.

Fatal dose (in a non-addict)

- Adult: 150-250 ml of absolute alcohol (Approximately 6 gm of ethanol/kg body weight).
- Children: 60 ml of absolute alcohol (Approximately 3 gm of ethanol/kg body weight).

Fatal period—12 to 24 hours though death maybe delayed for 5 to 6 days.

Toxicity rating—2.

Treatment[5,7,10,12,13]

The most important single factor in the treatment of the acutely alcohol-intoxicated patient is to provide respiratory support.

- Gastric lavage, induction of emesis, activated charcoal, etc are usually not indicated in cases of acute alcohol intoxication, but may be indicated when concomitant drug ingestion is suspected. However, gastric lavage and bowel evacuation with an alkaline solution (5% sodium bicarbonate in warm water) is effective when the patient is brought within 2 hours of ingestion.

- The patient should be kept warm.
- Vitamin B_6 is said to accelerate the metabolism of alcohol, is administered intravenously in a dose of 50-100 mg may effect a rapid recovery.
- Coramine 3-5 ml by slow intravenously may render amasing results.
- Isotonic saline with 5 per cent glucose (preferably fructose) may be required to deal with symptoms of hypogylcaemia, if present.
- Increased intracranial pressure may be treated with saline purges and intravenous hypertonic glucose solution.
- Artificial respiration with oxygen is essential when sever respiratory depression ensue.
- Acidosis (common with methyl alcohol poisoning), if present will require administration of sodium bicarbonate by mouth in a dose of 2 gm in 250 ml water every two hours. When oral therapy is not possible, 50 gm of sodium bicarbonate dissolved in 1 liter of 5 per cent dextrose solution can be given intravenously along with 10-15 units of insulin.
- Alcohol intoxicated person may become belligerent and require special precautions and attentions, protecting self and others.
- Haemodialysis or peritoneal dialysis may be life saving measures in very serious cases.

Postmortem Appearance[1-4, 6-7]

- Odour of alcohol around mouth and nose and upon opening the body.
- Clothes are in disorderly/torn conditions. Stains due to vomit or blood may be seen.
- Bruises are generally found on various parts of the body. Other injuries may also be present.
- Congestion of conjunctiva
- Rigor mortis is prolonged
- Decomposition is retarded
- Brain is oedematous and congested. In individuals who sustain head injury with subdural or epidural haemorrhage and survive for house to days, the peripheral blood may be negative for alcohol. However, chemical analysis of blood clot in case of epidural haemorrhages can give exact concentration of alcohol at the time of injury. Vitreous chemical analysis is also helpful in knowing accurate alcoholic status of the deceased.
- All the viscera are congested and smell of alcohol. In addicts liver may be cirrhotic and brain may show degenerative changes
- Blood—fluid and dark.

Chronic Alcohol Poisoning

(Synonyms: Alcohol Addiction, Alcoholism, Ethanolism)

Consumption of heavy doses of ethyl alcohol beverages for a long period, regularly, characterized by morbid desire to drink alcohol, blackout episodes with intoxication, and withdrawal symptoms on stopping alcohol consumption.

Types of chronic alcoholism There are eight different types[5] (Table 34.12)

Signs and Symptoms

The chronic alcoholic patient will suffer from a set of manifestations involving various systems and organs of the body. These basically comprise of gastrointestinal system, and central nervous system. The systems with organs affected in *ethanolism*, with presenting signs and symptoms and possible complications are tabulated below in Table 34.13.

Table 34.12: Various types of chronic alcoholism

Alcoholisms	*Explanations*
Alpha	Before the abuser develops psychological dependence on alcohol.
Beta	Long continued alcoholism develops cirrhosis, but no *psychological dependence*.
Gamma	Alcohol abuser goes on increasing, resulting in physical and social problems
Delta/French alcoholism	Long continued abuse which can not be abstained, will lead to serious physical complications
Epsilon	Abuser develops habit of dipsomania, when he technically drinks to death
Reactive	Victim drinks alcohol and seeks relief from psychological stress and strain.
Essential	It is he who has got drink for inherent genetic or biological factor but not for any psychological or cultural one.
Symptomatic	Who drinks for long standing psychological illness, psychosis/severe depression.

Treatment

- Use of antabuse therapy (*disulfiram, metronidazole*) – when these drugs are used in the treatment of alcoholism; causes nausea and vomiting if alcohol is ingested, which makes the alcoholic give up alcohol.
- Hypnosis and psychotherapy.

Postmortem Appearance

- Gastric mucosa is deep reddish-brown with patches of congestion or effusion and is hypertrophied.
- Liver is congested and shows fatty infiltration, enlarged or cirrhotic or contracted.
- The kidneys show granular degeneration.
- The heart is dilated and shows fatty degeneration.
- Brain with shrinkage of cerebral cortex (grey matter) is common in chronic alcoholics.

Sudden Stopping of Alcohol in Chronic Alcoholics

Sudden stopping of alcohol intake in a chronic alcoholic can provoke a *withdrawal syndrome/ reaction,* which could manifest as any one of the following:[5,10]

- *Common abstinence syndrome*
- *Alcoholic hallucinosis*
- *Alcohol seizures (Rum fits)*
- *Alcohol ketoacidosis*
- *Delrium tremens (DT)*
- *Wernicke-Korsakoff syndrome.*

Common Abstinence Syndrome

The syndrome complex comprise of manifestations such as tremors in the hands, legs, and trunk. He/she will present with a mental state of extreme emotional disturbance (agitation), sweating, nausea, headache and insomnia. All these events come into sight within 6 to 8 hours of stopping the alcohol intake.

Treatment: Studies with clonidine 0.2 mg orally given several times daily over a 4-day period suggest that it is effective in

Table 34.13: Body systems, organs involved, signs and symptoms, and complications noticed in chronic alcoholism

Systems	*Organs*	*Signs and symptoms*	*Complications noticed**
Gastrointestinal	General	Nausea, vomiting, anorexia, diarrhoea, etc.	Gastritis, periodic diarrhoea, increased incidence of oropharyngeal and oesophageal malignancy.
	Liver	Jaundice, when cirrhosis develops	Fatty liver with portal hypertension, hepatitis, cirrhosis, increased incidence of hepatic carcinoma.
	Pancreas	Acute pain abdomen	Acute/chronic pancreatitis
	General	Generalized oedema	Anasarca, hypoproteinaemia
Cardiovascular	Heart	Palpitation, irregularity in heart beating, rise in blood pressure.	Cardiomyopathy, dysrhythmias, hypertension
Central nervous	Brain and nerves	Tremors of tongue and hand, Insomnia, loss of memory, Impaired power of judgement, Peripheral neuritis.	Polyneuropathy, cerebellar degeneration, demyelination of corpus callosum (Marchiafava-Bognami disease), amblyopia, stroke.
Skeletomuscular	Muscles	Pain in the group of muscles/ all muscles	Myopathy
Neuropsychiatric	Mind	Amnesia and black outs, delusions, delerium tremens, dementia, hallucinations,	Disturbance of memory (amnesia and black outs), delusions, delerium tremens, dementia, alcoholic hallucinations, Wernicke's encephalopathy, *Korsakoff's psychosis,* etc
Respiratory	Lungs	Breathlessness	Aspiration pneumonia, alcohol-induced asthma
Endocrine	Endocrine glands	Incompetence of the gonads in male/ female pseudo-Cushing syndrome	Hypogonadism and feminisation in males, amenorrhoea, menorrhagia and infertility in females.
Haemopoietic	Blood	Anaemia	Anaemia, thrombocytopenia.

** Death is usually due to hepatic failure and coma*

plummeting some of the adrenergic manifestation of alcohol withdrawal.[13] Other drugs suggested to be of potential use,[10] are—dexamethasone, phenobarbital, chlormethiazole, beta-blockers (for mild symptoms), and subanalgesic doses of nitrous oxide, clorazepate, haloperidol, and hydroxybutyric acid in ameliorating some of the symptoms associated with alcohol withdrawal.

Alcoholic Hallucinosis

The person starts seeing objects with distorted shape and their shadows moving. He/she will complain of hearing some one shouting at him or snatches of music, etc. All these events come into sight within 24 to 36 hours of stopping the alcohol intake.

Treatment—Administration of phenothiazine (Chlorpromazine 100 mg, 8th hourly).

Alcohol Seizures (Rum Fits)

These comprise of clonic-tonic movements, with or without loss of consciousness. These manifestations appear within 6 to 48 hours of either cessation or precipitous decline of alcohol intake. True alcohol seizures will usually manifest prior to the onset of delirium tremens (TD). These seizures however do not require any long-term anticonvulsant therapy since the seizers are self limited.[17]

Risk factors: Usually this is reported among 40 per cent of adults with seizures admitted to a hospital and in about 15 per cent of patients with *status epilepticus*. This necessitates routine questioning about alcohol intake in all patient arriving with seizures to an emergency department of a hospital. Some of the risk factors associated with alcohol withdrawal and considered most likely to precipitate seizures are hypoglycemia, hypomagnesaemia, and respiratory alkalosis. Alcoholic withdrawal also heightens the photic sensitivity and can lead to television-induced seizures.[10]

Alcohol Ketoacidosis

Causes

This is found to develop with sudden *withdrawal* of or *heavy drinking* of alcohol. Gastritis or pancreatitis, which has lead to sudden reduction in alcohol intake, might be the other cause.

Clinical Features

Include drowsiness, confusion, tachycardia, and tachypnoea, and may progress to Kussmaul's breathing and coma.

Diagnosis

- Blood alcohol level is typically not high.
- Slight elevation of blood glucose.
- Weakly positive ketone assay.
- Marked elevation of serum ketones.
- Hypokalaemia.
- Hypochloraemia.

Treatment

- Infusion of normal saline with dextrose
- Potassium supplementation
- Thiamine (50-100 mg).

Delirium Tremens

Delirium tremens is medicolegally important toxicological problem as it has an element of unsoundness of mind due to *acute insanity* associated with chronic alcoholism, is the most dangerous complication of alcoholism.[7]

Causes

It is a dramatic onset of disordered mental activity due to long continued action of alcohol in chronic alcoholics when its consumption is suddenly stopped[18] for 3 to 5 days. Certain other factors that might precipitate delirium tremens are:[7]

- Temporary excess consumption or sudden withdrawal of alcohol in an alcohol addict.
- Shock on severe trauma such as fractured bone or undergoing surgery in a chronic alcoholic.
- Acute infection in chronic alcoholic.

Signs and Symptoms[10]

- It is characterised by clouding of consciousness, disorientation, and loss of recent memory.
- There are vivid hallucinations, which are visual, but also sometimes auditory in nature.
- There is severe agitation with restlessness and shouting, tremors and truncal ataxia.
- The condition is characterized by an acute attack of insanity in which the main findings are:
    - Coarse muscular tremors of the face, tongue, and hands.
    - Insomnia, restlessness, loss of memory.
    - Uncontrollable fear/agitation, with tendencies to commit suicide/ homicide/ violent assaults to property, etc.
    - Autonomic disturbances such as sweating, fever, tachycardia, hypertension, and dilated pupils
    - Dehydration and electrolyte imbalance are characteristic.
    - Disorientation of time and place
    - Peculiar type of delirium of horrors due to hallucinations of sight and hearing.

Treatment

- Reassurance
- Diazepam 10 mg I/V initially, followed by 5 mg I/V every 5 minutes until full control, later on switched on to 5-10 mg orally thrice a day to control the feeling of fear/ agitation.
- Thiamine
- Rehydration and correction of electrolyte imbalance.

Medicolegal Importance

When a person is in *delirium tremens*, he/ she is not held responsible for any act done for the reason that one is considered to be mentally unsound during delirium tremens state (Section 84 IPC).

Wernicke-Korsakoff Syndrome

This is a rare form of withdrawal syndrome, which is a combination of *Wernicke's encephalopathy and Korsakoff's psychosis.* Wernicke's encephalopathy is an acute form, characterized by drowsiness, amnesia, ataxia, peripheral neuropathy, horizontal nystagmus, and external ocular palsies. When the recovery from this is incomplete, a chronic amnesic syndrome develops called Korsakoff's psychosis, which is characterized by impairment of memory and confabulations (falsification of memory)

Treatment—For *Wernicke's encephalopathy* give thiamine 50 – 100 mg I.V. daily, infused slowly in 500 ml of fluid for 5-7 days, with fluid replacement**.**

Alcohol and Heart

This includes four conditions namely – *Cardiac dysfunction, alcoholic cardiomyopathy, cardiac conduction* and *hypertension.*[10]

Cardiac Dysfunction

A supraventricular arrhythmia can be induced by drinking binges. Also known as *'Holiday Heart Syndrome'* is developed with more than 6 drinks a day. Atrial fibrillation is the most common arrhythmia, but atrial flutter, atrial tachycardia, junction tachycardia, and multiple atrial premature beats have also been noticed. An isolated episode of atrial fibrillation is often the first complaint. About 10-20 years of high alcohol use (about 200 ml of 86 proof whiskey a day) may be required before cardiac decompensation becomes apparent. Early ECG changes constitute left ventricular hypertrophy with abnormal T-wave and nonspecific ST-T wave changes. Primary ventricular arrhythmias culminating in fibrillation may partially explain why alcoholics die suddenly and unexpectedly.[14] Decreased variability of heart rate – a sign of *cardiac vagal neuropathy* and a factor notorious for increase in risk of death after myocardial infarction – is relatively common finding among men dependent on alcohol. Most episodes of cardiac arrhythmias terminate within 24 to 48 hours either spontaneously or after treatment with beta blockers combined with adequate sedation, rehydration, and treatment of any potassium and magnesium depletion.

Alcohol Cardiomyopathy

It shares certain features with beriberi, heart failure found in malnourished, vitamin-deficient alcoholics: Cardiac chamber dilatation, tachycardia, elevated venous pressure, and peripheral oedema. However, thiamine-deficient patient exhibits a high cardiac output state and warm extremities, while the chronic alcoholic patients have depressed cardiac output and ventricular hypocontractibility. A controlled study suggests that susceptibility to alcoholic cardiomyopathy and myopathy is more pronounced in women than in men.[15]

Cardiac conduction—cardiovascular death is the most important cause of mortality in alcoholics, yet alcohol may protect against ischemic heart disease. QT-interval prolongation in some patients with alcoholic liver disease is associated with an adverse prognosis, especially sudden death.[16]

Treatment of Chronic Alcohol Poisoning

Successful treatment of chronic alcoholism requires careful planning and proper supervision of regime of treatment coupled with sincere intention of the addict to give-up the habit of drinking, which can be successfully carried out in an organized institution or nursing home. The treatment of chronic alcoholism thus comprise of three steps:

- *Psychotherapy,*
- *Treatment of withdrawal*
- *Aversion therapy*.

Psychotherapy

Group psychotherapy of supportive nature is considered to be more effective than individual psychotherapy. *Alcohol Anonymous (AA),* a self-support organisation, plays a vital role. This organisation has a base at USA and started as early as in 1935, has more than 35,000 groups all over the world. There is *no membership fees* to join AA. The only requirement to get the membership is the *'desire to stop drinking'*. Address of the

AA in a given area may be had from the local telephone directory for that area.

Treatment of Withdrawal Syndrome

In addition to treatment mentioned above with every specific withdrawal symptom above, following drugs are also tried with varying degree of success.

- *Carbamazepein* – It has been found useful in treating *alcohol withdrawal*, as well as *delirium tremens*.[19] It does not require any adjunctive medication.[20] It also offers advantage of rapid return to work or early induction into an alcoholism treatment program.[21]
- *Chloromethiazole* – This is a drug of choice in Britain for alcohol withdrawal. It has hypnotic, anxiolytic, and anticonvulsant properties. It is given in a rapidly tapering dose schedule for 6-7 days. However, it is also known that alcoholics rapidly become dependent to this drug.[10]
- *Clonidine* – Yet another newer drug, for alcohol withdrawal symptoms. Preliminary report suggests use of clonidine in a dose of 60-180 microgram/hour intravenously with gamma-hydroxybutyric acid in a dose of 50 mg/kg orally is useful in withdrawal syndrome symptoms.[22,23] However, it requires controlled clinical studies to evaluate the findings.[10]

Aversion Therapy (Alcohol De-addiction Therapy, Alcohol Detoxification Therapy)

Objective: To gradually wean off from ethanol, once the acute symptoms of withdrawal syndrome have been treated successfully.

Methods: Usually needs hospitalisation for a period of several days, under close medical supervision. Effective tackling of insatiable craving for alcohol is to be achieved here by deterrent measures.

Of the several methods the most successful method is *antabuse therapy* by administering a drug [*Available in India as: Antadict (Intas), Esperal (Torrent) and Disulfiram (Intas)*]. Disulfiram has different names all over the globe and they are—Antabuse, Abstensyl (Argentina), Abstynil (Switzerland) Alcophobin (USA), Antabus (Denmark, Germany, The Netherlands, Norway, Spain, Sweden, and Switzerland), Antietil (Italy), Antivitium (Spain), Aversan (Norway), Esperal (France), Refusal (The Netherlands), and Ro-Sulfiram-500 (USA).

Action of Disulfiram: Disulfiram is widely used all over the world as an alcohol deterrent. In spite of large number of individuals maintained on disulfiram and the common occurrence of disulfiram (Antabuse) reactions, reports of overdose are relatively uncommon.

Two different schools of thought claim two different ways for disulfiram to act:

1. According to one school of thought, disulfide molecule (*tetraethylthiuram*), interferes with the oxidative metabolism of ethanol at the acetaldehyde stage, and as a result of which *acetaldehyde accumulates* producing *obnoxious* symptoms which are:
   - — *Gastrointestinal tract* – Abdominal discomfort, Nausea, vomiting, etc.
   - — *Central nervous system* – Mental confusion, headache, weakness, a reeling sensation/ feeling about to fall and blurring of vision.
   - — *Cardiovascular system* – Chest pain, syncope, hypotension, tachycardia, arrhythmias.
   - — *Respiratory system* – Tachypnoea
   - — *Skin* – Perspiration, flushing, pruritus.
2. According to another school of thought, it is the metabolites of disulfiram, *diethyldithiocarbamate* and *carbon disulphide,* which produce all the *obnoxious symptoms*.

Therapeutic Dose

The recommended dose is scheduled over 5 days followed by alcohol challenge test as narrated below:

Manufacturer's recommendation—in USA:[24]

1. One to two weeks use of disulfiram orally 500 mg/day.
2. Then 15 ml (1/2 Oz) of 100-proof whiskey taken orally slowly, not to exceed 30 ml (1 Oz)
3. Patient should be hospitalized or have equivalent facilities available including oxygen.

Manufacturer's recommendation—Overseas:[25]

First day	: 4 tablets (800 mg) of disulfiram
Second day	: 3 tablets (800 mg) of disulfiram
Third day	: 2 tablets (800 mg) of disulfiram
Fourth day	: 1 tablet (800 mg) of disulfiram
Fifth day	: Challenge dose, 15 to 30 mL pure ethanol.

Brewer's Recommendations[24]

Give disulfiram on a consistent dose for 5 days to 2 weeks. *Alcohol challenge* is then done as an outpatient procedure. Patient is asked to take 12 ml brandy (5 ml ethanol) on empty stomach. A reaction of *flush* on the abdomen will appear in 20 minutes. If no reaction (flush) is observed, patient is instructed to have an additional 25 ml of brandy (approximately 10 ml of ethanol). This will usually produce a noticeable *flushing* with *tachycardia* and *slight fall in blood pressure*. If there is no reaction, *repeat the test* after 1 week with a higher antabuse dose.

DRUNKENNESS

Definition

The British Medical Council has defined this as: the person concerned must be so much under the influence of alcohol, so as to have lost control of faculties to such an extent which renders him unable to execute safely the occupation in which he was engaged with, at the material time.[1-4,6,7]

Procedure of Examination for Drunkenness Certification

Objectives

To decide whether:

- The subject is under the influence of alcohol
- His condition is due to illness or injury
- It is safe for him to be detained in Police Station, or he should be admitted to a hospital.

Consent

It is better to obtain informed written consent, prior to examination. However, remember following options also:

- If the consent is denied, doctor can use *reasonable force* for physical examination (Section 53-A) of Cr PC, when the requisition for examination is from an officer not below the rank of Police Sub-inspector.
- If the person is unconscious or intoxicated and is unable to give the consent, collect the blood and urine samples for analysis and treat him first. The doctor must admit him/her to the hospital till he turns normal and sober. Never divulge the findings of such patients to police without the consent, unless it is very vital.

Preliminary Data

At the outset note all preliminary data of the patient, such as—

- Name, age, sex, occupation, address
- Time of examination (Commencement and completion of examination)
- Two identification marks
- Person escorting (If the escort is police constable, note his number, name of the Police Station).

History

- Elicit history of drinking alcohol. Find out the time of conumption, the quantity consumed and its brand/the type.
- Ask for past history of any illness/diseases/disabilities he suffered from and any therapeutic preparations/drugs he has been taking for the same. Record its name and the dose taken and for how long he had been taking it.

Physical Examination

The drunkenness is diagnosed by taking proper history and set of signs and symptoms elicited by physical examination and by various tests, laboratory investigations, as given below:

General behaviour—General deportment/the way a person behaves toward other people.

State of the dress—shows spilling over the dress.

Speech—Normal/ slurred/ thick. Blurring of certain components is the earliest sign of inco-ordination. For example, ask him to say: "*British constitution*", "*truly rural*" and person who is going for inco-ordination will be unable to pronounce it properly and will go on attempting to say it completely or correctly.

Memory and mental alertness—Check for the awareness of recent events, time, etc. Ask him to solve simple sums of addition, substraction, etc. An inability to solve the same confirms the impairments of the faculties by alcohol.

Writing—Drunkenness makes the person take more time to write and also may show omission of certain letters/words, etc.

Pulse—It will be rapid, full and bounding.

Temperature Subnormal.

Skin It will be dry/moist, flushed/pale.

Mouth

- Tongue will be dry, furred or bitten
- Breath smells of alcohol.

Eyes

- Eyelids are swollen, red
- Conjunctiva—congested
- Pupils—dilated initially, but contracts later in coma
- Convergence test: negative (difficulty in convergence)
- Strabismus: Positive
- Nystagmus: Positive (strongly proves drunkenness).

Gait

Gait is disturbed. Watch the movement and timing of movement, etc. Alcoholic gait will be usually staggering gait.

Muscle co-ordination

Usually it is lost and is confirmed by various tests such as:

- *Finger nose test*—asks the patient to touch the tip of the nose by an outstretched hand closing the eyes. Inabilities to perform suggest impaired coordination.
- *Lighting the cigarettes with matchbox*—again for drunkenness condition, it is a difficult exercise.

Reflexes

- All reflexes are sluggish
- Plantar may be extensor/flexor type.

Laboratory Investigation

Always check the blood and urine concentration of alcohol, by collecting and sending the samples to forensic science laboratory.

Blood collection and preservation

- Collect the blood from antecubital or femoral vein
- Use soap and water to cleanse the site to be venepunctured (do not use surgical spirit as it contains alcohol)
- Preserve by adding 5 mg of sodium fluoride for 10 ml of blood and refrigerate the bottle closing tightly.

Urine collection and preservation

- Full quantity of urine passed must be collected
- Ask him or her to pass urine in a toilet where there is no water source nearby (this is to prevent him or her from adding water to urine collected and dilute the alcoholic concentration)
- Preserve by adding a crystal of sodium chloride or thymol.

Breath analysis

- Ask the person to blow into a rubber balloon
- Analyze the breath by a breath analyzer (*drunkometer, intoximeter, alcometer*).

Differential Diagnosis

Before diagnosing and opining the given case as a *drunkenness case*, it is better to rule out certain clinical emergencies, which may *mimic* the manifestations of drunkenness. These are:

- Severe head injuries (*postconcuss ional syndrome*)
- Metabolic disorders like—hypoglycaemia, uraemia, diabetic coma, etc.
- Drug overdosage—insulin, barbiturates, morphine, etc.
- Neurological conditions like—intracranial tumours, epilepsy, Parkinsonism, etc.
- Psychological disorders such as—hypomania
- In high fever
- Exposure to carbon monoxide gas poisoning.

Final Opinion

After examination opinion can be drafted with any *one* of the following three statements:

- He/she has not consumed alcohol
- He/she has consumed alcohol but is not under the influence of it
- He/she has consumed alcohol and is under its influence.

Authentication

Doctor should *sign* the report; mention his/ her name, registration number, designation and address (use a rubber stamp) at the end.

Drunkenness and Medicolegal Importance

The clinical diagnosis of drunkenness will not depend upon single sign like odour of alcohol in breath but a combination of signs and symptoms peculiar to this condition as already discussed above. Following discussion explains the medicolegal aspects of drunkenness:

- *Drunkenness and medical practice*
- *Drunkenness and disorderly conduct*
- *Driving, drunkenness and law*
- *Drunkenness and criminal responsibility*
- *Drunkenness and contractual obligations/wills*

Drunkenness and Medical Practice

On *no account*, a doctor in a drunken state should treat a patient. If he performs a surgery under drunken state and if the patient dies on the operation table, the doctor can be prosecuted for *rash and negligent act* (Section 304 A, IPC). He can be sued for '*damages*' for causing physical injury or death of the patient. Case is considered as *infamous conduct* and punished by *penal erasure.*

Drunkenness and Disorderly Conduct

If a drunken person behaves in disorderly manner or is unable to take care of himself in a public place, he will be liable to *imprisonment* and *fine* for being drunk and disorderly as per *Police Act of the State*. The term drunk will not connote the same meaning so far law and punishment are concerned; as for example, *law will not treat a drunken driver of motor vehicle to be in par with a drunken bullock cart driver or a drunken pedestrian.*

Driving, Drunkenness and Law

- Alcohol affects driving efficiency enormously; so much that safe driving is seriously affected. This is due to a drop in *reaction time* and the drunken driver will take 15-20 per cent more time to press the brake or change the gear, when required.
- Alcohol creates increase in false and unjustified *confidence*. Here when the driver thinks he is driving exceedingly well and with due caution, his driving is worst without proper precautions such as he skips over traffic signals, tries to overtake other vehicles dangerously, etc.
- Alcohol impairs concentration and dulls the power of judgement. He cannot concentrate on his duty properly, and gets diverted off his attention more often.
- Alcohol affects vision and driver cannot see properly, as his *visual acuity* gets diminished depending upon BAC, as he drives with sunglasses on in twilight of darkness. With alcohol concentration > 100 per cent stronger illumination will be required for distinguishing objects. Strong beam of lights from the opposite vehicles often dazzles him. The peripheral vision is also affected and a as a result driver cannot make out the vehicles coming from the sides or pedestrians standing on the road sides. This renders risk not only to himself but also to others, and may be punishable under *Section 107, Motor Vehicle Act 1949* entailing *imprisonment up to 3 months or a fine up to Rs. 500.00.*[1-4, 6, 7,18]

Drunkenness and Criminal Responsibility[7]

The drunken person can only be absolved from the criminal responsibility, if it can be proved that under the effect of drinking, he was unable to understand the nature and consequences of his act at the time of doing it, vide *Section: 85 IPC,* provided the drink was given to him without his knowledge and/against his will.

In cases of voluntary drunkenness, where the incapacity to form the intention cannot be proved definitely, he may be punished, as if he had the intention to commit the crime vide Section: 86, IPC, criminals often take alcohol to steady reduce their nerves to commit the crime.

Drunkenness and Contractual Obligations/Wills

A drunken person, if is in such a state of intoxication, as not to understand the nature or terms of contract or its effect on his interest, then he will not be bound by such contract vide Indian Contract Act 1872. If drunkenness affects sound disposing state of mind, then the subject loses his power of testamentary capacity.

Medicolegal Importance

- *Uses*—Various uses of alcohol are enumerated in Table 34.8 are self-explanatory on popularity of alcohol.
- *Incidence of poisoning*—Usually *accidental*. It could be *suicidal* rarely, by consuming along with other poisons such as barbiturates, organophosphorus compounds, etc.
- *Acute intoxication* is usually by overdrinking of alcoholic beverages.
- *Chronic alcoholism* or *alcohol addiction* may result in medicolegal problems such as: Road traffic accidents, industrial accident, problems of marital relationship, etc.

METHYL ALCOHOL

Synonyms

Methanol, Wood spirit, Wood naphtha, Colonial spirit, French polish, Denatured spirit.

Composition

Methyl alcohol contains three ingredients-90 per cent by volume ethyl alcohol, 9.5 per cent wood naphtha, and 0.5 per cent crude pyridine.

Physical Properties

Methyl alcohol is colourless, clear, volatile liquid with a characteristic odour (felt to be similar to that of ethyl alcohol) and a bitter taste.

Absorption and Metabolism

Methyl alcohol is rapidly absorbed through the stomach and intestines, and also through the lungs and skin. Though action resembles that of ethanol to a great extent, its rate of oxidation is 1/5th that of ethanol. With repeated small doses it tends to accumulate in the blood. 80 mg/100 ml of blood is dangerous level.

Methanol is oxidized in the liver to *formaldehyde*, which is 33 times more toxic than methanol. Formaldehyde in turn is oxidized to *formic acid*, which is 6 times more toxic than methanol. Formic acid is responsible for associated *metabolic acidosis* and the *retinal toxicity*.

Formate may also inhibit the cytochrome oxidases chain increasing *lactate production* and *metabolic acidosis*. It is distributed in the tissues according to their water content, and a high concentration is found in *vitreous body* and *optic nerve*.

Thus, the metabolized products of methyl alcohol, namely *formaldehyde* and *formic acid*, have specificaction on optic nerve producing *optic neuritis* and *atrophy* resulting in *permanent blindness*.

Elimination

Methanol is excreted unchanged from lungs to an extent of 80 per cent while kidneys excrete another 3-5 per cent in urine unchanged.

Signs and Symptoms

These are same as ethyl alcohol, resulting into *drunkenness*, with not so prominent inebriation, but effects are much prolonged compared to ethanol. Symptoms may appear within an hour, or within 24 hours of consuming methanol.

General: They comprise of headache, dizziness, and vertigo. There is marked muscular weakness.

GI system: Include nausea, vomiting and pain or severe cramps in the abdomen. Liver may show severe toxic effects. Absence of GI symptoms does not rule out serious toxicity. Pancreatitis, as defined by elevated serum amylase, occurs commonly [26], seen in two-thirds of recent series of cases.[27]

Cardiovascular system: This includes depressed cardiac action and hypothermia.

Respiratory system: Breath usually emits alcoholic odour. There may be dyspnoea and cyanosis. Death usually occurs due to respiratory failure.

Central nervous system: The effects on CNS are more intense and persistent than with ethyl alcohol. There may be delirium and coma, which may last for 2–3 days.

Excretory system: Kidney shows severe toxic effects, with strongly acidic urine, which may contain acetone and trace of albumin.

Acidosis is caused by inhibitory effects on oxidative enzyme system produced by methanol with the resultant accumulation of lactic acid and other unidentified acids. Severe non-diabetic acidosis in suggestive of methyl alcohol poisoning.

Eyes: All changes enumerated below are attributed to *optic neuritis* and *atrophy* from the effects of *formic acid* on *optic nerve* with gross and microscopic degenerative changes in the retinal ganglion cells and optic disc:

- The pupils are dilated and fixed.
- Visual disturbances comprise of:
  - *Photophobia*
  - Blurred or misty vision (*flashes* and *snow-storm* or *snow field* vision)
  - Seeing spots
  - Central and peripheral *scotoma*
  - Decreased light perception
  - Concentric diminution of visual fields for colour and form
  - Sudden failure of vision or complete blindness

In fatal cases convulsions are usual as a terminal event, and death occurs from respiratory failure.

Fatal dose: 60 to 200 ml.

Fatal period: 24 to 36 hours. May be delayed for 2-4 days.

Toxicity rating: 3.

Treatment

- Keep the *airway clean*. Place the patient in a left lateral decubitus position with head down to avoid aspiration of the vomit.
- *Gastric lavage* with 5 per cent sodium bicarbonate solution, espoused by leaving another half litre in the stomach at the end. Activated charcoal can reduce the fatality, by reducing the absorption of alcohol from the stomach and intestines. It also acts by creating a concentration gradient in favour of movement of alcohol and its metabolites back into the gut.
- *Antidote* is ethanol (ethyl alcohol-50%) in a dose of daily 0.75 to 1 ml per kg body weight, every two hours for 3 to 4 days. It is preferably given intravenously, as 10 per cent solution starting with a loading dose of around 500 ml given as an intravenous infusion. (*To prepare 10% ethanol, withdraw 100 ml of 5% dextrose from 1 litre dextrose bottle and substitute it with 100 ml of absolute alcohol after filtering through filter paper of 0.22u*). It acts combining with alcohol dehydrogenase enzyme, thereby, blocking the metabolism of methyl alcohol, which is then excreted in less harmful unmetabolized form. Intravenous infusion is to be continued till blood methanol falls below 25 mg%. However, during the treatment it is necessary to maintain the serum ethanol level at 100-150 mg%, which is certainly towards better prognosis. Oral administration of ethanol is not desirable as to avoid gastritis.
- *Haemodialysis* is the treatment of choice in case of severe poisoning. It can effectively reduce the half-life of methanol from 40 hours to about one hour. However, peritoneal dialysis or haemoperfusion is not recommended.
- Treat *acidosis* – 50 to 75 mg of folinic or folic acid, 4 hourly can help in the elimination of formic acid, decreasing metabolic acidosis and reducing the symptoms. Giving soda bicarbonate I/V is also effective in combating metabolic acidosis.
- *Administer crystalloid* therapy, dextrose, thiamine, and phosphate—correction of potassium and magnesium defects comprise basic correction of alcoholic ketoacidosis.
- *Eye care* – Protect the eyes from bright light by keeping them covered.
- Symptomatic measures.

Postmortem Appearance

- Prominent *cyanosis* of *upper part* of the body
- Blood presents with *absence* of *postmortem clotting*
- Stomach and duodenum is *congested* and *inflamed* with *tiny haemorrhagic spots*
- Pancreas may present with *haemorrhagic pancreatitis*[10]
- Lungs show *congestion, oedema, emphysema*, and *desquamation of alveolar epithelium*
- Brain is *oedematous* and with local *petechial haemorrhages*
- Liver and kidneys present with toxic damage. Liver may also show *fatty changes* or early *necrosis*, while in the kidneys there may be *tubular degeneration*.
- Urinary bladder often shows—*congestion*
- Eyes—may show *retinal oedema* or *optic atrophy.*

Preservation of the Viscera

It is to be done in saturated sodium chloride solution.

Chemical Analysis

Methyl alcohol and formic acid is reported in all organs, blood and urine. Formaldehyde cannot be detected for the simple reason that it can combine rapidly with proteins and get oxidized into formic acid.

Medicolegal Importance

- An anecdotal report suggests that methanol poisoning may follow intentional sniffing.[28]
- It is present in certain home-made beverages, antifreeze, paint removers, leather dyes, varnish and shellac, windshield washing fluid, and embalming fluid.
- Circumstances of poisoning incidences are mostly due to accidental consumption of methyl alcohol *adulterated* with cheap liquor by the poor people. This is mainly because it is presumed to give the kick effect earlier with a smaller dose, compared to other ethyl alcoholic beverages. At times it is also serves as intoxicating beverage when ethyl alcohol is not available.

ISOPROPANOL *(Isopropyl Alcohol, IPA)*

Classification

Isopropanol is an inebriant neurotoxic substance.

Properties

IPA is a colourless, clear, volatile liquid with aromatic odour and a burning bitter taste, mainly now used as an industrial solvent. It can be an ingredient of antifreeze, liquid detergent, paint and varnish remover, racing motor fuel, etc.[1,10]

Absorption and Action

It is a central nervous depressant and has twice the CNS depressant potential than ethanol.[1] Oral ingestion of approximately 30 ml (1 oz) of 70% isopropyl alcohol (IPA) can lead to peak serum concentration of 28 mg/dL in 30 minutes. Skin absorption is relatively little, but can contribute to toxicity with prolonged contact.[10]

IPA is said to act by its metabolites namely —acetone, acetic acid and formic acid, formed in the liver. In a diabetic with ketoacidosis, and in starvation with high levels of acetone, acetone can get converted in to isopropyl alcohol.

Signs and Symptoms

It can produce nausea, vomiting, pain abdomen, hypotension, tachycardia, headache, confusion, coma and death when not treated. On examination, there will be acetone like odour in breath and urine examination can turn positive for acetone.

Fatal dose: About 250 ml.

Treatment

- Gastric lavage if brought early
- Haemodialysis/peritoneal dialysis
- Cardiac and respiratory monitoring
- Symptomatic/supportive line of treatment.

Postmortem Appearances

Same as in ethanol poisoning.

Medicolegal Appearances

- Often coloured blue in the *hospital* to distinguish from other colourless liquids, and this has lead to the designation as '*blue heaven*' by abusers.[29]
- Poisoning is usually accidental as an industrial exposure cases.
- Suicidal/homicidal cases are yet to be reported.
- An alcoholic addict may consume preparations of this having isopropanol in it.

ETHYLENE GLYCOL

Classification

Ethylene glycol is an inebriant neurotoxic substance.

Properties

Ethylene glycol is a colourless, nonvolatile liquid with bittersweet taste.

Action

Toxic action is mainly due to the metabolites-glycolic acid, lactic acid and oxalic acid.

Signs and Symptoms

Though symptoms resembling mild drunkenness appear at first, may present with nausea, vomiting, convulsions and coma; cardio respiratory symptoms like tachycardia, tachypnea and congestive heart failure develop in 12 to 14 hours. Death may supervene within a day or so, because part of the metabolic pathway converts to glycol through formic acid. Renal failure occurs, with envelope shaped crystals of calcium oxalate in the kidney tubules and interstitial tissues, resulting in tubular necrosis within 24 to 72 hours. Liver damage may also occur.

Fatal dose: 100 to 200 ml.

Fatal period: 24 hours or so.

Treatment

- Gastric lavage if brought early
- Haemodialysis/peritoneal dialysis
- Ethyl alcohol (same as for methanol poisoning)
- Pyridoxine (50 mg) and thiamine (100 mg) given by IM route can hasten metabolism of ethylene glycol.

Postmortem Findings

Cerebral oedema, toxic damage to liver and kidneys.

Medicolegal Importance

- Extensively used as industrial solvent, coolant and antifreeze, agent for motor engines.
- The glycols are sometime misused either for suicide or more often, as a cheap substitute for alcoholic drinks.
- Accidental ingestion also occurs, usually because glycols have been put into a soft drink bottle

CHLOROFORM

Classification

Chloroform is an inebriant cerebral poison.

Properties

Chloroform is a heavy, colourless, volatile liquid with a strong odour and a burning sweet taste.

Action

Chloroform acts as an anaesthetic and respiratory depressant.

Signs and Symptoms

- Oral ingestion—symptoms are similar to alcohol. However, there may be burning pain in the mouth, throat and stomach and vomiting. Within ten minutes, unconsciousness and coma with slow stertorous breathing occurs. Pupils are dilated and pulse feeble, rapid and irregular.
- Nasal inhalation—produces four stages: Analgesia, excitement, anaesthesia and paralysis. All the muscles are relaxed, pupils dilated and reflexes are lost completely. Body temperature is subnormal. Death is due to cardiac or respiratory failure.

Fatal dose: 30 ml by mouth (>0.04% in blood).

Fatal period: 30 minutes.

Treatment

- Orally ingested cases—gastric lavage, demulcent drinks, and stimulants and symptomatic measures.
- Nasally inhaled cases—give artificial respiration, oxygen and cardiac stimulants.
- Maintain body warmth.

Postmortem Appearances

They are not characteristic except marked congestion and chloroform odour. Internal examinations reveal:

- Irritation of the gastrointestinal and respiratory mucosa
- Chloroform odour for contents of the stomach, in the serous cavities, lungs and brain
- Findings of asphyxia predominate when death is due to inhalation poisoning.

Medicolegal Importance

- Chloroform was the first general anaesthetic used, but presently is being used as an industrial solvent

- Poisoning is usually accidental when liquid chloroform is swallowed accidentally.
- Delayed chloroform poisoning was common even after 2-4 days of recovery from general anaesthesia.

ETHER

Classification

Ether is an inebriant cerebral poison.

Properties

Ether is a colourless, volatile, highly inflammable liquid with penetrating ethereal odour and sweetish pungent taste.

Action

Ether acts as an anaesthetic and respiratory depressant.

Signs and Symptoms

- Oral ingestion—burning of throat, oesophagus and stomach, nausea, vomiting, followed by inebriation as with alcohol
- Nasal inhalation—same as chloroform.

Fatal dose: 30 ml by mouth.

Fatal period: May be immediate due to syncope during anaesthesia—rarely.

Treatment: Gastric lavage, demulcents, stimulants and anticonvulsants. Maintain blood pressure.

Postmortem Appearances

- Smell of ether on opening the body
- Congestion of proximal gastrointestinal tract when taken orally
- Findings of asphyxia, oedematous lungs and brain. Abundant mucus in the respiratory tract.

Medicolegal Importance

- Accidental deaths are common while administering anaesthesia (if dies due to anaesthesia—report to the police immediately).
- An alcoholic addict may consume this when he or she is deprived of alcohol.
- Presently, it is not used as anaesthetic, but as an industrial solvent.

CHLORAL HYDRATE ($C_2H_3CbO_2$)

(Trichloroacetaldehyde)

Classification

Chloral hydrate is a sedative (induces calming effect on CNS) and hypnotic (induces sleep).

Physical Properties

- It is a crystalline substance
- Taste—peculiarly bitter sweet, but nauseating
- Odour—aromatic.

Action

- In small dose—acts as a hypnotic (0.3 to 1.2 gm)
- Large/toxic dose—can paralyze the vital centres and CNS
- Absorption—well absorbed from gastrointestinal tract.

Signs and Symptoms

Signs and symptoms can occur in acute and chronic forms.

Acute Poisoning

Ingestion of fatal doses can result in burning sensation in the throat, followed by nausea and vomiting. Later drowsiness develops, merging to unconsciousness with anaesthesia; loss of reflexes, muscular relaxation, etc. followed by depression of medullary centres, resulting in fall of BP, respiratory rate, convulsions and ultimately death. Pupils are usually constricted and '*pinpoint*.' Since chloral hydrate is radio-opaque, radiograph can help in diagnosing oral massive ingestions.

Chronic Poisoning (Chloral Hydrate Addiction)

Causes—Frequent consumption of the drug over a long period can develop following manifestations:

- Digestive symptoms such as epigastric pain, nausea, vomiting and severe gastritis
- Vasomotor skin disorders producing erythematous rashes
- Nervous manifestations such as neuralgia, tremors, convulsions, etc.
- Psychic phenomena of depressive character, rarely resulting in severe depression, delirium tremors, etc.

Fatal dose: 5 to 10 gm. However, recoveries are reported even with large doses.

Fatal period: 8 to 12 hours, but may be delayed for 2 to 3 days.

Treatment

- Gastric lavage with water containing an alkali which can decompose unabsorbed chloral hydrate
- Maintain body warmth
- Give:
  - Cardiorespiratory stimulant
  - Artificial respiration
  - Intravenous hypertonic glucose.

In chronic poisoning

- Withdrawal of the drug
- Put the patient on high protein and carbohydrate, but low fat diet.

Postmortem Findings

- Postmortem findings are usually that of asphyxia
- Stomach contents will emit peculiar odour of chloral hydrate
- Chronic-poisoning cases will show fatty degeneration of liver, kidney, heart and other internal organs
- The poison deteriorates rapidly after death from the body and hence, the viscera are sent immediately for chemical analysis. The results may be negative; unless the analysis is done immediately (other similar poison is sodium nitrite).

Medicolegal Importance

- It is used as a hypnotic therapeutically and may be responsible for many deaths from inadvertent overdosage.
- Rarely used as suicidal agent
- It cannot be used for homicide due to smell and taste, though can be given with beer.
- *Dry wine* is a combination of chloral with alcohol is used in certain parts of Punjab to produce sleep
- *Knock-out drops or Mickey Finn*—is an illicit liquor adulterated with chloral to potentiate intoxication or for the criminal use to facilitate robbery, rape, etc. and make their victims helpless
- Abuse of drug—chronic use can lead to drug addiction.

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BARBITURATES

SLANG NAMES

Barbs, goofballs, downers, yellow jackets, red devils, reds and blues, rainbows, pinks, block busters; Christmas trees are some of the popular slang names.[1]

Introduction

Barbiturates are basically derived from *barbituric acid*, the oldest being veronal barbitone. Even though this group of drugs were used extensively in the past, benzo-diazepines have replaced the use of many of the barbiturates; they are still available and abused. Using barbiturates in conjunction with alcohol is especially dangerous; alcohol is a central nervous system (CNS) depressant so the harmful effects of each are multiplied. Overdose deaths are more frequent when alcohol and barbiturates are mixed, whether accidentally or deliberately. One feature remains common to all barbiturates: depending on tolerance, there is only a slight difference between a dose that produces sedation and a dose that may cause death.[1-5]

Classification

Barbiturates are sedative, hypnotic type of cerebral poisons, used in medical practice as sedatives, hypnotics, anesthetics, and antiepileptics or in strychnine poisoning cases, etc. Barbiturates are functionally grouped into *two groups* such as *long-acting* and *short-acting* agents; the latter further consists of three types: *ultrashort, short*, and *intermediate-acting agents*. Barbiturates are thus classified into four types, depending on their time of onset and duration of action.[1,5]

- ***Long acting***—acts within 2 hours and duration of action lasts for 6 to 12 hours, e.g. *barbitone, phenobarbital, mephobarbitone, methyl phenobarbital, diallylbarbiuric acid, etc.*
- ***Intermediate acting***—acts within 1/2 to 1 hour and the duration of action lasts for 3 to 6 hours, e.g. *amylobarbitone, butobarbitone, probarbitone sodium, amobarbitone, aprobarbital, vinbarbital, allobarbitone, etc.*
- ***Short acting***—acts within minutes and the duration of action lasts <3 hours, e.g. *cyclobarbitone, pentobarbitone, amobarbitone, aprobarbitone, butobarbitone, hexabarbitone, seconal, ortal, etc.*
- ***Ultra-short acting***—acts immediately and the duration of action lasts for < 15-20 minutes. Since they act immediately and the action passes of within short time, they are basically used as anaesthetic agent, e.g. *thiopentone sodium, methohexitone, pentothal sodium, hexobarbital sodium, kemithal sodium, thiamylal sodium,* etc.

Absorption, Distribution and Excretion[1-5]

Barbiturates whenever used as sedative hypnotics are administered orally. Intravenous administration is usually reserved for management of status epilepticus or induction/ maintenance of general anaesthesia. Barbiturates are absorbed from the gastrointestinal tract, including rectum, and from the subcutaneous tissues.

After absorption they are eventually distributed widely in the body fluids. Metabolism of barbiturates occurs by oxidation in the liver resulting in the formation of alcohols, ketones, phenols, or carboxylic acids, which are excreted in urine as such or in the form of glucuronic acid conjugates. About 25 per cent of phenobarbitone is excreted unchanged in urine.

Mechanism of Action[1,3,5,6]

Barbiturates act as a *depressant* at all levels of CNS. However, degree of depression can be altered from mere tranquility to deep coma with alteration of dose. It can also act synergistically with analgesics and such other drugs, e.g. with alcohol, it increases the action of alcohol. The drug has cumulative effects too, as its metabolism and excretion is very slow (*cumulative poison*). Therefore, barbiturates are not indicated in cases with hepatic and renal damage.

Barbiturates bind to specific sites on gamma-aminobutyric acid (GABA)-sensitive ion channels found in the central nervous system (CNS), which is the major inhibitory neurotransmitter in the CNS. Barbiturates also block glutamate (excitatory neurotransmitter) receptors in the CNS.

Compared to long-acting agents, short-acting agents are more lipid soluble, more protein bound, have a higher pKa, a more rapid onset and shorter duration of action, and are metabolised almost entirely in the liver to inactive metabolites (which are excreted as glucuronides in the urine). Long-acting agents, which are less lipid soluble, accumulate more slowly in tissue and are excreted more readily by the kidney as active drug. For instance, urinary excretion accounts for 20-30 per cent of phenobarbital and 15-42 per cent of primidone elimination (both long-acting agents).

Short-acting agents have an elimination half-life less than 40 hours compared to long-acting agents, which have an elimination half-life longer than 40 hours.

Barbiturates stimulate the hepatic cytochrome P-450 mixed function oxidase microsomal enzyme system; thus, barbiturates affect the drug levels of medications that are dependent on this system (e.g. coumadin).

Central Nervous System Effects

Barbiturates mainly act in the CNS and, as a consequence, affect other organ systems. Direct effects include sedation and hypnosis at lower dosages. The lipophilic barbiturates, such as thiopental, cause rapid anesthesia because of their tendency to penetrate brain tissue quickly. Barbiturates all have anticonvulsant activity because they hyperpolarize cell membranes; therefore, they are effective adjuncts in the treatment of epilepsy.

Pulmonary Effects

Barbiturates can cause a depression of the medullary respiratory centre and induce a respiratory depression. Patients with underlying chronic obstructive pulmonary disease (COPD) are more susceptible to these effects, even at doses that would be considered therapeutic in healthy individuals. Barbiturate overdose fatality is usually secondary to respiratory depression. Pulmonary embolism has also been reported following barbiturate over dosage.

Cardiovascular Effects

Cardiovascular depression may occur following depression of the medullary vasomotor centres; patients with underlying congestive heart failure (CHF) are more susceptible to these effects. At higher doses, cardiac contractility and vascular tone are compromised, which may cause cardiovascular collapse.

Mortality/Morbidity

Mortality rates range from 1-10 per cent. Fatality associated with barbiturate overdose is rare, but life-threatening complications are abundant. Morbidity by complications includes pneumonia, pulmonary oedema, shock, hypoxia, acute renal failure and coma.

Pregnancy

Barbiturates freely cross the placenta and can have adverse effects on the fetus,[2] such as decrease in fetal intelligence, possible addiction, and possible withdrawal. Over activity, visible tremors, hypertonicity, hyperphagia, and vasomotor instability characterize neonatal withdrawal syndrome, while withdrawal begins 4-7 days after birth and may last up to 4 months.

Signs and Symptoms

According to recent views, the patient with barbiturate toxicity may present with any or all of the symptoms enumerated in Table 34.14.

It may also present as acute intoxication, barbiturates *automatism* and chronic intoxication. Each one of them is discussed individually.

Acute Intoxication[8]

Early manifestations There will be giddiness, ataxia, and slurred speech - initially, followed by stupor. The limbs become flaccid, reflexes are lost, pupil reaction varies, and there may be diplopia. Pupil may show *hippus.*

Late manifestations As the poisoning advances the face tends to become cyanotic progressively, the respiration becomes slow, sighing and periodic (Cheyne-Stokes) which soon turns rapid and shallow. There will be fall of blood pressure. Body temperature will be subnormal. Oliguria may develop with urine containing albumin and sugar.

Coma and death Finally coma will supervene as the late manifestation leading to death due to respiratory failure, as a result of oedema lungs, bronchopneumonia, cardiac failure, etc. all of which may occur suddenly and unexpectedly.

Stage of recovery Occasionally patient may recover gradually from coma. On recovery patient will have physical weakness, low blood pressure, anaemia, etc.

Differential Diagnosis

Poisoning due to carbon monoxide, meprobamate, etc. However, other possible differential diagnoses, which may have to be ruled out, are tabulated in Table 34.15.

Barbiturate Automatism

Taking barbiturate tablets repeatedly to get sleep out of mental/ sleepy confusion, compelling him to take more and more tablets, ultimately resulting in acute toxicity is called *barbiturate automatism*. Although initially it was believed this contributed to a quarter of 488 cases of barbiturate intoxication,[10] subsequently the scienfic studies have failed to explain the facts or observations.

Table 34.14: Signs and symptoms due to barbiturate toxicity

Systems	*Signs and symptoms*
CNS	Lethargy, coma, hypothermia, vertigo, slurred speech, ataxia, decreased deep tendon reflexes
Eyes—pupil	Eye present with—Nystagmus, strabismus. Patient may complain of diplopia. Pupillary reaction usually varies and initially they are constricted to light, while late in the course of poisoning, hypoxic paralytic pupillary dilatation occurs. Pupil also may present with alternate dilatation and contraction called *hippus*.
Psychiatric	Impairment in thinking (e.g. memory disturbances, poor judgment, limited attention span), a cardinal feature, irritability, combativeness, paranoia
RS	Respiratory depression, apnoea, hypoxia
CVS	Tachycardia, bradycardia, hypotension, diaphoresis and shock
GIS	Decreased bowel sounds
Skin	*Barbiturate blisters*[7,8]—These are *bullous lesions* typically found on the hands, buttocks, and knees (Fig. 34.6), at friction areas such as axilla, inner aspects of knee, calf, inter-digital clefts, etc. The blistering once were thought to be sufficiently characteristic for acute barbiturate intoxication as to be diagnostic, are now explained to be due to the dependent skin oedema resulting from poor venous return, consequent to immobility with prolonged unconsciousness or coma. These lesions have also been observed in overdosage with methaqualone, meprobamate, glutethimide, opiate and tricyclic drugs.

Table 34.15: Differential diagnosis of barbiturate poisoning[2]

Causes	*Types*
CNS causes	Drug abuse—Alcohol and substance abuse evaluation, encephalitis, encephalopathy, head injury, hypothermia etc.
Coma due to	Hypothyroidism and myxoedema, hypoglycaemia
Paediatric	Urinary tract infections and pyelonephritis
Shock due to	Carcinogenic and haemorrhagic origin
Poisoning with	Benzodiazepines, carbamazepine, carbon monoxide, clonidine, cyclic antidepressants, gamma-hydroxybutyrate, neuroleptic agents, sedative-hypnotics, etc.

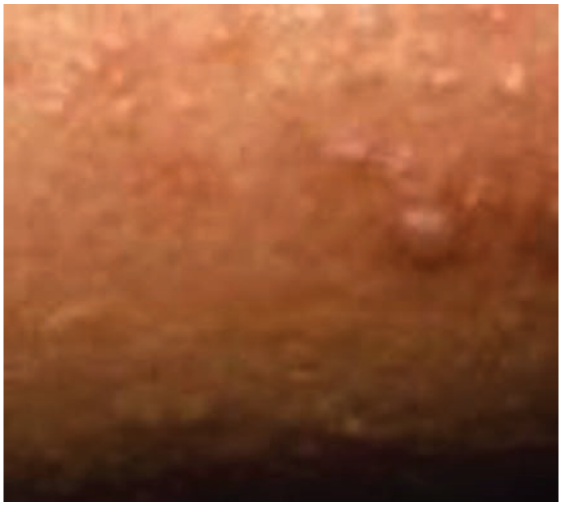

Fig. 34.6: Barbiturate blisters[1]

Chronic Intoxication (Barbiturate Addiction)[11-13]

Barbiturates are some of the most addictive drugs. They are often a substitute for alcohol (as similar effects are produced.) People use Barbiturates to get a sense of euphoria and relaxation. It is illegal to take barbiturates without a doctor's prescription and supervision. However, according to *Sing et al*, incidence of barbiturate poisoning in India has been drastically reduced since mid 1970s.[11]

Signs and Symptoms

- Chronic intoxication will manifest as—apathy, loss of power of concentration, somnolence, vertigo, tremors, ataxia, thick speech, delirium, hallucination (visual), emotional instability, general mental disorientation, etc.
- Urine will show—albumin, sugar and casts microscopically.

Laboratory Tests to Confirm Poisoning

- All barbiturates except thiocompounds give a violet colouration with copper or cobalt isopropylamine reagents.
- Minute quantities of barbiturates can be detected and measured in protein free blood and tissue fluid extracts by ultraviolet spectrophotometery.
- Presence of barbiturates in urine, stomach contents or in scene residue can be detected by TLC (Thin Layer Chromatography), GC (Gas Chromatography) or HPLC (High Pressure Liquid Chromatography) methods.

Assessing the Severity of Poisoning

EEG and bowel sounds are both utilized as an important means for the purpose.

- EEG with *Alpha coma* indicates poor prognosis.
- If the patient is unconscious and if the bowel sounds are not heard it suggests poisoning of severe degree. If the bowel sounds are not heard, normally, it suggests poisoning is not of severe degree.
- If a patient who is in a phase of recovery, if the bowel sounds are heard reasonably early, it is a sign of favourable good prognosis and recovery.

Fatal dose—Fatal dose when taken alone usual fatal dose for different barbiturates is:

- Long acting—3 to 4 gm.
- Intermediate acting—2 to 3 mg
- Short acting—1 to 2 gm

The lethal blood levels for different barbiturates are:

- Long acting—8 to 10 mg per 100 ml
- Intermediate and Short acting—3 to 4 mg per 100 ml

When taken with alcohol—alcohol potentiates the action of barbiturates and thus even a sublethal dose of either can bring about death easily.

Fatal period—24 to 48 hours, however, patient may be in coma for several days and then die.

Toxicity rating—ranges between 4 and 5.

Management of Poisoned Patient

Patients with barbiturate toxicity generally need to be monitored closely and should be in an ICU setting. With proper treatment death rate is rare and it includes:[2,9]

1. Assess the airway and adequacy of respiration and perform ET intubation as necessary.
2. Provide oxygen continuously to improve cyanosis. Artificial respiration may be necessary
3. If the patient is hypothermic, immediately perform a careful rewarming.
4. If the patient has low blood pressure/ in hypovolaemia shock, initiate fluid therapy. Give pressors (e.g. norepinephrine, dopamine) if shock persists or worsens.
5. GI decontamination
   — *Perform GI decontamination* once the airway is protected and haemodynamic stabilisation has been addressed. However, gastric lavage has not proven to be beneficial
   — *Activated charcoal* orally or by nasogastric tube is recommended for all patients with potential barbiturate toxicity.
   — *Induction of emesis* with ipecac syrup is contraindicated in these patients because the depressed neurologic response increases risk of aspiration.
7. Alkalinisation of the urine enhances the elimination of phenobarbital and, likely, other long-acting barbiturates by ion trapping. Urinary alkalinisation is not recommended for short-acting barbiturate toxicity.
   — Enhanced urinary elimination has been well established for phenobarbital. Phenobarbital's low pKa (7.2), higher water solubility, and slow hepatic metabolism and subsequently long half-life allow a larger proportion to be renally excreted.
   — Urinary elimination may be accomplished by an infusion of dextrose and sodium bicarbonate at 200-300 cc/h.
8. If the coma is prolonged, give:
   — I/V fluids
   — Amphetamine sulfate 10 mg, every 30 minutes till improvements occur
   — Prophylactic antibiotics to prevent pulmonary infection and complications
   — *Dialysis/exchange transfusion, etc* are valuable in saving life. Haemodialysis and haemoperfusion enhance elimination of barbiturates (most are well established for phenobarbital). Haemoperfusion is more efficacious than haemodialysis but associated with a higher incidence of complications. Haemodialysis or haemoperfusion is recommended for patients resistant to standard supportive care, in stage IV coma, or with shock, severe hypothermia, renal failure, and pulmonary oedema. Some recommend extracorporeal removal to shorten the duration of coma when patients are apnoeic or have serum concentrations of barbiturate >100 mg/L.

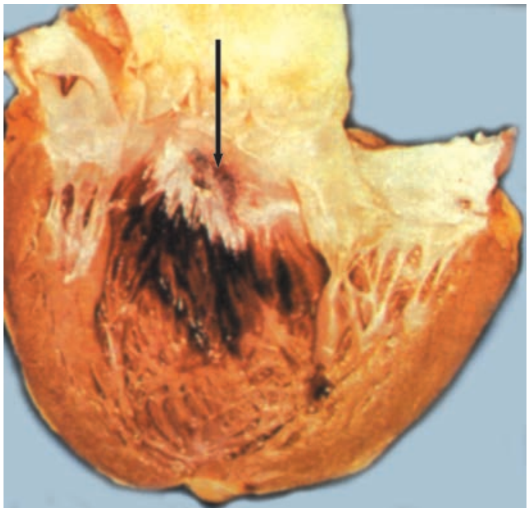

Fig. 34.7: Subendocardial haemorrhage in acute barbiturate poisoning case (arrow)

Treating Chronic Barbiturate Poisoning

- Same as for drug addiction
- Patient may develop withdrawal convulsions after 4 to 5 days of stopping the drug.

Postmortem Findings[5,11,13-15]

- Mainly of *asphyxia* or *comatoasphyxia—peripheral cyanosis*
- Froth exudes from nose and mouth (rule out opium, Tick-20, endrin poisonings and drowning).
- *Barbiturate blisters* at the dependent parts of skin surface such as buttocks, inner aspects or back of thighs, calves and forearms.
- Stomach—may show capsules/tablets/powder. Mucosa may be congested and eroded.
- Lungs—are intensely congested, may show evidence of *bronchopneumonia* or *oedema*.
- Heart—may reveal *subendocardial haemorrhage* (Fig. 34.7).
- Other viscera—congested.
- Urine—may show evidence of barbiturates.

Medicolegal Importance

- Alcoholic and opiate addicts often take barbiturates when they do not get alcohol or opiates. They are also taken to enhance or modify the effects of certain drugs of addiction. For example, emotional tension developed by cocaine or amphetamine addict may be controlled by taking barbiturate along with it. Likewise, intoxication of alcohol may be prolonged if the same is taken with barbiturates.[15]
- Therapeutically barbiturates are used as sedative hypnotic and for preoperative sedation and in controlling/ treatment of convulsions/ seizure disorders.
- Most of the deaths by barbiturates are accidental or suicidal by over dosage. Recently, death due to accidental overdosage has been drastically reduced due to reduction in its use as sedative hypnotics due to availability of safer substitutes like benzodiazepines. Barbiturate poisoning today are usually due to deliberate self-ingestion.
- Homicidal cases by barbiturates are rarely reported.
- Addiction is usually due to excessive use of barbiturates to relieve the hurrying pace and anxieties of modern life. Most remembered case of early 1960s for barbiturate overdose in the history is that of Hollywood superstar, actress *Marilyn*

Monroe, who became addicted to alcohol and barbiturate, ended her life at the peak of her career at the age of 36 years.

- The barbiturate (sodium pentothal) is often used in extracting truth from criminals and known as "*truth serum.*" However, it really does not cause people to tell the truth. Rather, it may lower a person's inhibitions and make people more talkative.

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BENZODIAZEPINES

INTRODUCTION

Death by benzodiazepines alone in the absence of other significant toxicological agents or pathology is uncommon, although benzodiazepines alone can cause death in absence of significant natural diseases or advanced age.[1] Death has been reported with flunitrazepam, diazepam, alprazolam, triazolam, temazepam, and flurazepam.

Acute overdose toxicity has been associated with *short acting* benzodiazepines such as midazolam and triazolam and *intermediate-acting* flunitrazepam than with diazepam, lorazepam and nitrazepam.[2,3] However, benzodiazepines are most commonly used drugs in clinical practice as *sedatives, hypnotics, anxiolytics, muscle relaxants and anticonvulsants*.[3,4] Table 34.16 enumerates commonly used benzodiazepines with their trade names and uses.

ACTION

It acts as CNS depressant and can bring about relief of anxiety. It also acts as muscle relaxant and used to control convulsions.

Table 34.16: Common benzodiazepines available in India with their trade names and uses

Types	*Trade names*	*Availability*	*Uses*
Alprazolam	Alpam, Alpraquil, Alprax, Alprose, Alzam, Alzolam, Alzomax, Alzopax, Anax, Anxigon, Anxit, Aprox, Anzilum, Nitril, Restyl, Zenax, Zolam, Zolax, Zoldax.	0.25 mg/0.50 mg/1.00 mg, available in India	Anxiolytic
Chloroliazepoxide	Librium	10 mg, available in India	Sedative hypnotic
*Diazepam******	Anxol, Calmpose, Calmod, Dizep, Peacin, Placidox, Valium, etc	02 mg, 05 mg, 10 mg, available in India	Anxiolytic, Anticonvulsant, and muscle relaxant
Flunitrazepam ****	—	Not available in India	—
*Flurazepam******	Nindral	15 mg, not available in India	Sedative hypnotic
Lorazepam	Ativan, Calmese, Larpose, Trapex	01 mg, 02 mg, available in India	Anxiolytic
Midazolam ******	Fulsed, Mezolam, Midosed, Shortal	Not available in India	—
Nitrazepam	Nipam, Nitravet, Nitrosun	Available in India	Sedative hypnotic
Triazolam ******	—	Not available in India	—
Oxazepam	Serepax	15 mg, 30 mg available in India	Anxiolytic
Zolpidem	Nitrest	Not available in India	—

*** Short acting benzodiazepines[2] – They are most lipophilic and have shortest duration of action in the CNS as they are rapidly and extensively redistributed[4]; ** Intermediate Acting[2]

SIGNS AND SYMPTOMS

Two types of poisoning encountered:

- *Acute poisoning* – Usually it manifests *mild* and *moderate* to *severe* form.
  - — *Mild*: Drowsiness, sedation, somnolence, diplopia, dysarthria, ataxia, amnesia and weakness.
  - — *Moderate to Severe*: Vertigo, slurred speech, nystagmus, partial ptosis, lethargy, coma.
- *Chronic poisoning* – Chronic use of benzodiazepines is associated with development of tolerance with development of mild *withdrawal reaction*. It is characterized by fits and psychosis, anxiety, insomnia, headache, spastic muscles, anorexia, vomiting, tremor, weakness, convulsions and psychiatric disturbances (rarely) such as disordered perception comprising of feelings of unreality, abnormal bodily sensation and hypersensitivity to stimuli may be seen.
- *Pregnancy/lactation*[3] – Significant depression is seen in neonates, ranging from the floppy baby syndrome characterized by hypotonia, lethargy, respiratory depression, hypothermia, and poor reflexes to simply lethargy and poor feeding.[5] Seizures, prolonged hypotonia, and prolonged respiratory depression are seen in newborns after treatment with lorazepam.[6] Dysmorphic features include craniofacial abnormalities including low nasal bridge, short palpebral fissures, epicanthic folds, a short upturned nose, slightly malformed and/or low-set ears, and hypoplastic mandible were reported in infants after maternal benzodiazepine use in early pregnancy in a controlled study.[7] However, this was not reported in further similar studies and also that confounding factors such as concomitant use of alcohol and substance abuse can not be ruled out in these studies, there by necessitating additional controlled studies.[3]

FATAL DOSE AND FATAL PERIOD

Not known. Benzodiazepines are remarkably safe. There are reports in literature favouring young adults who can ingest 30–40 times the therapeutic doses without any significant CNS depression.[4]

Toxicity rating—3.

TREATMENT

Acute poisoning

- Gastric lavage—helpful in a case brought within 6–12 hours of ingestion.
- Administer activated charcoal—can adsorb benzodiazepines
- Emesis/cathartics—may be also effective
- Establish airway—Oxygen and assisted ventilation are often useful.
- Antidotes
  - — Doxapram (100 mg IV) and physostigmine are tried
  - — Specific antidotes - *Flumazenil* is an imidazodiazepine, which can block the central effects of benzodiazepines, and effectively reverse the symptoms of benzodiazepine poisoning. It is given slowly IV in a dose of 0.1 mg/minute as infusion to a total of 1 mg. If resedation occurs in 20-120 minutes, the dose can be repeated until a cumulative dose of 3.5 mg is reached.

Chronic Poisoning

Most frequently used method is the replacement of a short half-life benzodiazepine (such as alprazolam) with a long half-life benzodiazepine (such as clonazepam), before initiating a taper and final discontinuation. Diazepam given IV 5 mg every few hours till tapered off is the line of treatment in cases withdrawal syndrome.

Postmortem Appearances

Not specific or as for symptomatology.

MEDICOLEGAL IMPORTANCE

- Often used in management of anxiety disorders, seizure disorders, insomnia, or as an adjunctive therapy in mania and movement disorders.
- Rarely death due to overdose. Suicidal attempts are relatively frequent but usually nonfatal, as these drugs are having a wide safety margin.
- Deliberate parent administration must be considered in children with benzodiazepine ingestions.[8]
- Only hazard associated with benzodiazepines is their addictive potentials.[9]
- These drugs are often used to induce deliberate *amnesia /memory loss* in certain individuals in order to accomplish iniquitous acts, such as *date rape*.[10]

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PESTICIDES (INSECTICIDE/AGROCHEMICALS)

Pesticide is an umbrella term used to describe any substance or mixture of substances intended for preventing, destroying or controlling any pest (*insects, rodents, fungi, mites, ticks, molluscs, weeds and herbs*) including vectors of human or animal diseases.[1] Pesticides are generally classified by the type of pest they control (Table 34.17).

Pesticides usually come in various preparations such as the dusting powder, emulsions, solutions, water dispensable powders, fumigants etc. The pesticides are *biocides* also capable of killing all forms of life. World Health Organisation (WHO) has classified pesticides according to the degree of hazard to mammals (Rat) as extremely hazardous, highly hazardous, moderately hazardous and slightly hazardous (Table 34.18).[2,13]

HAZARDS FROM PESTICIDES

The pesticide hazards include not only hazards to human health but also hazardous to rural environment, pest resistance and resurgence, and impact on food. Figure 34.8 highlights the *pesticide cycle* in environment (*vicious cycle*). Pesticide can result in cancer, birth defects, endocrine disruption, immunosuppression, allergies, and decreased fertility with chronic exposures. Table 34.19, highlights the hazards of pesticide on human being and environment briefly.

BIOMAGNIFICATION IN FOOD, WATER AND CROPS

India gets the credit of being the second largest producer of fruits/ vegetables in the world. Farmers being ignorant, use harmful pesticides to get improved crops. Farmers do not follow the recommended interval in spraying pesticides such as diclorvos, dimethoate, monocrotphos, endosulfan, avermectin, etc. Among the fruits, pomegranate, and grapes and among the vegetables tomato, cabbage, chillies, cauliflower, etc are sprayed with pesticides like dimethoate, quinalphose, carbendazim, and deltamethrin. Researchers found that of the pesticides applied either as sprays or directly into the soil in the granular formulations, only 15 per cent affects pests and rest is distributed in soil and air. In porous soil, pesticides can readily flow into ground water and contaminate well for long periods. It also affects the microbial flora of the soil.

MEDICOLEGAL IMPORTANCE FOR ALL INSECTICIDES[10]

- Insecticides constitute most common poisons consumed to commit suicide in India today, reason being they are easily available.
- Accidental poisoning as a occupational hazards is common among those who are engaged in spraying insecticides in agricultural farms.
- Mass disaster due to accidental food contamination is not unusual in India.
- Homicide by these poisons is not possible because of the strong kerosene-like odour.
- Organophosphate compounds used as nerve gas for warfare are particularly potent. They cause death more rapidly and

Table 34.17: Classification of pesticides depending on type of pests controlled

Types of substance	*Types of pests*	*Examples*
Insecticide	Insects	**Organochlorine Compounds** e.g. Endosulfan, Aldrin, BHC, DDT, Chlordane, etc **Organophosphorus Compounds** e.g. Malathion, Parathion, Monocrotophos, Phorate etc **Carbamate Compounds** e.g. Aldicarb, Carbaryl, Carbofuran, Methomyl, etc
Herbicide	Weeds	e.g. Paraquat, Diquat, Atrazine, chlorates, Nitrofen, 2, 4-D, Chlorophenoxyacetate (hormone) weed killers, etc.
Fungicide	Fungus	e.g. Captan, Captafol, Bavistin, Vitavax, Carbamates Thiocarbamates, Hexachlorobenzene, Sodium azide, etc.
Rodenticide	Rodents	e.g. Zinc Phosphide, Coumachlor, Warfarin, Thallium, P, As, Strychnine, etc.
Nematicide	Nematodes	e.g. Ethylene Di bromide (EDB), DBCP, etc.
Acaricide	Mites, Ticks, and Spiders	e.g. Azobenzene, Chlorobenzilate, Tedion, Kelthane, etc.

Adopted from 'Health Hazards of Pesticides and Its Management', Voluntary Health Association of India, New Delhi, 1996

Table 34.18: WHO classification of pesticides depending on LD (Rat) in mg/kg body weight

Hazard		***Oral***		***Dermal***	
Class	*Colour represented*	*Solid*	*Liquid*	*Solid*	*Liquid*
I. A. Extremely hazardous	Red	5 or less	20 or less	10 or less	40 or less
B. Highly hazardous	Yellow	5-50	20-200	10-100	40-400
II. Moderately hazardous	Blue	50-500	200-2000	100-1000	400-4000
III. Slightly hazardous	Green	Over 500	Over 2000	Over 1000	Over 4000

Source: WHO-UNFP, 1990

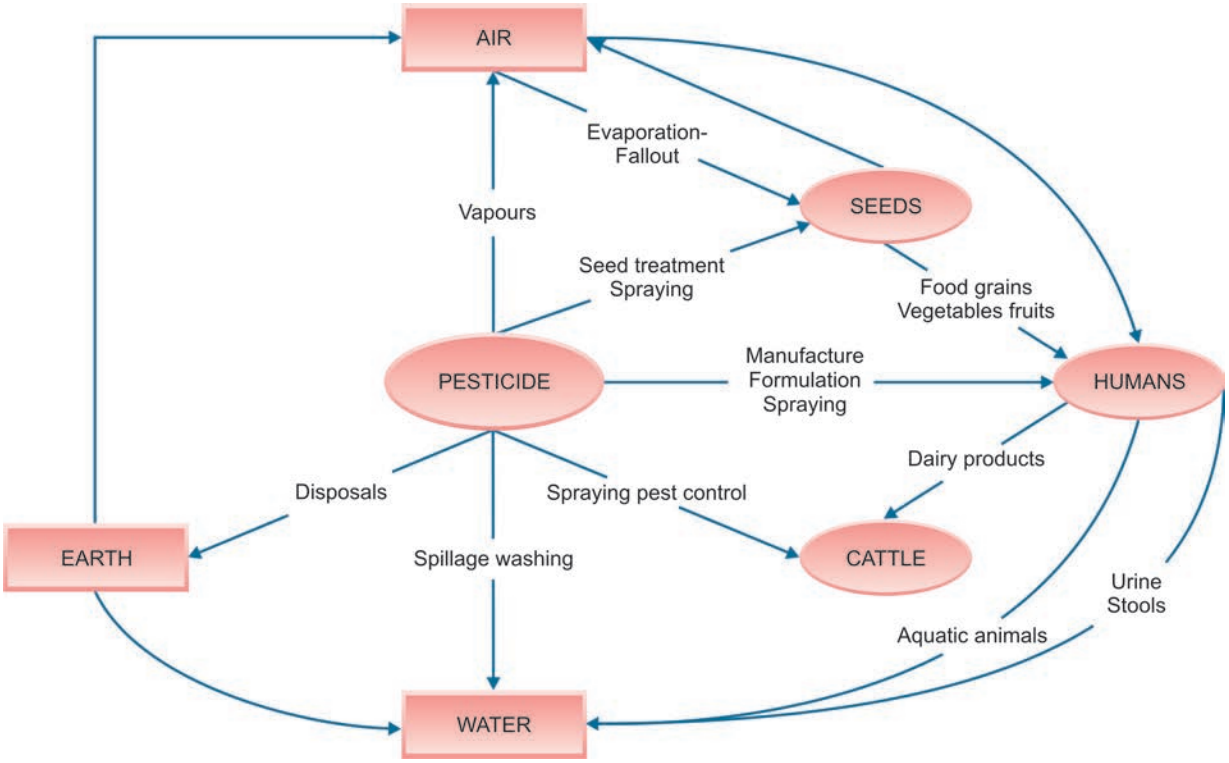

Fig. 34.8: Pesticide cycle in environment (*Source:* ITRC, Lucknow)

frequently than the organophosphate compounds used as pesticides.

Among these—organochlorines, organophosphorus insecticides, aluminium phosphide, carbamate, pyrethroids, and strychnine are discussed individually as they are considered to be very important pesticides in India.

ORGANOCHLORINES (CHLORINATED HYDROCARBONS)[5,14-18]

Classification

Organochlorines (OC) are cerebral poisons. These are also used as insecticide agents at home, garden and agricultural fields, which were popular once, now they are phasing out all over the world due to its cumulative effect and health hazards.

Some of the examples (Table 34.17) are Endrin referred often as plant penicillin), Aldrin, Chlordane, Dieldrin, *Endosulfan*, Lindane (Gammexane), Dichloro-Diphenyle-Trichloroethane (DDT), etc. DDT is rarely used now because of its carcinogenic effect also.

Properties

These are insoluble in water. DDT is a volatile solid (slow of course) having a faint odour. It is highly soluble in benzene and chloroform and moderately soluble in kerosene (11%) and slightly soluble in ethyl alcohol (2%). Other chlorinated hydrocarbons are soluble in fat solvents including kerosene. Aldrin, dieldrin and chlordane are polycyclic, polychlorinated hydrocarbons. Methoxychlor is methoxy analogue of DDT and Lindane, Gammexane and Benzene hexachlorides are isomers of hexachloro-cyclohexane. Being insoluble in water, these are used often as a dust or emulsion or mixed with solvents like kerosene.

Action

They act by interference with nerve impulse transmission. It initially stimulates the CNS and later on depresses it resulting in death. Sometimes their solvents are toxic than themselves.

Signs and Symptoms

Acute: Poisoning could occur in field workers exposed to very large amounts of organochlorine from drift, being accidentally sprayed, or from spills. Nausea, vomiting, muscular tremors, convulsions, paralysis of limb muscles, pulmonary oedema are important. Usually presents in two forms:

Mild/moderate poisoning can present with dizziness, nausea, stomach pain, vomiting, weakness, excitability, unusual amount of fearfulness and irritability.

Severe poisoning symptoms include muscle twitches, general seizures, and difficulty in breathing.

Eyes and skin: Skin shows irritation and rashes. Death may occur due to respiratory failure. Severe poisoning symptoms include muscle twitches, general seizures, and difficulty in breathing.

Victims of poisoning will have risk of *hepatitis*; GI disturbances with vomiting (often mixed with blood), melena, bile in urine and tachycardia may persist. Gammexane though cause more irritation is less toxic than DDT. Cases may persist with loss of appetite and weight loss. Aldrin, dieldrin, endrin, chlordane, toxaphane are more active convulsants. With these there is dizziness, nausea, headache and ataxia. Convulsions come suddenly. If convulsions or unconsciousness occurs without any other proceeding symptoms, then the prognosis is grave.

Chronic: Many organochlorines (OC) can cause chronic skin rashes from regular exposures. Nervous system disorder, possibly involving brain,and peripheral nerves can occur. Cases may

Table 34.19: Human health and environmental hazards due to the pesticides

Human health hazards	*Hazards to the environment*
• Nausea, vomiting and dizziness • Loss of memory, loss of appetite • Skin and allergic reactions • Delayed neurotoxicity • Behavioural changes • Lesions • Changes in CNS • Peripheral neuritis • Carcinogenic and oncogenic diseases • Effects on human reproduction leading to sterility, miscarriage, birth defects • Cataract formation • Lung and kidney damage • Effect on immune system • Enzyme imbalance–cholinesterase • Cholinesterase inhibition, etc.	**The adverse impact of pesticides on land base include** Pesticides reduces natural fertility of the soil, soil structure and its aeration, kills the useful insects in the soil, and also the predator, parasite species that feeds on these harmful insects and lead to more virulent and resistant species of insects and vectors. It produces toxic chemicals, which pollute air, land and water, poisons the food and animal feeds. **Pest resistance and resurgence** An alarming number of cases of resistance to pesticides used in farms, households and vector control, have developed in insects, pathogens, vertebrates and to some extent in weeds. Unfortunately, resistance has developed also in vectors of human diseases such as malaria, filaraia, Japanese encephalitis, Kala-azar, etc. Due to killing of non-target organism resurgence of pests occur. **Impact on Water** Pesticide residue limits have not so far been fixed for water although contamination of rivers, lakes, ground water and drinking water has officially been shown. Due to pesticide pollution in water average annual yield of "HILSA" fish has dropped considerably since 1950s. This means the pesticide residues in fish have increased and this in turn has caused threat to human beings when consumed. Pesticides in industrial effluents are found to range from 0.011 – 0.034 mg/L as against 0.001 mg/L normal value. **Impact on Food** Pesticide residues in food continue to be widespread and are a cause for concern. The picture becomes even grimmer in the light of evidence, which shows that metabolites and degradation products of pesticides like DDT, malathion, urea-herbicide become several time more toxic. Exposure of dieldrin to light for instance makes it ten times more toxic. The young children are exposed to various lethal pesticides from raw fruits and vegetables, which are above safety levels. The children's physiological vulnerability is greater leading to higher susceptibility to carcinogens due to increased cell division and longer period to accumulate and work. Cancer in children and women due to pesticides has been causing serious concern. A large number of domestic animals are poisoned every year. Significant amount of meat and milk are contaminated. Right from the day a child is born he/she is taking poison from breast milk contaminated with organochlorines. We should try to find ways and means to minimize/reduce pesticide residues in milk products. Studies have reported that milk has high levels of DDT and BHC. Infants fed with this were taking eight times more than '*acceptable daily intake*' of Arsenic, cadmium and lead, which could damage the brain and body organs. **Beverages** *The Pollution Monitoring Laboratory* (*PML*) of the Centre for Science and Environment (CSE) tested the beverages samples for 16 organochlorine pesticides, 12 organophosphorous pesticides and 4 synthetic pyrethroids. All of these are commonly used in INDIA as insecticides, in agricultural fields as well as at home. *Organochlorine pesticides* in the Indian beverages samples reported of 0.0021 mg/L of *Lindane* and 0.0015 mg/L of *DDT* and its metabolites as against the normal values of 0.0001 mg/L and 0.0001 mg/L respectively for these insecticides by European Economic Commission (EEC). Likewise *organophosphorus pesticides* in these beverages reported to be 0.0042 mg/L of *Chloropyrifos* and 0.0137 mg/L of *Malathion* as against the normal values of 0.0001 mg/L and 0.0001 mg/L respectively for these insecticides by European Economic Commission (EEC).(*Source: Pollution Monitoring Laboratory of Centre for Science and Environment*) **Factors Influencing toxicity to man** The severity of any adverse effects from exposure to a pesticide depends on the dose, the route of exposure, how easily the pesticide is absorbed, the types of effect of the pesticide and its metabolites and its accumulation and persistence in the body. The toxic effects also depend on the health status of the individual. Malnutrition and dehydration are likely to increase sensitivity to pesticide. When two or more pesticides are used simultaneously they may interact and become more toxic and hazardous to man, and there is no study done on various health hazards due to combined effect of various pesticides including their treatment and antidote. (*Source: Agriculture and people, South Solidarity, 1991*.)

present with loss of appetite and weight loss. Evidence of brain and nerve damage is more in human beings with DDT, Chlordecone and Endrin. Lindane and endrin have shown kidney and liver damage in experimental animals.

Fatal dose 15-30 gm for DDT and Lindane, 5-7 gm for Chlordane, 2.5 gm for Aldrin, Dieldrin and Endrin.

Fatal period Up to 6 hours.

Toxicity rating 4 for DDT and Lindane, 5 for others.

Treatment

- Remove the contaminated clothing and wash the contaminated skin with soap and water if poisoning is through surface contamination
- Perform gastric lavage (give activated charcoal) or induce emesis and catharsis if the poison has been ingested.
- Cholestyramine 3 to 8 gm, four times a day can enhance fecal excretion of organo-chlorines from gastrointestinal tract
- Diazepam may be given to control the convulsions
- Rest of the treatment is on symptomatic lines
- Giving atropine or oximes are not effective for poisoning by organochlorines.

Postmortem Appearances

Same as for organophosphate poisoning (*refer below*).

ORGANOPHOSPHOROUS INSECTICIDES[2-13]

Organophosphorous (OP) insecticides are most popular insecticides, often involved in serious/fatal human poisoning incidences. Victims are usually children, farmers and unskilled laborers.

History

Organophosphorus compounds were initially developed as chemical warfare agents. They were recognized after World War II. More than 50 highly effective organophosphorus compounds are in use presently. TEPP (tetraethyl pyrophosphates) were synthesized in 1800s, and are considered most dangerous orally or through the skin.

Chemistry

Chemically these are the organic derivative of phosphoric acid. They act via all routes including transdermal, transconjunctival, inhalational, across the gastrointestinal and genitourinary mucosa and through diurect injection.

Classification

There are two classifications – an *older* and a *modern* classification

Older Classification

- Alkyl compounds include—hexaethyl tetraphosphates (HETP), tetraethyl pyrophosphates (TEPP), octamethyl pyrophosphoramide (OMPA), malathion, cystox, dipterex, etc.
- Aryl compounds include—parathion, para-oxon, methyl parathion, chlorothion, diazinon, (Tick-20), etc.

Modern Classification

- Agriculture insecticides (*highly toxic*)—OMPA, TEPP-Parathion, Phosdrin, Disyston, etc.
- Animal insecticides (*moderately toxic*)—Ronnel, Coumephos, Trichlorfon, etc.
- Household insecticides (*low toxic*)—Malathion (Kill bug, Cimexol, Jyothi oil, Bugsolin-20, etc.), Diazinon (Tick-20), Vapona, etc.

Uses of Organophosphorus (OP) Compounds

In agriculture fields these are either used as aerial spray, mixed with suitable liquid or dust as their vehicle, or they are mixed with soil. When sprayed in air, absorption in plants occurs through leaves and stems. When used by mixing with soil, absorption occurs through the roots of plants. In cases of herbicidal agents, no harm is caused to the plant. When the insect sits on the plant, the poison is absorbed through their exoskeleton or when it eats the leaves of the plant, it consumes the poison along with. In human beings consuming the grain, fruits or other parts of plants causes no harm.

Absorption, Fate and Excretion

Organophosphorous compounds are well absorbed through the mucous membrane of the gastrointestinal tract, respiratory tract and intact skin. Parathion is stored in the body fat and is slowly released in the circulation, prolonging the duration of its toxic action. It is first metabolized into paraoxon, which is the active toxic agent of the preparation and then to paranitrophenol which is excreted through urine. Malathion is metabolized in the liver by the esterases. A part of metabolized product is excreted in urine as phosphate. However rate of excretion varies. Some of the preparations remain in the body longer than others. Parathion may be retained for a period of about a week and Malathion for a period of more than one week.

Pathophysiology

Acetylcholine is an important chemical transmitter helping transmission of impulses at synaptic junctions and neuromuscular junctions (NMJ). Enzyme - *acetylcholinesterase* (in nerves/ RBC) can hydrolyze and convert acetylcholine into inert products—acetic acid and choline. Organophosphates irreversibly bind to cholinesterase, causing the phosphorylation and deactivation of acetylcholinesterase. This results in accumulation of acetylcholine at the neural synapse causing an initial overstimulation, followed by eventual *exhaustion* and *disruption* of postsynaptic neural transmission in the central nervous system (CNS) and peripheral nervous systems (PNS). If the organophosphate/ cholinesterase bond is *not* broken by pharmacologic intervention *within 24 hours*, large amounts of cholinesterase are destroyed, causing long-term morbidity or death.

The *muscarinic (parasympathetic) effects* (Table 34.20) from potentiation of postganglionic parasympathetic activity of smooth muscles may cause *smooth muscle contractions in all organs (e.g. lung, Gastrointestinal tract, eye, bladder, secretory glands) and reduction of sinus node and AV conduction, causing bradyarrhythmias or resultant ventricular dysrhythmias.*

The *nicotinic (sympathomimetic) effects* (Table 34.21) from accumulation of acetylcholine at motor end plates cause persistent depolarisation of skeletal muscles, resulting in *fasciculations, muscle weakness, hypertension,* and *tachycardia*.

Table 34.20: Muscarinic effects immediate clinical effects

Anatomic site of action	*Physiologic effects*
Sweat glands	Excessive *sweating* leads to *hypothermia* and electrolyte imbalance
Pupils	*Constricted*
Lacrimal glands	*Lacrimation* (red tears) chromolachryorrhea
Salivary glands	Excessive *salivation*
Bronchial tree	*Wheezing*
Gastrointestinal	Cramps, *vomiting, diarrhea*, tenesmus
Cardiovascular	Bradycardia, fall in BP
Ciliary body	Blurred vision
Bladder	*Urinary incontinence*

Table 34.21: Immediate nicotinic clinical effects

Anatomic site of action	*Physiologic effects*
Striated muscle	Fasciculations, cramps, weakness, twitching, paralysis, respiratory distress, cyanosis/arrest
Sympathetic ganglia	Tachycardia, BP raised
CNS effects	Anxiety, restlessness, ataxia, convulsions

Table 34.22: Important facts on cholinesterase

Specimen - 5 ml of whole blood in EDTA tube.

Storage Instructions - Refrigerate entire sample at 2°C to 8°C; do not separate.

Normal level - 5300-10,000 units/L

Use - Erythrocyte cholinesterase is measured to diagnose organophosphate and carbamate toxicity and to detect atypical forms of the enzyme. Cholinesterase is irreversibly inhibited by organophosphate insecticides and reversibly inhibited by carbamate insecticides. Serum or plasma pseudocholinesterase is a better measure of acute toxicity, while erythrocyte levels are better for chronic exposure. (Serum level returns to normal prior to normalising of red cell level).

Limitations - Values decrease as erythrocytes become senescent.

Methodology - Spectrophotometery - kinetic.

Additional Information - The cholinesterase activity in human red cells is highly but not exclusively specific for acetylcholine. It is referred to as true or specific cholinesterase. Cholinesterase activity present in the serum/plasma hydrolyses both choline and aliphatic esters, has a broader range of esterolytic activity and is referred to as "*pseudo-*" or "*nonspecific*" cholinesterase. It hydrolyses acetylcholine only slowly. The systematic name for acetyl cholinesterase is acetylcholine acetylhydrolase. Systematic name for cholinesterase (serum/plasma) is acetylcholine acetylhydrolase. Both RBC acetyl cholinesterase and plasma cholinesterase are usually inhibited. However, the effect on the plasma enzyme is more marked, and serum levels are usually utilized in diagnosis and assessment of recovery.

CNS effects may cause excessive stimulation (e.g. seizure), leading to depression and coma. Actual signs and symptoms depend on the balance between muscarinic and nicotinic receptors.

Certain important facts about cholinesterases are highlighted in Table 34.22.

Signs and Symptoms

Patients will be:

- Comatose
- Pinpoint pupil
- Odour of the insecticide (kerosene like) emitted from clothing/breath
- Respiratory distress
  - — Bronchospasm, wheezing
  - — Aspiration pneumonia.

However, clinical manifestations which may be mild initially progress further to severity as time passes due to the total paralysis of cholinergic system and result in death, if not treated immediately. The immediate clinical manifestations are enumerated in Tables 34.20 and 34.21.

The mnemonic DUMBELS, which describes the symptoms of cholinergic excess,[5] is listed as follows:

- Diarrhoea
- Urination
- Miosis
- Bronchospasm
- Emesis
- Lacrimation
- Salivation.

Findings are consistent with *cholinergic excess*, but predominance of muscarinic or nicotinic effects is dependent upon the specific poison. Several other poisons can mimic some symptoms of organophosphate poisoning, including the muscarine-containing mushrooms, paraquat, and inhalational agents that cause noncardiogenic pulmonary oedema. Delayed neuropathies, as part of two recognized syndromes (i.e. intermediate syndrome, organophosphate-induced delayed neurotoxicity) should be considered in the differential with other peripheral neuropathies.

- Miosis, muscle fasciculations, and increased secretions are the most consistently reported symptoms.
- Predominate muscarinic effects include hypotension, bradycardia, and excessive pulmonary and/or GI secretions.
- Predominate nicotinic effects include hypertension, tachycardia, muscle cramps, and fasciculations.
- One study showed that 92 per cent of persons with toxic exposure had muscarinic symptoms, 44 per cent had nicotinic symptoms, 40 per cent had CNS symptoms, and 17 per cent had combined symptoms.

Diagnosis

Normal RBC (Red Blood Cell) cholinesterase levels are 5300-10,000 units/L. Markedly decreased RBC cholinesterase levels (<30% normal) are diagnostic. RBC cholinesterase levels regenerate slowly (0.5-1% per day). Depending on the RBC cholinesterase activity the levels of poisoning have been also classified into:

1. Mild poisoning—activity is 20-50 per cent of baseline activity.
2. Moderate poisoning—activity is 10-20 per cent of baseline.
3. Severe poisoning - activity is < 10 per cent of the baseline activity.

Management/Treatment[5-13]

Organophosphorous poisoning is always an emergency and is done in *two phases*: Prehospital measures and hospital care.

1. **Prehospital measures:** These include:
   - — Decontamination, which is of paramount importance with organophosphate poisoning cases. This should begin before or during the initial medical management and is done by removing all the clothing and irrigating the

exposed areas of the body. This can help in removing the residual material from the skin.

- — Supportive care, including airway control, oxygenation, ventilation, and seizure management, is a must and equally important.
- — Placement of an IV line and cardiac monitoring are indicated.
- — The presence of agricultural spray equipment, bottles of insecticides, odours (garlic/ kerosene like), and other clues to diagnosis are often unavailable to other health care personnel.

2. Hospital Care

   I. Maintain and protect airway

   II. Supplemental oxygen

   III. Gastrointestinal decontamination.

Gastric Lavage

- Consider if patient presents within 60 minutes of ingestion.
- Insert orogastric tube/gastric lavage tube.
- Attempt aspiration first, followed by 100 to 200 ml normal saline, then aspiration.
- Relatively contraindicated in hydrocarbon ingestion
- Gastric lavage with 1: 5000 $KMnO_4$ solution
- Neurologically impaired: cuffed endotracheal tube prior to lavage
- Gastric lavage not routinely used in poisonings. It is indicated only when potentially life-threatening amount of poison is ingested and the procedure can be done within 60 minutes of ingestion.

Cathartics

- Used only in combination with activated charcoal
- Sorbitol (1 to 2 ml/kg or 70% solution in adults, 1.5 to 2.5 ml/kg of 35% solution in children)
- Single dose only
- Not recommended in poisonings that produce diarrhea (organophosphates, carbamates, heavy metals in particular) or those that produce ileus (paraquat and diquat)
- Cathartics alone have no place in management of poisoned patient.[5] No definite indication for use of cathartics and its routine use with activated charcoal have been endorsed. If it is used, it should be as a single dose. Some of the contraindications include: absent bowel sounds, abdominal trauma or surgery, intestinal perforation or obstruction, volume depletion, hypotension, or ingestion of a corrosive substance.

Activated Charcoal

- Used in conjunction with a cathartic if patient presents within 60 minutes of ingestion (Some authorities would use beyond 60 minutes if serious poisoning is suspected).
- Dose: Adults and children >12 years, 25 to 100 gm in 300 to 800 ml water; children <12 years, 1 gm/kg in 300 ml water.
- Dose may be repeated in 2 to 4 hours if bowel sounds are present.
- Antiemetic suppository for nausea.
- Administered by nasogastric tube if unable to tolerate or unable to swallow.
- Protect airway if hydrocarbon-containing pesticide or unknown pesticide contents.
- Activated charcoal should not be used routinely in management of poisoned patients.[5] Charcoal appears to be most effective within 60 minutes of ingestion and may be considered for use for this time period. There is insufficient evidence to support or deny its use beyond 60 minutes after ingestion.

Syrup of Ipecac

- No longer indicated for routine use.
- Dose: adults and children >12 years, 15 to 30 ml followed by 240 ml water; children <12 years, 15 ml preceded or followed by 120 to 240 ml water; infants six to 12 months of age, 5 to 10 ml preceded or followed by 120 to 240 ml water.
- Dose may be repeated in all age groups if emesis does not occur within 20 to 30 minutes.
- Contraindicated in patients with diminished airway protective reflexes, ingestion of hydrocarbons with aspiration potential, ingestion of a corrosive substance, or ingestion of a substance for which advanced life support may be necessary within the next 60 minutes.
- Ipecac syrup should not be administered routinely in poisoned patients.[5] If it is used, it should be administered within 60 minutes of ingestion. Considered only in alert patients who have ingested a potentially serious toxin.

IV. Administration of specific antidotes:

- — Draw red cell cholinesterase and plasma pseudocholinesterase levels before therapy. Do not delay treatment while awaiting results.
- — *Atropine* administration.
- — *Pralidoxime* administration

Atropine Administration

Atropine is administered immediately to save life and continued till the achievement of the state of "*Atropinisation*", which is diagnosed by clinical manifestations (in the patients) of flushing, dry mouth, pupillary dilation, etc. Use atropine, which is preservative-free, if possible

Dose

Adults and children >12 years: 2 to 5 mg IV every 15 min, until pulmonary symptoms controlled.

Children < 12 years: 0.05 to 0.1 mg/kg every 15 min.

Doses repeated as needed for symptom control (up to 24 hours). It should be then tapered slowly after 24 hours.

Pralidoxime (2 PAM Chloride, 2-PAM, Protopam) Administration

Pralidoxime brings about anticholinergic "atropine-like" effects resulting in freeing and reactivating cholinesterase enzymes by cleaving phosphorylation—acetyl cholinesterase bond. It directly reacts and detoxifies organophosphate molecules. It can get disappear coma temporarily and also can stop fasciculation and facilitate return of strength, well being, etc.

Dose

Adults and children >12 years: 1 to 2 gm IV, repeated every 10 min.

Children <12 years: 20 to 50 mg/kg body wt over 30 min, repeated in 1 to 2 hours and at 10- to 12-hour intervals as needed for symptom control; alternatively: continuous IV infusion of 10 to 20 mg/kg/hr (up to 500 mg/hr) after initial bolus and continued for 24 hours.

V. Furosemide (Lasix): It is given in a dose of 40 to 160 mg IV to alleviate the pulmonary congestion remaining after full atropinisation

VI. Benzodiazepine (Diazepam - Valium): It is given in a dose of 5 to 10 mg slow IV push for seizures, repeated every 5 to 10 minutes to control or maximum upto 30 mg in adults; 0.2 to 0.5 mg/kg IV every 5 minutes to maximum of 10 mg in children > 5 years, 5 mg in children < 5 years; Lorazepam may also be used.

Complications

Certain ***immediate*** and ***delayed*** complications of organophosphorus (OP) poisoning are enumerated in Table 34.23.

Precautions during Treatment

- Person nursing should wear gloves, mask, etc. to prevent getting poisoned by absorption via skin or inhalation of poisonous fumes through respiratory tract
- Never give the following drugs:
  - — Morphine
  - — Aminophylline
  - — Phenothiazine
  - — Reserpin, etc.
- Perform blood cholinesterase level estima-tion to diagnose the case or assess prognosis with treatment.

Fatal Dose, Fatal Period and Toxicity Rating

Fatal dose, fatal period and toxicity rating for some of the important organophosphates are depicted in Table 34.24.

Postmortem Findings[10]

Postmortem findings/changes are (Figs 34.9A to D).

External

Face—cyanosed, blood stained fine froth at nose and mouth with kerosene-like smell.

Internal

1. Stomach
   - — Kerosene-like smell
   - — Contents would be blood stained
   - — Mucosa is congested
   - — Submucosa shows petechial haemorrhages.
2. Other findings
   - — Pulmonary oedema
   - — Capillary dilatation
   - — Hyperaemic lungs, brain and other organs.
3. Organophosphorus usually delays putrefaction.
4. Preservation of viscera for chemical examination is usually done in saturated solution of sodium chloride (organophosphorus compounds are soluble in alcohol and hence rectified spirit should not be used).

CARBAMATES[3,11,19]

Carbamate poisoning exhibits a similar clinical picture to organophosphate toxicity. However, unlike organophosphates, carbamate compounds *temporarily bind* cholinesterase for approximately 6 hours with no permanent damage. Carbamates have poor CNS penetration and cause minimal CNS symptoms.

Classification

Carbamates are *cerebral poisons*. They are also popular *insecticides* producing similar manifestations as organophosphates. They are of two types namely:

- Highly toxic—e.g. Aldicarb, Aprocarb (Baygon), Carbofuran.
- Moderately toxic—e.g. carbaryl (Sevin), primicarb, propoxur (Hit spray).

Action

By carbamylation it brings about reversible inactivation of acetylcholine.

Signs and Symptoms

Similar to organophosphates but less severe and of short duration.

Fatal dose and fatal period: Uncertain.

Toxicity rating: 4 to 5.

Treatment

Similar to organophosphate poisoning. However, oximes are contraindicated

Table 34.23: Complications of OP poisoning

Complications	*Type*
Immediate complications	• Pulmonary oedema • Aspiration pneumonia • Chemical pneumonitis • Hyper/hypoglycaemia • Transient liver function and coagulation abnormalities • Death if not treated within 24 hours of poisoning (complete recovery with proper treatment needs 10 days).
Delayed complications	• Complications related to CNS – paralysis • Peripheral neuropathy • Guillain-Barre syndrome, etc.

Table 34.24: Fatal dose, period and toxicity rating of the important organophosphates compounds

Compounds	*Fatal dose*	*Fatal period (hours)*	*Toxicity rating*
TEPP	100 mg	0-6	6
OMPA	200 mg	0-6	–
Malathion	1 gm	0-6	4
Parathion	100 mg	0-6	6
Diazinon (Tick 20)	1 gm	0-6	4

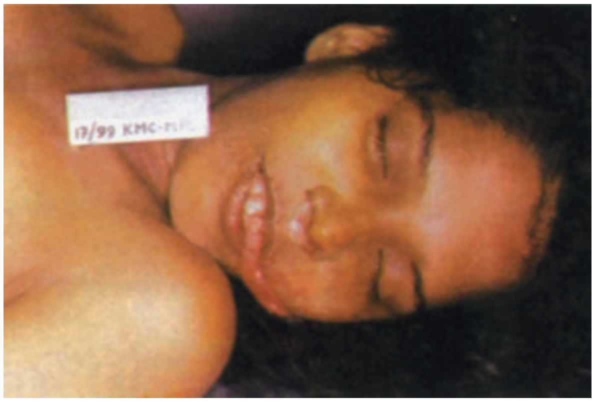

Fig. 34.9A: Autopsy findings of organophosphate poisoning case—blood stained froth around nostrils and mouth

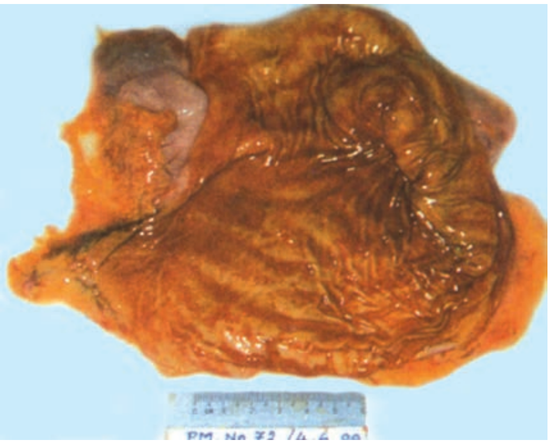

Fig. 34.9B: Autopsy findings of organophosphate poisoning case—note the changes in the stomach mucosa

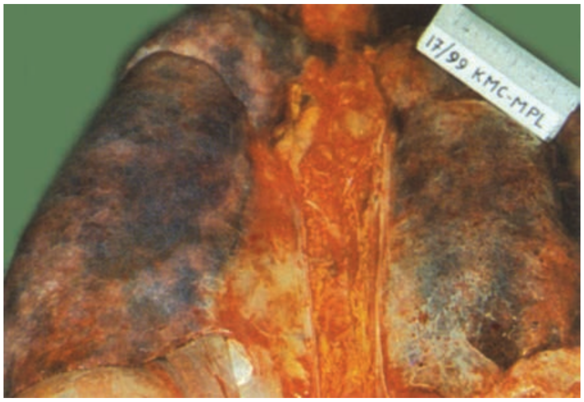

Fig. 34.9C: Autopsy findings of organophosphate poisoning case—petechial haemorrhage in the right pleura

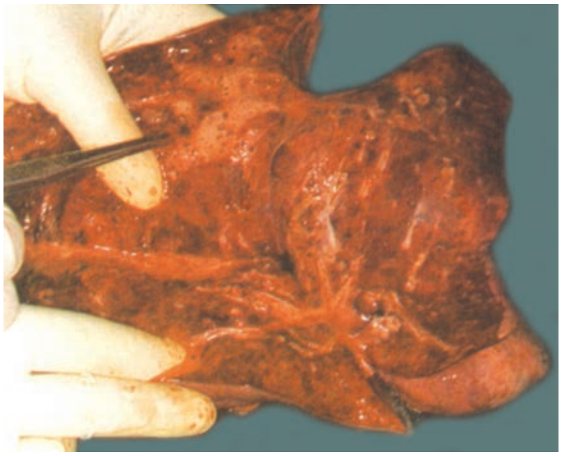

Fig. 34.9D: Autopsy findings of organophosphate poisoning case—cut section lung congested oedematous with frothy oozings

Postmortem Appearances

Same as organophosphates.

ALUMINIUM PHOSPHIDE[10,20,21]

Aluminium phosphide is a solid fumigant pesticide, widely used as a grain preservative in India. It is marketed as greyish green tablets (Trade names: Alphos, Celphos, Chemfume, Delicia, Fumigrain, Phosphume, Quickphos, Synfume, etc.).

Mode of Action

On exposure to air or moisture, it liberates phosphine and can produce multiorgan damage.

Signs and Symptoms

Metallic taste, garlicky odour, nausea, pain in gullet, stomach, abdomen, vomiting, diarrhoea, cough dyspnoea, respiratory failure, headache, anxiety, hypotension tachy/bradycardia, CCF, myocarditis, hepatosplenomegaly renal failure, coma, etc.

Fatal dose: 5 gm

Fatal period: Up to 24 hours

Treatment

- Remove clothing and wash the contaminated skin with water
- Emetics may be given
- Gastric lavage with 3 to 5 per cent $NaHCO_3$
- General supportive measure, vitamin K.

Medicolegal Points and Source

- Suicidal, homicidal, dowry death, accidental rodenticide.

NAPHTHALENE (MOTHBALLS)[20,21]

Naphthalene is an insecticide, a solid volatile substance obtained from the middle fraction of coaltar distillation and has chemical properties similar to benzene. It occurs as large, lustrous, crystalline plates with characteristic odour. Soluble in water, but dissolves freely in ether, chloroform, alcohol and oils.

Action

Naphthalene is an irritant, nephrotoxic, haemolytic and hepatotoxic.

Signs and Symptoms

Nausea, vomiting, abdominal pain, strangury, haemoglobinuria, nephritis, jaundice, haemolytic anaemia. Optic neuritis, profuse perspiration, cyanosis, convulsions, coma, etc.

Fatal dose: 2 gm

Fatal period: Uncertain.

Treatment

- Stomach wash.
- Magnesium sulfate.
- Sodium bicarbonate orally to alkalinize urine.
- Blood transfusion.
- 25 mg hydrocortisone hemisuccinate IV glucose.

Postmortem Appearance

- Skin and gastrointestinal mucosa, yellowish congested or inflamed.
- Liver and kidney may show severe damage.
- Respiratory tract show signs of irritation.
- Other organs congested.

Medicolegal Importance

- Accidental poisoning is common in children
- Suicidal cases have also been reported
- Mothballs as pesticide
- Used as deodorant in lavatories
- Manufacturing indigo and azo dyes.
- Chlorinated naphthalene is used in industry as an insulating coat on electric wires and other electric equipments.

HYDROCARBONS

Refer domestic/household poison.

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DATURA

Common name—Jimsonweed, *Thorn apple, Stinkweed, Angel's trumpet, and Jamestown weed* (as the first record of physical symptoms following ingestion occurred in *Jamestown, Virginia,* in 1676[1]).

CLASSIFICATION

Datura is a vegetable deliriant type of cerebral poison.

Distribution—Worldwide, especially in wasteland. It may also be grown in gardens for its beautiful flowers which are offered in religious prayers.

Plant [2-3]—Datura plant is about 1.2-2 meter height with widely spread branches. Fruit is about the size of a walnut, spherical and covered with sharp spiny projections (therefore called *thorn apple*). Each fruit on an average contains about 500 seeds. They are yellowish-brown in colour and resemble chilly seeds. Table 34.25 enumerates the differences between *datura* and chilly seeds. The leaves are dark green, alternate and broadly ovate in shape. Flowers are bell shaped or tubular (Fig. 34.10A to D). Although all parts of the plant are toxic, the highest concentrations of toxic principles occur in the seeds (equivalent to 0.1 mg of atropine per seed).[1]

Botanical name—*Datura stromanium.* Indian species include *Datura fastuosa*, *Datura Atroxa* and *Datura metel*. There are two varieties in India: *Datura Alba* (with white flowers) and *Datura Nigra* (with blackish or purple flowers).

Family—Solanaceae

Active principles are alkaloids such as:

- Hyoscine (Scopolamine).
- Hyoscyamine.
- Atropine.

ACTION

Poisoning occurs only if seeds are masticated and swallowed. It is bitter in taste and can initially lead to, stimulation of higher centres of brain.

Table 34.25: Differences between *datura* and chilly seeds

Characteristics	*Datura seeds*	*Chilly seeds*
Size	Bigger	Smaller
Color	Brownish	Yellowish
Shape	Kidney shaped	Rounded
Smell	Odourless	Pungent
Taste	Bitter	Pungent
Convex border	Double edged	Single edged
Embryo curvature	Inwards	Outwards

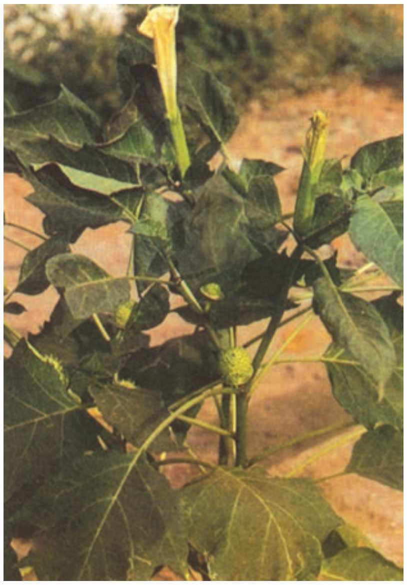

Fig. 34.10A: Datura plant with flower and fruit

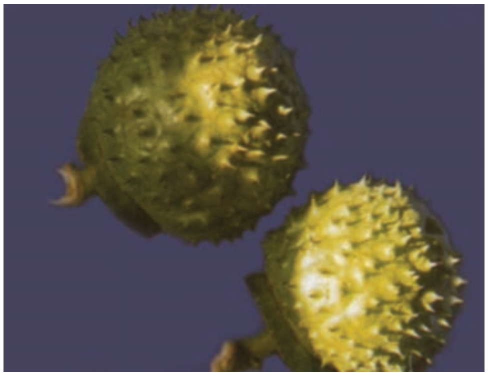

Fig. 34.10B: Thorn apple (Datura fruit)

Later the vital centres are depressed, resulting in death mainly due to respiratory paralysis.

SIGNS AND SYMPTOMS

It produces characteristic manifestations of anticholinergic poisoning[1-3] (remember as ***six D's***):

- Dryness of mouth, nausea, vomiting.
- Dysphagia
- Dysarthria
  (All the three are due to inhibition of salivation)
- Diplopia (due to dilated pupil)
- Dry, hot (due to inhibition of sweat secretion), and red (due to the dilation of cutaneous blood vessels) skin, especially in the face/ chest.

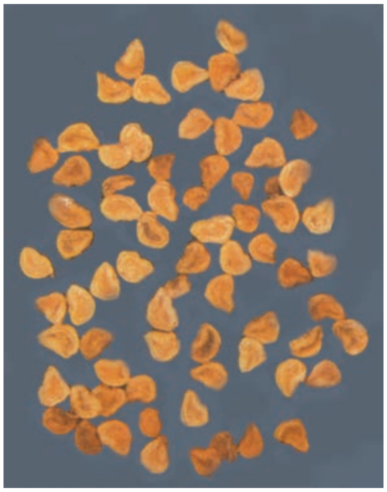

Fig. 34.10C: *Datura* seeds

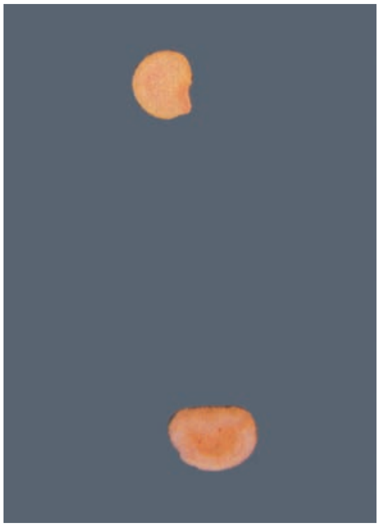

Fig. 34.10D: Capsicum (upper) seed in comparison with Datura seeds (lower)

- Drowsiness leading to coma.
- *Other finding*: The patient will be in a confused state, i.e. deliriant (muttering delirium) and hallucinating, exhibiting typical pill rolling movements, or with movements like pulling imaginary threads from fingertips. There may be urinary retention or dysuria. Death is usually due to respiratory failure or cardiac arrhythmias.[4]
- In short the clinical features of datura poisoning is described in its classic phrase as – *blind as bat, hot as hare, dry as bone, red as beet, and mad as hen*.

Fatal dose—50 to 75 seeds (0.6-1 gm)

Fatal period—3 to 4 hours to 24 hours

Toxicity rating—Seeds-3, atropine-5.

TREATMENT

- Consists of supportive care, gastrointestinal decontamination by gastric lavage with potassium permanganate ($KMnO_4$) 1:5000 solution. Due to decreased GI motility, a lavage may be useful even in late stage of poisoning. Activated charcoal in multiple doses is useful in reducing the absorption of toxins from the gut.

- Prostigmine 0.5 mg injection subcutaneously
- Pilocarpine nitrate
- Chloraldehyde or a slow-acting barbiturate (do not give morphine).
- Physostigmine should be reserved for severe hallucinations and agitation cases only. Give - 2 mg by slow IV over few minutes. Repeat the dose every ten minutes until the cessation of life-threatening condition. However do not exceed 4 mg in thirty minutes.[1,2]

POSTMORTEM FINDINGS

External

Those of asphyxia

Internal

Seeds may be detected in the stomach, small intestine.

Mydriatic Test

- It is an animal test for confirming the clinical diagnosis of datura poisoning
- A drop of urine (victim's) is instilled in the eye of a cat
- Dilatation of pupil within 30 minutes confirms datura poisoning.

Medicolegal Importance[3,4]

- *Uses* – Active principle of the plant has several medicinal uses such as – Mydriatic, spasmolytic, antidote in organophosphorous and carbamate poisoning, pre-anaesthetic medication, bronchial asthma.
- *Stupefying purpose*
  - Mixed with tobacco in cigarettes to produce state of unconsciousness or for stupefying, facilitating robbery, rape, etc.
  - Many a time robbers who disguise as saints offer the *prasadam* (i.e. the eatables of sacred value in India) mixed with datura seeds and robbed passengers when they were in a stupefied state, especially in train. This enabled datura to acquire the popular title of *railroad poison*.
- *Accidental*—Often reported cases are those of accidental poisoning:
  - Due to being mistaken for edible plant especially among the children.
  - By certain quack doctors prescribing it as quack remedies, etc.
- *Suicidal*—In villages, it is frequently used for committing suicide.
- *Homicidal*—Exceptionally rarely used for committing homicide.

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CANNABIS

CLASSIFICATION

Cannabis is classified under deliriant cerebral neurotic plant poison. It is also classified as mild hallucinogen or a sedative or a narcotic. In fact the drug is believed to produce all these effects in various individuals in a different way. However, presently it is considered as the *most abused drug* all over the world.[1,2] Slang terms for cannabis include *hash, grass, pot, ganja, spliff* and *refer*.[1]

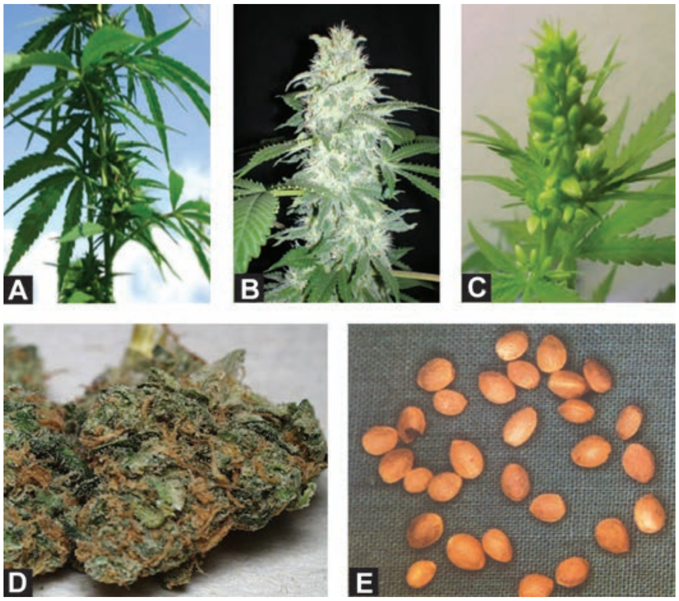
SOURCE

Cannabis is a collective term used for psychoactive compounds derived from the plant *Cannabis sativa* or *hemp plant.*[1,3] It belongs to the botanical family *Cannabinaceae* and is a tall weed growing up to 15 feet in height. Cannabis is often referred to as - *Indian hemp* (in India), *Dagga* (in South and Central Africa), *Hashish* (in Egypt), and *Marijuana* (in United States).[4] It grows all over the globe especially more in India, Africa, Egypt, and USA. Whole plant is poisonous (Fig. 34.11A to E). Active principle is usually a fat soluble oleoresin called *cannabinol (i.e., Delta*^{1} *Tetrahydrocannabinol – THC*[3,4]*)*.

CANNABIS PREPARATIONS

- Cannabis is usually dried and either smoked or eaten.[1] The cannabis preparations usually emit a peculiar odour which is described as that of *burnt rope*.[5]
- In India Cannabis is used in *three* forms and they are:

  Ganja—It is the resinous mass composed of small leaves and bracts of inflorescence (flowering tops) from female plants,[2] which contains about 25 per cent of the active principle. It is usually mixed with tobacco and smoked in a pipe (*Chilam*).[5,6] The drug is commonly consumed by



Figs 34.11A to E: *Cannabis plant:* (A) Plant; (B) Female plant with flowering buds with white hairs; (C) Male plant; (D) Dried Weed ready to smoke; (E) Seeds (*Source:* http://www.cannabis-pictures.com/female-cannabis-plant.htm)

Indian *Sadhus* and *Fakirs*. A person under the effect of *ganja* smoking, though is able to discharge ordinary duties; he feels lazy and indulges in day dreaming. It has been stated to help build nerves before performing act of *violence* or any *crime* like homicide.[5]

Charas—It is also called as *Hashish* and is the purest form of cannabis. It is the dried resinous exudate from the flower tops.[1,3] It is dark green or brown in colour, and contains 25 to 40 per cent of the active principle. This is also mixed with tobacco and used to smoke by a *pipe* or *hookah*.[2,4,5]

Bhang—Also called as *siddhi, patti* or *sabji* is the crudest form of cannabis. It is prepared as a decoction of dried mature leaves and flower stems,[3] and contains 15 per cent of active principle. It is usually consumed in the form of a *beverage*. It is a very mild form and just produces a feeling of exhilaration for about 3 to 4 hours, followed by sleep.[5] *Majun* is a sweetmeat preparation made out of *bhang,* flour, milk and butter. Datura seeds may be added to increase its effect and commonly used to stupefy persons to facilitate robbery.[5]

- Other forms of cannabis used elsewhere are:
  Marijuana (pot, grass, tea, Mary, Jane, etc.) is the most common illicit drug used in the United States.[3] It refers to any part of the cannabis plants or its extract that is used to induce psychotomimetic or therapeutic effects.[1,3] It is eaten alone or as a part of confection, or drunk in beers, or some other beverage, or smoked in pipes or after being rolled into cigarettes (*reefer cigarettes)*, people consuming these are affirmed to recall things forgotten since long.[5]

Sinsemilla is unpollinated or seedless female plant, accounts for about 85 per cent of domestic production in US especially at California, and contains about 5 per cent of active principle in it.[3]

Routes of absorption can be absorbed through both gastrointestinal and respiratory tracts.

SIGNS AND SYMPTOMS

The clinical effects experienced by a particular person depend on their mood, personality, environment and the dose taken. With smoking the onset of effects is in 10-30 minutes and on ingestion the onset is in 1-3 hours. The effects may last for about 4 – 8 hours. However crude extract of cannabis may also be injected intravenously may cause nausea and vomiting, diarrhoea, abdominal pain, fever, hypotension, pulmonary oedema, acute renal failure, disseminated intravascular coagulation and death.[1]

Cannabis toxicity usually presents in two forms:

- Acute poisoning
- Chronic poisoning.

Acute Poisoning[1]

Clinical features vary with dose consumed.

I. With low dose changes perceived in the victim comprise of:
   - Initial *euphoria* with:
     - Over talkativeness
     - Perceptual alterations.
   - This may be followed by:
     - Relaxation
     - Drowsiness
     - Hypertension
     - Tachycardia
     - Slurred speech
     - Ataxia
     - Excessive appetite
     - Eating food with great relish, etc.

II. With higher dose changes perceived in the victim comprise of:
   - Conjunctival congestion and miosis
   - Acute paranoid psychosis
   - Anxiety
   - Depersonalisation
   - Confusion
   - Hallucinations (especially of sexual character, hence cannabis is considered as an *aphrodisiac*)
   - Disorientation to time and space.
   - This may be followed by:
     - Giddiness
     - Confusion
     - Drowsiness
     - Dilated pupils
     - Tingling and numbness in the extremities
     - Generalized anesthesia (may be seen in severe cases)
     - The victim will then go into deep sleep and can be woken up soon without depression, nausea or any hangover effects
     - Rarely the victim may go into paralysis of muscles, loss of reflexes, coma and death.
     - However, a few individuals may turn *violent* and go into state called '*run amock*' (refer below*)*.

Chronic Poisoning

Chronic poisoning can present in two forms.

- Cannabis addiction
- Hashish insanity

Cannabis Addiction

- It is a chronic poisoning state; resulting from continued use of the drug in any form and is characterized by—anorexia, loss of weight, weakness, tremors, impotence and moral deterioration. The victim might become lethargic, apathetic, and disinterested to work, and suffer from poor concentration (*Amotivational syndrome*).

HASHISH INSANITY

- A cannabis addict is often found to suffer from mental disorders such as *hallucinations* and *delusions* of the persecution nature, presention with an irresistible desire to destroy life and property willfully or commit homicide out of *sexual jealousy* of which there will be no recollection afterwards. A condition called '*Run Amock' is* rarely reported with *continued use* or *sudden consumption* of cannabis, characterized by a frenzied desire to commit *murders*. After the intake of the drug, the person kills a number of individuals, the first few being those against whom he or she has some enmity (real or imaginary), followed by others who are just in the way, until the homicidal tendency lasts. The person may finally commit suicide or surrender himself or herself to the police.[5]

FATAL DOSE

- Lethal dose is: 2000 mg of *charas*
  8000 mg of *ganja*
  10,000 mg/kg body wt of *bhang*.
- For mere excitement effects just 1 to 5 gm of *Cannabis Indica* is sufficient (equivalent to 3 refer cigarettes) for non-addict.

FATAL PERIOD

Death may occur in 12 hours with lethal doses.

TREATMENT

Acute poisoning cases

- Gastric lavage with warm water/emesis
- Activated charcoal orally is effective in cases reported within an hour of ingestion.
- Give strong tea, coffee
- Provide artificial respiration, if necessary
- Saline purgatives may be helpful.
- Haloperidol is to be given to control psychotic manifestations if any
- Psychotherapy

Chronic poisoning cases

- Gradual withdrawal of the drug
- Diazepam for sedation
- Haloperidol for psychotic reaction
- Psychotherapy.

POSTMORTEM FINDINGS

- No specific findings
- There will be usual findings of asphyxia.

MEDICOLEGAL IMPORTANCE

- Multiple drug abusers with alcohol, amphetamines, benzodiazepines, cocaine, opiates, etc continue to be a problem with cannabisa users. Cocaine dependents often happen to be dependent on cannabis, fact that must be carefully elicited on admission by detailed history. Phencyclidine may be intentionally combined with cannabis/marijuana ('*super weed*') to obtain a more intense hallucinogenic experience.[6]
- Cannabis is commonly used as an *aphro-disiac agent*. It is supposed to evoke the desire for sexual enjoyment and also increase the duration of sexual act
- The drug though develops tolerance and addiction, it does not develop any physical deterioration or withdrawal symptoms.
- Cannabis has been recently appreciated for its medicinal value and is being used as an *antiemetic agent* against the nausea and vomiting produced by the *anticancer therapy*. It is also suggested in treatment of convulsions, anxiety and inflammation.[7] However currently the cannabis preparations are banned not only in India but also globally.

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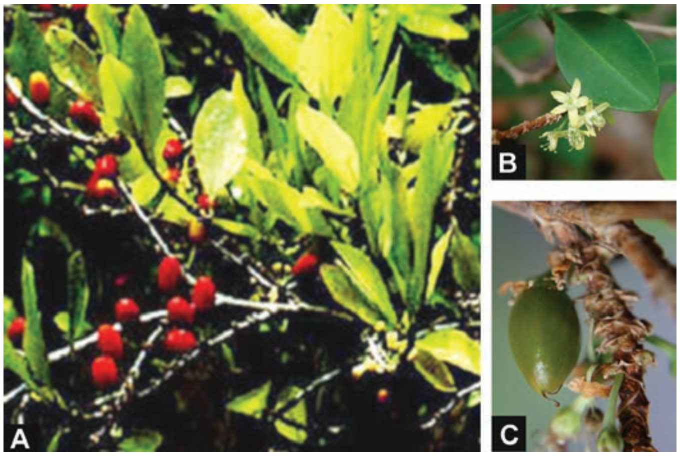
COCAINE

CLASSIFICATION

Cocaine is a *deliriant cerebral neurotic*.

SOURCE

Cocaine is an alkaloid obtained from the dried leaves of *coca plant* (*Erythroxylon coca)*.[1,2] *Coca plant* however, may not be confused for *cocoa plant* which contains *caffeine* rather than cocaine. Figure 34.12, presents Coca plant.



Figs 34.12A to C: *Coca plant (Erythroxylon Coca):* (A) Plant with leaves and fruits (ripe); (B) Flower; (C) Fruit (unripe)

COMMON NAMES

The slang names for cocaine are—*Snuff, Rock, Crack, Coke, Snow, Cadillac, White lady,* etc.[1-7]

DISTRIBUTION

Coca plant grows widely in South America, Indonesia and India.

PROPERTIES

- Cocaine hydrochloride is a white, colourless, crystalline substance, which has a bitter numbing taste. It is slightly soluble in water, but freely soluble in alcohol. Cocaine hydrochloride is soluble in water, chloroform and glycerine. It imparts feeling of numbness to tongue and mucosa of the mouth on taking it orally. It has synthetic substitutes, namely novocaine, nupercaine, etc which are used frequently as local anesthetics.
- Cocaine as such cannot be used for smoking as it gets decomposed on heating. *Crack cocaine* is a cocaine preparation which has been separated from its hydrochloride base (free base) by adding *baking soda* and water, followed by heating and then drying, which can be mixed with tobacco and smoked.[2] The name '*crack*' arises from the *noise made* when it is being prepared, as well as due to the *fissured* appearance of heated cocaine.[7] Processing of cocaine[1,4] are shown in Figure 34.13.
- Chronic consumption can develop addiction to cocaine.

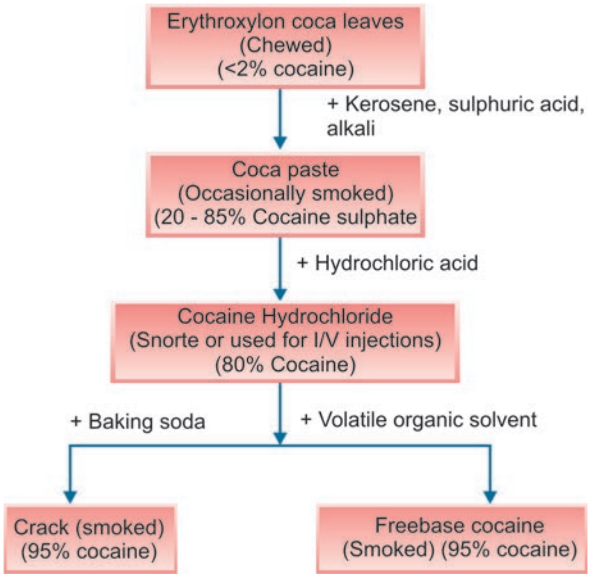


Fig. 34.13: Processing of cocaine: starts with chewable leaves and ends with nearly pure smokable cocaine[1-4]

SIGNS AND SYMPTOMS[1-6]

Depending on the route of intake, peak '*high*' effect develop and it weans off at its own time interval. Table 34.26, illustrates the fact. In most of the cases, effects of cocaine begins to resolve in about 20 minutes, except when taken intravenously. However, in fatal cases, onset and progression of symptoms is accelerated and death may occur in minutes, randomly.[2]

Cocaine poisoning usually presents in two form:

- Acute poisoning
- Chronic poisoning—called as *cocanism*.

Acute Poisoning

Usually has a stage of initial stimulation of CNS showing signs and symptoms of *mild to moderate intoxication*, followed by *stage of depression*.

- There will be a sense of well-being '*euphoria*' and increased sense of physical and mental energy, may express increase in *sexual desire*. Bruxism (teeth grinding), nausea, vomiting, pallor, dizziness, head ache, cold sweats, tremors, twitching, dryness of mouth with bitter taste are other findings in mild to moderate cases.[7]
- Excessive dose stimulation of motor cortex may result in restlessness, excitement and delirium. Brief stimulation of medullary centres can result in face flushes, and dilated pupils with blurring of vision. Victim may also present with tachycardia, hallucination (*tactile, auditory, olfactory*), ventricular ectopics, hypertension and increase in respiratory rate and body temperature (*cocaine fever*). Severe hypertension may cause haemorrhagic stroke. Coronary artery may suffer spasm resulting in *myocardial ischaemia* or *infarction* even in patients with normal coronary arteries. It may also lead to hypotension, cyanosis, cardiac arrhythmias, rhabdomyolysis, acute renal failure disseminated intravascular coagulation (DIC).[8-10] As the stimulation spreads down to lower centres, incoordination, muscular twitches and convulsions may develop.
- CNS depression when develops is characterized by loss of reflexes, muscle paralysis, pulmonary oedema, feeble respiration, circulatory and respiratory failure, resulting in death is also noticed in severe poisoning.[2]

Severe Complications of Acute Cocaine Poisoning

- Stroke, including subarachnoid and intracerebral haemorrhage and cerebral infarcts.
- Cardiovascular complications such as myocardial infarction, ventricular arrhythmias and cardiac arrest.
- Intestinal ischaemia.

Essential Investigations in Cocaine Intoxication Cases

- Early and frequent blood pressure recording and ECG monitoring.
- Detect cocaine in urine by simple drug of abuse screening tests.
- Metabolites of cocaine may be detected in the urine even after 2-3 days postexposure.
- Cocaine is an unstable in blood and hence must be collected in fluoride oxalate bulbs/ tubes.

Fatal dose

Cocaine—1 gm (however, direct application of 30 gm of cocaine to mucous membrane is also lethal as absorption through this route is rapid).

Fatal period—About 2 hours.

Treatment

- Depending upon route of administering the drug:
  - — *If applied to mucosa*—wash with warm water or normal saline
  - — *If swallowed*—perform gastric lavage with dilute solution of potassium permanganate or tannic acid or medicinal charcoal. Give activated charcoal (adult 50 gm and child 1 gm/kg) if brought within first hour of oral ingestion of any amount.
- Rest of the treatment is purely supportive care.
  - — Monitor blood pressure, heart rate, and ECG and body temperature.
  - — Give diazepam (0.1-0.2 mg/kg body weight) for controlling excitement, agitation or psychotic patient. This

Table 34.26: Route of intake and occurrence of '*peak high*' effect

Routs of intake	*Time required to attain peak 'high' effect*	*Time required to wean off the effects*
Intranasal *	20-90 minutes	20 minutes post onset
Intravenously	10 minutes	> 20 minutes post onset
Orally	45-90 minutes	20 minutes post onset
Smoking *crack*	10 minutes	20 minutes post onset
Per vaginally [7]	20-90 minutes	20 minutes post onset
Per rectally [7]	20-90 minutes	20 minutes post onset

** Chronic use through this route is known to cause ulceration of nasal septum rarely. A nasal swab is a must in suspected cases either during clinical examination or at autopsy for chemical examination at FSL*[7] also reduces central stimulation, which in turn can decrease tachycardia, hypertension and pyrexia. However, in controlling psychotic reactions better avoid *phenothiazines* and *haloperidol* as they can lower the threshold for convulsions.

— Cardiorespiratory stimulants and artificial respiration may be provided if necessary. Since cocaine induced myocardial infarction is due to spasm rather than a thrombus, it is better to avoid thrombolytics. Further administration of thrombolytics may act fatal resulting in intracranial haemorrhages. However, if the patient suffers from *cocaine induced angina*, admit the patient under coronary care unit and give intravenous or buccal nitrates.[2]

Prognosis

- If the patient survives for first *three hours* of acute poisoning, prognosis is favourable towards recovery.

Postmortem Findings

- External examination may reveal the erosions /ulcerations of the nasal septum especially among the *cocaine snorters*. Perforation of the nasal septum may also be noticed rarely (Fig. 34.14).
- Generalized congestion of the viscera and findings of asphyxia or cardiac failure are usual.
- Other findings include—cerebral and pulmonary oedema.
- Detection of poison in the viscera by chemical analysis is usually difficult as cocaine is metabolized and destroyed in the human system rapidly.

Cocainism

Synonym—Chronic cocaine poisoning, cocainophagia, cocainomania, cocaine addiction.

Causes

Chronic consumption of cocaine, for the euphoric effects of cocaine. Addicts usually take cocaine by subcutaneous injection or eat in *paan* or inhale it as snuff.

Signs and Symptoms

The victim presents with an unstable nervous system or initial psychopathic tendency, which presents with manifestation, such as:

- Anorexia, loss of weight, weakness, tremors, impotence, moral deterioration, insanity, increased erotic tension, can make a woman nymphomaniac, etc.
- Occasionally sexual pervert or homosexual individuals may present with shameless libidinous outrages
- The cocaine insanity may present with characteristic delusions of persecution and hallucination, chiefly tactile (Magnan's symptom) or of visual origin.

Magnan's Symptom (Cocaine Bugs)

Magnan's symptom is a type of tactile hallucination making the addict feel like insects (bugs) are crawling under the skin of the part of their body. He or she may even complain of presence of sand grain under the skin.

Treatment

- Stop the intake of drug
- Since withdrawal symptoms are not disgusting, treatment is usually successful with cooperation of the patient
- The teeth and tongue are black in chronic consumers of cocaine

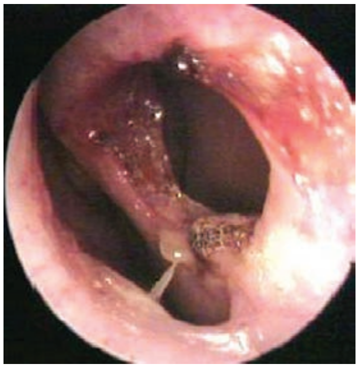


Fig. 34.14: Nasal septal perforation: Cocaine abuse—snoring

- The cocaine inhaler generally presents with perforation of nasal septum.

Medicolegal Importance

- Cocaine is used clinically for vasoconstriction and anesthesia of mucous membranes. Usually cases of acute poisoning are accidental when used for anesthetic or aphrodisiac effects.
- Urethral/vaginal injections taken for exhilarating feelings are dangerous.
- As a street drug it is sniffed into the nose (snorted) or injected intravenously.
- **Minipackers Syndrome** (*Synonyms: Bodypacker Syndrome, Bodystuffing*)[11-13]: Cocaine being a drug of abuse, it is in demand internationally. Since this is a banned drug, it is smuggled across international boundaries illegally through the body of live human beings. Those who act as *carrier* for the drug are paid highly for transporting the drug so and are often referred as *mules, bodypackers, or bodystuffers*. Cocaine filled in tiny packs such as balloons, polythene pouches or even condoms, etc. closed tightly, are made to be swallowed by these individuals, who then board a flight and reach the destination. On arrival they are taken to another hide out where made to defecate the packs with cathartic treatment. Packs passed out so are then retrieved, cleaned, and sold. Cases of stuffing packs in to the rectum, vagina, etc. are also reported. At times when the packets burst open within the gastro-intestinal tract, they release massive quantities of cocaine, resulting in acute overdose and causing the victim to succumb. Radiological examination of the suspect by X-ray, ultra sound, or CT scan methods can confirm the crime successfully (Refer to Figs 29.10A and B). Apart from cocaine, among the other banned drugs *heroin* is also reported to have been transported illegally by same method.

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STRYCHNINE

- ***Classification:*** It is a spinal poison.
- ***Distribution:*** worldwide, waste lands especially.
- ***Botanical name:*** *Strychnos nux vomica, Nux Vomica*
- ***Common name:*** Kuchila plant.

TOXIC PART

Seeds are the toxic part of the plant. These seeds are grayish brown in colour, and measure about 2.5 cm in diameter 0.65 cm in thickness and are discoidal in shape with central depression (Fig. 34.15A to C).

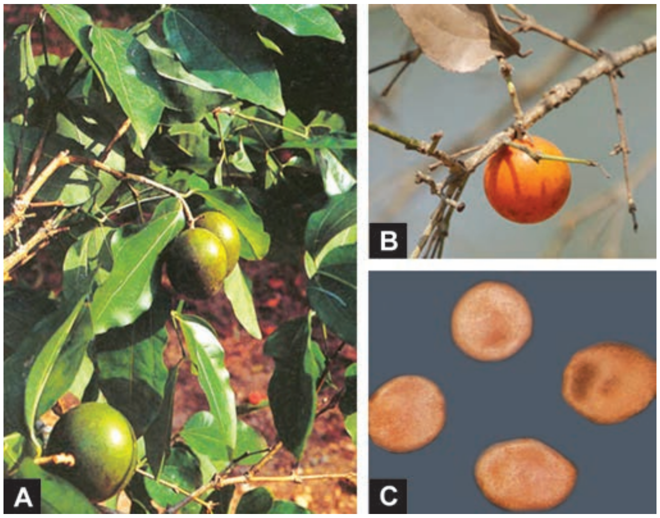

Fig. 34.15A to C: *Strychnos nux vomica (Strychnine) plant:* (A) Plant with leaves and fruits (unripe); (B) Ripe fruit; (C) Seeds inside the fruit

ACTIVE PRINCIPLES

Active principles are alkaloids such as:

- *Strychnine*—is colourless, and odourless crystalline substance with bitter taste,
- *Brucine*,
- *Loganine*.

ACTION

For the onset of action the seeds must be *masticated* and swallowed, the site of action being anterior horn cells of spinal cord. It acts by competitive antagonism of inhibitory neurotransmitter glycine at the postsynaptic spinal cord motor neurons.[1-5]

SIGNS AND SYMPTOMS

Bitter in taste. Within 15 minutes to one hour of orally taking the poison it produces—

Epigastric pain initially, followed by stiffness in the muscles with onset of the typical strychnine convulsions which are of two types:

- Clonic (intermittent) initially
- Tonic (sustained) later.
- They affect both the flexor and extensor muscles of the body simultaneously resulting in:
  - Facial muscles get fixed in a "grin" clinically called—*risus sardonicus* and prolonged spasm of the jaw muscles producing "lock-jaw" called *trismus*.
  - Other muscles of the body may contract and get fixed in one of the following postures
    - *Opisthotonus*—i.e. the body is bent backwards (hyperextension of spine) making it rest on the occiput and heels like a bow
    - *Emprosthotonus*—i.e. the body is bent forwards.
    - *Pleurothotonos*—i.e. the body is bent laterally (to left/right)
- Other findings being—cyanosis, dilated pupils, frothy salivation, respiratory distress and failure, leading to death.
- Consciousness is retained clearly till the very end, resulting in an agonising death.

COMPLICATIONS

May result in hyperthermia, rhabdomyolysis, myoglobinuria, renal failure, respiratory failure.

DIFFERENTIAL DIAGNOSIS

Strychnine poisoning case may resemble to tetanus. However, Table 34.27 depicts differentiation between the two conditions:

Table 34.27: Differences between strychnine poisoning and tetanus

Particulars	*Strychnine poisoning*	*Tetanus*
History	Of poison consumption	Of injury
Onset	Sudden	Gradual
Trismus	Late feature	Early feature
Convulsions	Generalized	Lock jaw is earliest
Relaxation between convulsions	Complete	Not complete
Chemical analysis results for strychnine	Positive	Negative

***Fatal dose*—**1-2 crushed seeds (15 to 50 mg/ 5-8 mg/kg body weight of strychnine).

***Fatal period*—**1 to 2 hours.

TREATMENT

- Treat the patient in a quiet and dimly lit room.
- Give intravenous diazepam or barbiturates for convulsions. If they are not effective, give general anesthesia or neuromuscular blockade after connecting the patient to a ventilator.
- Oxygen—artificial respiration.
- Administering succinylcholine chloride 50 mg IV may be helpful
- Gastric lavage with tannic acid after controlling the convulsions

POSTMORTEM FINDINGS

External

- Are those of asphyxia
- Rigor mortis sets early/immediately and passes off quickly
- Dead body presents with postmortem caloricity.

Internal

- Putrefaction—resisted
- Seed remains are often found in the stomach and the entire gastrointestinal tract is deeply congested.
- Preserve specially the brain and spinal cord for chemical examination in addition to routine viscera preserved.
- Frog test—inject suspected solution into the dorsal lymph sac of frog. Frog develops tetanic convulsions, if the solution has strychnine in it.

MEDICOLEGAL IMPORTANCE

- Poisoning cases when brought to clinic/OPD, require to be distinguished from tetanus as the line of treatment differs.
- Usually poisoning with strychnine is *accidental* by oral ingestion of seeds especially among the children. Among the adults it may be consumed inform of *folk medicines* resulting in accidental poisoning
- Suicide is *rare* due to agonising death
- *Homicide poisoning* is rare because of its bitter taste. *Dr William Palmer's Case* is of historical significance, wherein the doctor not only used strychnine to kill his wife for financial gains, but also managed to destroy the viscera preserved for chemical analysis for lab investigation. However a *second autopsy* of the deceased and confession of the crime with police by Dr Palmer solved the mystery.[3]

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Chapter
35
Cardiac Poisons

Cardiac poisons act mainly on the heart, either directly or through the nerves. Though there may be several cardiac poisonous plants, three are important and they are (i) oleanders (nerium and cerbera) (ii) aconite, and (iii) nicotine.

OLEANDERS

DISTRIBUTION

Oleanders are widely cultivated in the gardens in India for their ornamental flowers.

FAMILY

Apocynaceae.

PLANT VARIETIES AND DESCRIPTIONS

Oleanders are of three types

1. *Nerium odourum*: Common names—*White/Pink Oleander, Kaner*
   Nerium odourum is plant with white/pink fragrant flowers (Fig. 35.1), which are 2-5 cm in width and have five petals or in double blooms with many petals. The leaves are narrow, lanceolate, leathery, dark green on upper surface, lighter beneath, and 10-25 cm long. Fruits turn brown, dries and splits, releasing small seeds tipped with brown hairs.
2. *Cerbera thevetia*: Common names—*Yellow oleanders, Peela Kaner, Exile, Bastard Oleander*[6]
   Cerbera thevetia is a plant with yellow, bell-shaped flowers (Figs 35.2A to C), which are 5-7 cm long and 5 cm wide, the five lobes are spirally twisted and spreading. Leaves are lanceolate. The fruit is globular, light green, about 4 to 5 cm in diameter and contains a single nut, which is triangular (Fig. 35.2C) with deep groove along the edge. Each nut contains five pale yellow seeds. The seeds contain five per cent of cardiac glycoside.
3. *Cerbera odollum*: Common names—*Dabur, Dhakur, Pilikibir*
   Cerbera odollum is a small plant or a shrub (Figs 35.3A and B). Leaves are dark green, fleshy and lanceolate, 20–30 cm long and 4–6 cm broad. The flowers are white, like those of jasmines. The fruit resembles an unripe mango, is globular and dark green and has a thick fibrous mesocarp, which encloses a single seed usually. The seed is flattened and ovoid and contains two kernels (Fig. 35.3B), which are partly white, and when dry it may have bluish tinge or become gelatinous. Milky acrid juice exudes from all parts of the plant.

TOXIC PARTS

All parts of the plant are poisonous, especially fruit with *kernels* or seeds and the nectar from the flowers, which yields poisonous honey. In cerbera thevetia the milky juice exuding on cutting from any part of the plant yields the toxic principles.

TOXIC PRINCIPLES

Toxic principles are glycosides:

- *Nerium odourum*—has nerin, containing cardiac glycosides: (i) neriodorin, (ii) neriodorein, (iii) karabin. Other active principles in it are (iv) oleandrin (v) folinerin (vi) rosagenin.
- *Cerbera thevetia* has three glycosides—(i) Thevetin (one-eighth as potent as *ouabain* which is similar in action to *digitalis*; (ii) thevitoxin, is similar to but less toxic than thevetin and (iii) Nerifolin, which is more potent than thevetin. Other active principles are (iv) peruvoside (v) ruvoside, (vi) cerberin.
- *Cerbera odollum*—has only one glycoside (i) cerberin.

ACTION

Oleanders act like *Digitalis,* which can produce malignant dysrhythmias and cardiac failure, cardiac arrest and convulsions with lethal dose.[1]

ROUTES OF ABSORPTION

Oleanders are absorbed easily via skin and gastrointestinal route.

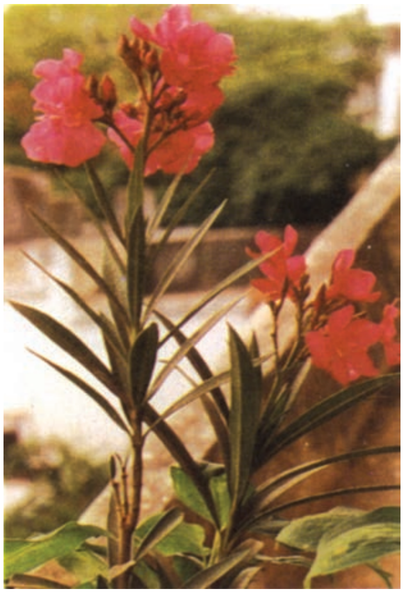

Fig. 35.1: Cardiac poisons: Pink oleander (*Nerium odourum*)

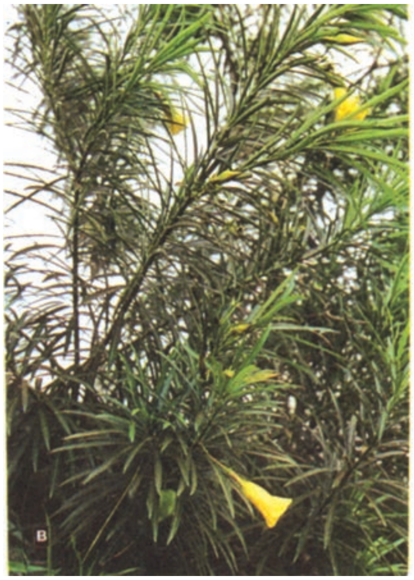

Fig. 35.2A: Cardiac poisons: Yellow oleander (*Cerbera thevetia*) whole plant

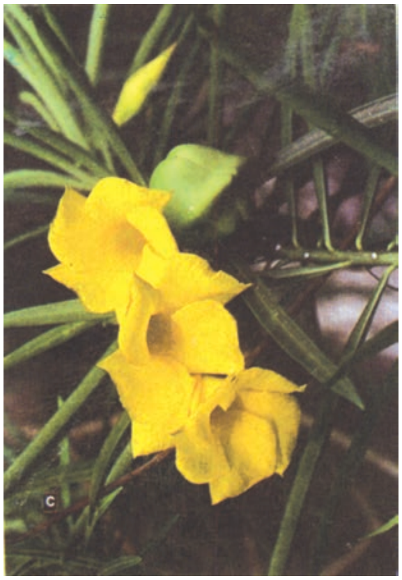

Fig. 35.2B: Cardiac poisons: Yellow oleander (*Cerbera thevetia*)—bell-shaped yellow flowers and unripe fruits

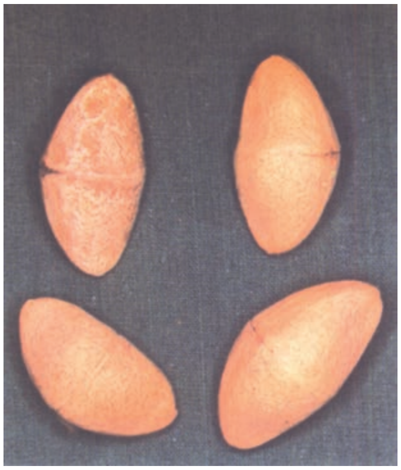

Fig. 35.2C: Cardiac poisons: Triangular nuts (kernels) from the fruit of *Cerbera thevetia* (these nuts contain pale yellow seeds inside)

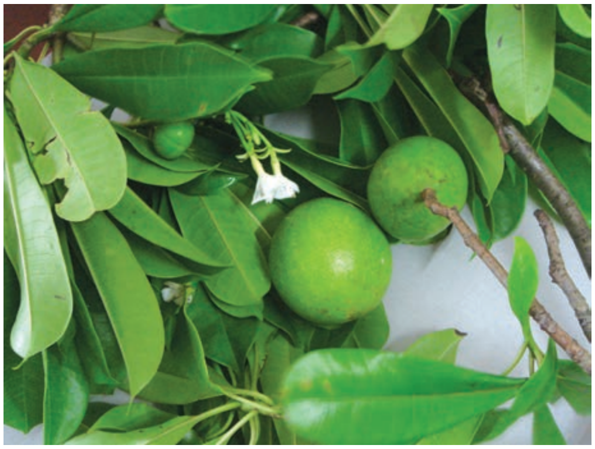

Fig. 35.3A: *Cardiac poisons: Cerbera odollum*—Plant with dark green fleshy, lanceolite large leaves and jasmine like flowers and globular-unripe fruits resembling unripe mango

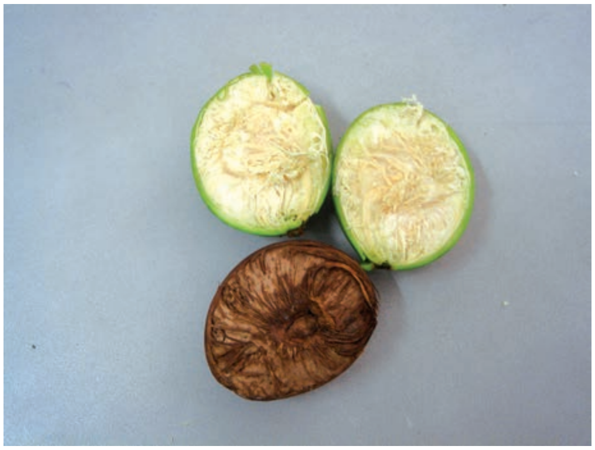

Fig. 35.3B: *Cardiac poisons: Cerbera odollum*—Unripe and dried sectioned fruits. *Note*- thick fibrous mesocarp and single seed within

SIGNS AND SYMPTOMS

General Manifestations with All Three Oleanders

Oleander poisoning in general closely resembles digitoxin poisoning with predominantly gastrointestinal and cardiac symptoms.[1] All gastrointestinal symptoms manifest within several hours. Serious toxic effects result from cardiotoxicity and specifically from ventricular ectopy and cardiovascular collapse. Conduction delay may persist for 3 to 6 days, displaying classical digitalis toxicity as characterized by increased ectopy and conduction delay (e.g. supraventricular tachycardia with atrioventricular block).[2] In a fatal suicidal ingestion, a 40 kg, 96 years old woman, developed cardiac arrest shortly after arrival in the emergency department.[3] Cardiac monitoring displayed a ventricular ectopy, including ventricular tachycardia and ventricular fibrillations, both of which were unresponsive to standard therapies. Her admission potassium level was 8.6 mEq/L. In another case a 30-year-old woman presented in cardiogenic shock with an idioveventricular rhythm 10 hours after ingestion of an oleander tea. She died after 1 hour of resuscitation with a potassium level of 6.6 mEq/L.[4]

Specific Manifestation with each Oleander

Nerium odourum—Nausea, vomiting and pain abdomen, restlessness, slow and weak pulse. Soon, respiration becomes hurried. Victim may suffer dysphagia; lock jaw, etc. followed by tetanic convulsions, leading to exhaustion, drowsiness, coma, heart failure and death.[5]

Cerbera thevetia—Burning sensation in mouth, tingling of tongue, dryness of throat, vomiting, diarrhea, headache, dilated pupils, irregular action of heart followed by drowsiness, coma, collapse and death.[5]

Cerbera odollum—Same as for cerbera thevetia (see above).

FATAL DOSE

- *Nerium odourum*—15 gm of root.
- Cerbera thevetia—10 seeds.
- *Cerbera odollum*—Kernel of one fruit.

FATAL PERIOD

- *Nerium odourum*—24 – 36 hr.
- *Cerbera thevetia*—If powdered and fed to a child mixed with milk – 3 hour.
- *Cerbera odollum*—1 to 2 days.

TREATMENT

- Treatment is mainly supportive.
- Gastric lavage, fluid administration, atropine, isoproterenol, antiarrhythmics, and early administration of activated charcoal may be beneficial.
- Haemodynamic decompensation may require temporary use of cardiac pacemaker and digoxin-specific Fab antidote fragments. Doses of 200 mg and 480 mg intravenously have improved life-threatening oleander intoxication.[6]

POSTMORTEM APPEARANCES

Nerium Odourum

- Nonspecific
- Petechial haemorrhages on the heart are important.
- *Note: Poison is heat resistant. Hence, it can be detected even in the ashes of a burnt dead body (other such poisonarsenic).*

Cerbera Thevetia

- Gastrointestinal irritation
- Congestion of viscera
- Generalized venous engorgement
- Heart—subendocardial haemorrhage
- *Note: Poison retards putrefaction and can be detected even on exhumation.*

MEDICOLEGAL IMPORTANCE

- The roots, fruits, leaves of *oleanders* are often consumed in the form of a paste, decoction, tea, etc. for committing suicide in the rural community.
- Accidental poisoning is also common when used as *an indigenous medicine* for folk remedy in villages.[1]However, *Cerbera thevetia* and *cerbera odollum* resembles unripe mangoes and are often reported to have killed many by accidental poisoning when consumed mistaken for raw mangoes. Accidental poisoning is also reported frequently whenever the plant is consumed as an aphrodisiac (*Love philter*).[5]
- The powdered kernel of *cerbera odollum* mixed with alcohol is rarely used for homicidal purposes.
- Roots or extracts of *Nerium odourum* are used locally or internally to induce abortion.
- Oleanders are often used as *cattle poison* as well in the rural India.

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ACONITE

DISTRIBUTION

Himalayan ranges. Also grown in the garden for its showy flowers.

COMMON NAME

Monkshood, blue rocket, Wolf bane, *Mithazaha/Mitha Vish* (meaning '*sweet poison*' in Hindi), etc.

BOTANICAL NAME

Aconitum plant. This has several species, common ones being:

- *Nepellus (European species),*
- *Ferox, A. chasmanthum, A. balfouri (all Indian species), etc.*

FAMILY

Ranunculecae.

PLANT DESCRIPTION

Plant is a large herb, with palmate leaves and white or blue showy flowers (Fig. 35.4A).

TOXIC PARTS

Whole plant is poisonous. However, roots are highly toxic and are used commonly. They have a conical, tapering shape with wrinkled appearance (5-10 cm long) (Fig. 35.4B) and a bitter-sweet taste. Apparently it resembles the *edible horse radish*.

TOXIC PRINCIPLE

Toxic principles are deterpene alkaloid, known as *aconitine, misaconitine,* and *hypaconitine* are. These alkaloids are sparingly soluble in water and considered as most virulent poison with sweetish taste. Other alkaloids present in small quantities in the plant are: *picraconitine*, *pseudoaconitine*, and *aconine*.

ACTION

Deterpene alkaloids are known *neurotoxins* that can cause conduction block and paralysis through their action on voltage-sensitive sodium channels in the axons.[1-3] This can result in initial

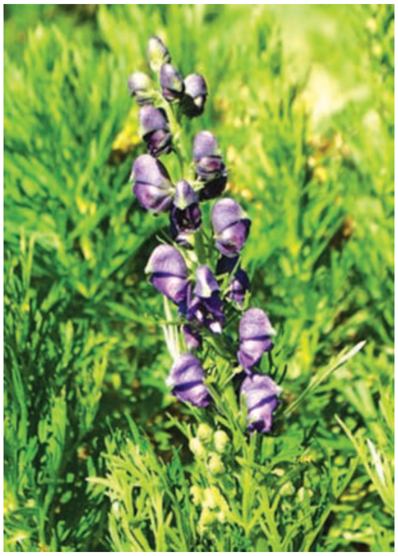

Fig. 35.4A: Aconite (Monk's hood) plant with leaves and bluish flowers (*Source:* http://www.horizonherbs.com/images/products/Aconite_monkshood_napellus.JPG)

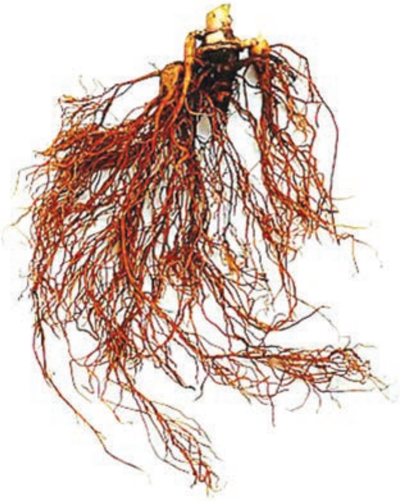

Fig. 35.4B: Aconite roots – uncut; long and tapering shapes. (*Source:* http://www.horizonherbs.com/images/products/Aconite_monkshood_napellus.JPG)

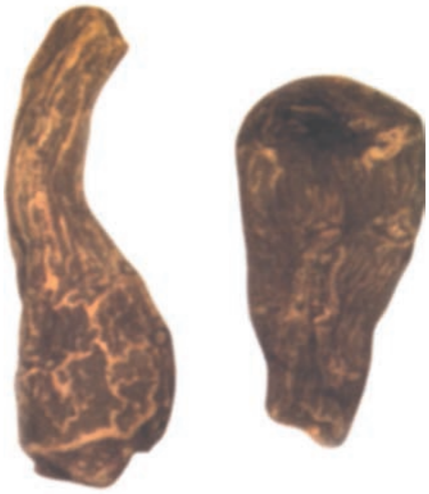

Fig. 35.4C: Cardiac poisons: Aconite roots

neurological stimulation, followed by depression of myocardium, smooth and skeletal muscles, central nervous system (CNS) and peripheral nervous system.

ROUTES OF ABSORPTION

Aconite is absorbed via skin and oral route.

SIGNS AND SYMPTOMS

Symptoms generally appear within 30 to 90 minutes after ingestion of the poison and lasts up to approximately 30 hours.

Typical Aconitine Poisoning

Some of the typical features are: [4, 5]

- Cardiovascular – Palpitation, hypertension, ventricular ectopics/ arrhythmias
- Gastrointestinal – Nausea, salivation, pain in stomach, vomiting, and diarrhoea
- Neurological – Paraesthesia, tingling and numbness in the lips, mouth, tongue, and pharynx. It may extends all over the body, followed by profuse sweating, weakness impending paralysis of the extremities and/ seizers. Deep tendon reflexes may be absent.
- Eye – There may be difficulty in vision due to *hippus,* which means initially there is alternate dilatation and constriction of pupils, followed by complete dilation.[6]
- The conscious state of the victim may also be reduced.
- Hypertensive, sustained, ventricular tachyarrhythmias are the direct cause of death. However, convulsions and respiratory paralysis may also precede death.
- Manifestations of acidosis and hypokalaemia are also often seen.

FATAL DOSE[6]

- 1 gm of root (Indian)
- 250 mg of root extract
- 20 drops of tincture extract
- 3 to 5 mg (average 4 mg) of alkaloid – Aconitine.

Fatal period 3 to 24 hours maximum (average 6 hours).

TREATMENT

- Gastric lavage with warm water, and weak solution of iodine in potassium iodide or tannic acid to precipitate the alkaloid, or animal charcoal.
- Artificial respiration (oxygen)
- Atropine – 1 mg, given to prevent vagal inhibition of heart.
- 0.1 per cent novocaine – 50 ml controlling the cardiac arrhythmias.

POSTMORTEM APPEARANCES

Findings are characteristic:

- Root pieces may be detected in stomach contents
- Mainly asphyxial features are seen.
- Aconite is highly unstable and excreted in urine, body fluids. It gets destroyed early and hence may not be detected on chemical analysis. However to prevent this, acetic acid is added to the rectified spirit (1:2 conc.) used to preserve the vomitus to be sent for chemical examination.

MEDICOLEGAL IMPORTANCE

- Most virulent poison ever known
- Accidental poisoning is more common due to consumption of raw roots, mistaking them for horseradish, which is edible. So also when consumed as a medicine (as quack remedy for common cold, criminal abortion, etc. in villages—due to over dosage).

- Homicidal/suicidal incidences though not common are also reported by the ingestion of *paan* (betel leaf) mixed with the poisonous root/tincture. Aconite, however, is considered as an ideal homicidal poison. It is not an ideal suicidal poison as it produces painful death.

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NICOTINE (TOBACCO)

DISTRIBUTION

Nicotine grows in all tropical regions of the world.

- **Common name** – Tobacco plant.
- **Botanical name** – *Nicotiana tobacum*.

TOXIC PARTS

Dried leaves (Fig. 35.5) and stems of *Nictiana* species. These include *N. tobacum* (cultivated tobacco), *N. attenuate* (wild tobacco), *N. glauca* (tree tobacco), and N. trigonophylla (dessert tobacco). *Lobeline* is the chief constituent of Indian tobacco, obtained from the leaves and tops of *Lobelia inflatea*, is an alkaloid similar to nicotine, but less potent than nicotine, and is used in antismoking tablets and lozenge.[1,2]

TOXIC PRINCIPLES

Nicotine contains an alkaloid known as *nicotine*, which is colourless liquid which turns amber on exposure to light.

ACTION

Stimulation and depression followed by paralysis of cells of peripheral autonomic ganglia, midbrain, spinal cord, muscles, etc.

ABSORPTION AND METABOLISM

Nicotine is absorbed via intact skin and oral mucosa, GI tract and respiratory system. It is detoxified in the body, mainly in the liver, but also in the kidneys and lungs. The major metabolic product of nicotine is *cotinine,* which with non-metabolized form of nicotine is excreted in urine.[1] Elimination is complete in 16 hours. An acidic urine increase urinary excretion. Nicotine is also excreted in the milk of lactating woman.[3]

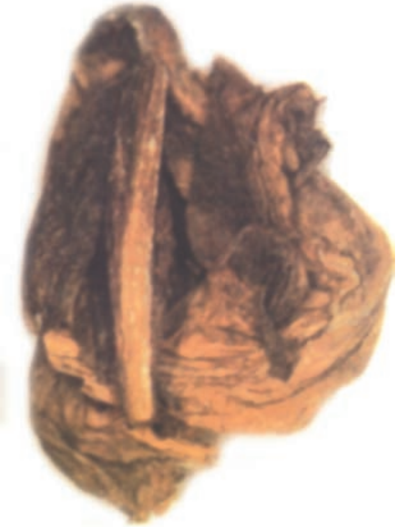

Fig. 35.5: *Cardiac poisons:* Nicotine (tobacco) dried up leaves

SIGNS AND SYMPTOMS

More than 95 per cent of reported cases are either asymptomatic (70%) or mild (25%). Most of the recently reported serious toxic states from nicotine have been from accidental exposure to animal control agents by their handlers.[4] It is evident from the literature that no nicotine related deaths have been reported in recent years.[1]

Mild Poisoning

Frequently occurs by chewing the dried leaves producing dizziness, nausea, vomiting, headache, perspiration, weakness, cardiac irregularities, etc. Victim will turn normal in a few hours.

Acute Poisoning

Acute poisoning occur schiefly due to the central/ peripheral stimulations. Manifestations include all mild poisoning features mentioned above and other findings such as burning in mouth, throat, stomach, prostration, etc. followed by convulsions and cardiac irregularities, sometimes even cardiac arrest and death. Virtually all toxicity from nicotine is reported from cigarettes. More than 90 per cent of toxic exposure from cigarettes in USA is reported in children less than 5 years of age. Germany reports of such incidence among infant aged 7 months.[1]

Nicotine Withdrawal Symptoms

Considerable evidence that nicotine is a highly addictive substance exists. Nicotine withdrawal symptoms (Table 35.1) can be prolonged and debilitating.[5]

Chronic Poisoning

Chronic poisoning is known as nicotine addiction and is common among the nicotine insecticide sprayers, tobacco chewers, cigarette smokers, etc. Victims may manifest with chronic cough, bronchitis, laryngitis, pharyngitis, dermatitis, angiospasms, tremors, amblyopia and narrowing of field of vision, blurring, etc. The effect of nicotine on the heart is called "*tobacco heart*", characterized by extra-systole, angina, etc. Chronic cases may also frequently manifest with *thromboangiitis obliterans (TAO)* of extremities.

FATAL DOSE

2 gm of tobacco or 60 mg of nicotine. One cigarette usually contains 1 gm of tobacco and 15-25 mg of nicotine. However,

Table 35.1: Symptoms during nicotine withdrawal[5]

- Nervousness
- Drowsiness
- Anxiety
- Lightheadedness
- Headaches
- Energy loss
- Fatigue
- Constipation or diarrhoea
- Insomnia
- Dizziness
- Sweating
- Cramps
- Tremors
- Palpitations

the smoke contains less than 3 mg of nicotine per cigarette. Usually a non filtered brand cigarette smoke contains 1.2 to 2.4 mg and filtered brands between 0.2 to 1.0 mg of nicotine per cigarette and 5 ml of any of the nicotine insecticides.

FATAL PERIOD

A few minutes to a few hours.

TREATMENTS

- Gastric lavage with activated animal charcoal
- Purgative
- Atropine 1.5 mg (if there is salivation)
- Cardio respiratory stimulants
- Symptomatic treatment
- Stop smoking permanently in TAO cases.

POSTMORTEM FINDINGS[6-9]

- Asphyxia alone or *comatoasphyxial* findings
- Stomach emits typical tobacco smell with brownish discolouration of the stomach wall
- Brownish stain may be seen on skin also
- Mucosa may show signs of irritation
- Lung—shows pulmonary edema
- Nicotine resists putrefaction.

MEDICOLEGAL IMPORTANCE

- Nicotine is most commonly used for smoking in the form of tobacco as cigarettes all over the world. Common blends of tobacco vary in their nicotine content. The average cigarette contains 15-25 mg of nicotine with the smoke containing less than 3 mg per cigarette. However, a non filtered brand smoke contains 1.2 to 2.4 mg and filtered brands between 0.2 to 1.0 mg. In India, it is also consumed by chewing (with betel leaf) or by inhalation as snuff.[1]
- More recently, nicotine sulphate has been used as a *dog control agent*, with approximately 285 mg of nicotine/ml. It is also used as animal tranquilizer darts; with strength of 240 mg of nicotine/ ml. However, its use as an *insecticide* in 1920s and 1930s has been greatly diminished at present.
- Most of the criminal use of nicotine in India is – to induce *Infanticide,* either by applying the nicotine extract over the nipples of the nursing mother or feeding the same with milk. Keeping the tobacco leaves in the arm pits for a few minutes can bring about local rise of temperature rendering it to *malinger fever* and thus sickness. This is the other form of criminal use.[6-9]
- Chronicity can lead to—tobacco heart (see above), lung cancer, and abortion.[10]

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Chapter 36

Asphyxiants

CLASSIFICATION

Asphyxiants are gaseous poisons, which produce respiratory embarrassment, leading to asphyxia. These gaseous poisons are basically four types:[1]

- Chemical asphyxiants
- Simple asphyxiants
- Respiratory Irritant asphyxiants
- Systemic asphyxiants

Chemical Asphyxiants

Chemical asphyxiants reduce the body's ability to absorb, transport, or utilize inhaled oxygen. In other words by their specific toxic action they render the body incapable of *utilising* an adequate oxygen supply. They are often active at very low concentrations (a few ppm in air). Examples include: *carbon monoxide, cyanides* and *hydrogen sulphide*.

Simple Asphyxiants

Simple asphyxiants are inert gases and they *deprive* tissue of oxygen by their ability to displace oxygen. Thus these asphyxiants displace the oxygen from the inspired gas mixture creating diminished uptake. Examples include: *carbon dioxide, helium, nitrogen, nitrous oxide, aliphatic hydrocarbon gases such as butane, ethane, methane and propane,* and *noble gases namely argon, helium, neon, radon and xenon.*

Respiratory/Pulmonary Irritant Asphyxiants

Respiratory or pulmonary asphyxiants are gases, which can damage the respiratory tract by destroying the integrity of mucosal barrier and produce non-cardiogenic pulmonary oedema, which impairs the oxygen diffusion across the alveolar membrane. Examples include: *ammonia, chlorine, formaldehyde, hydrogen sulphide, methyl isocyanate, and oxides of nitrogen, ozone, phosgene,* and *sulphur dioxide.*

Systemic Asphyxiants

These are gases, which produce significant systemic toxicity by specialized mechanisms. Examples include: *Carbon monoxide, cyanide,* and *smoke*.

However, only carbon monoxide, carbon dioxide, war gases, hydrogen sulphide, cyanides and ammonia would be discussed.

CARBON MONOXIDE (CO)

Carbon monoxide accounts for many deaths in developed countries such as United States, Europe, etc. with suicide attempts and through accidental poisoning.[2]

Properties

- Carbon monoxide is a colourless, tasteless, odourless and nonirritant gas, produced whenever there is incomplete combustion of carbon.
- It is soluble in water.
- It burns with a blue flame.

Sources

Most important source is due to the incomplete combustion of almost all forms of fuel such as wood, charcoal, gas, kerosene, etc. Other sources include automobile exhaust, fires and tobacco smoke. Endogenous CO resulting from haem degradation constitutes another source of CO. Thus CO is an end product of normal metabolism and is formed during the conversion of haeme into biliverdin.[3] However, this can never reach toxic levels. Normal CO level in plasma is 1 to 5 per cent and this may rise up to 7 to 8 per cent in chronic smokers. City dwellers and smokers may show Carboxy Hb (COHb) level of 3.5 per cent in their blood.[4-6] Levels vary with the CO contamination of the atmosphere.[2]

Mechanism of Action

Carbon monoxide has got 200 to 300 times more affinity for haemoglobin (Hb) than oxygen (O_2) forming carboxyhaemoglobin, which are quite stable.[2] This renders haemoglobin incapable of carrying oxygen resulting in *tissue anoxia*. As long as CO is in the atmosphere, it goes on accumulating and gets fixed in blood, leading to acute chemical asphyxia. The CO absorbed by the lung avidly combines with haemoglobin (85%). Elimination occurs exclusively through lungs.

Signs and Symptoms

The clinical manifestation poisoning presents in two forms: *acute* and *chronic*:[4-7]

Acute Poisoning

The severity of poisoning usually depends on percentage of COHb. Thus the signs and severity depends on degree of saturation of CO in blood. Table 36.1 highlights the signs and symptoms of acute poisoning depending on gradient of increasing levels of carboxy Hb saturation in the blood along with the severity of poisoning. Formation of carboxy Hb can lead to anoxaemia resulting in weakening of the vascular walls; anoxia leading to the degeneration of nerve elements; weakening of the heart due to lack of nutrition and ultimately death due to deprivation of O_2.

Table 36.1: Sequence of symptoms related with gradual increase of COHb

Saturation of COHb (%)	*Symptoms*	*Severity of poisoning*
0 - 10	No appreciable symptoms	Mild (10-30%)
10 - 20	Breathlessness, slight headache, lassitude, skin flushes	
20 - 30	Throbbing headache, irritability, emotional instability, disturbed judgement, defective memory, buzsing in ears, breathlessness.	
30 - 40	Severe headache, nausea, vomiting, dizziness, dimness of vision, breathlessness, mental confusion, muscular weakness and incoordination,	Moderate (30-40%)
40 - 50	All symptoms are intensified (may be mistaken for drunkenness), increasing confusion, sometimes hallucinations, severe ataxia, rapid respiration and collapse with attempts exertion.	Sever(>40%)
50 - 60	Syncope or coma with intermittent convulsion, tachycardia with weak pulse, rapid respiration, Cheyne-Stoke response, respiratory paralysis resulting in coma. Skin may shoe pink or red discolouration.	
60 - 70	Increasing depth of coma with incontinence of urine and feces.	
70 - 80	Profound coma with depressed or absent reflexes, weak thready pulse, shallow irregular respiration and death.	
> 80	Rapidly fatal due to respiratory arrest.	

Table 36.2: ***Fatal dose and fatal period (for CO)***

Conc. of gas (percentage)	*Fatality (in hours)*
0.02	Death in 4
0.04	Death in 1

Fatal Dose and Fatal Period

Table 36.2 enumerates the dose and fatality observed in a healthy adult. However, fatality with 0.04 per cent is rapid, if the victim is asleep in an ill-ventilated room. Low burning of kerosene or other hydrocarbons are often the culprits.

Chronic Poisoning

This is due to frequent exposure to CO leading to tissue injury seen in person working in gas houses, automobile workers, traffic police, houses and shops near heavy traffic roads.

Signs and symptoms include nausea, digestive upset, dull frontal headache, palpitation, and aggravation of angina, anemia, visual disturbance, loss of sensation in fingers, etc. There may be symptoms of atrial fibrillations, bundle branch blocks, AV block, abnormal left ventricular function, decreased cognitive ability, loss of memory, mental retardation, psychosis, parkinsonism and incontinence.

Delayed Deaths

Coma is accompanied by degenerative changes in brain and capillaries. Neurological symptoms such as blindness, decerebrate rigidity, etc may also be developed.

Diagnosis

Carboxyhaemoglobin (COHb) level: 0 to 5 per cent normally, but may be as high as 10 per cent in heavy chain smokers.

Pulse oxymetry: This is a simple non-invasive technique of detecting oxygen saturation measured by discrete wavelengths of light corresponding to saturated and unsaturated haemoglobin (660 and 940 nm) by a photo detector.

ECG: ST depression or elevation, T-wave flattening or inversion, dysrhythmias, etc are diagnostic.

Chest radiograph: This may reveal ground glass appearance, perihilar haze, peribronchial cuffing, and intra-alveolar oedema.

Bedside tests:

1. Add a drop of blood suspected to contain CO to 10 to 15 ml of water and shake well. A pink colouration of water detects presence of CO. With normal blood, colour will not be pink.
2. Take 1 ml of blood from suspected CO poisoning victim. Add to it 10 ml of water and 1 ml of 5 per cent solution of sodium hydroxide. Following colouration suggests presence of various concentrations of carboxy haemoglobin – (COHb).
   - Brownish colouration – Normal blood (due to the oxyhaemoglobin – HbO_2)
   - Straw yellow colouration – < 20 per cent COHb
   - Pink colouration – > 20 per cent COHb.

Treatment

- Shift the patient to fresh air immediately.
- Provide artificial respiration and provide 100 per cent oxygen using a tight fitting mask or endotracheal tube, until COHb falls to 15 to 20 per cent.
- Gastric lavage in early stage helps in preventing aspiration pneumonia.
- Monitoring the cardiac and respiratory status. Keep the patient at complete rest for minimum 48 hours.
- Prevent cerebral oedema by hyperventilation (PCO 25 to 30 mm Hg), head elevation, infusion of mannitol (0.25 to 1 gm/kg of 20 per cent solution for 30 minutes) and avoid administering steroids.
- Diazepam or phenytoin to control convulsions.
- In order to prevent lung infection antibiotics are given prophylactically.
- A whole blood transfusion is useful.
- ***Antidote***: Administration of *hyperbaric oxygen* (HBO) is considered, as specific antidote by few authorities, may be helpful in reducing neuropsychiatric symptoms. However giving HBO has its own risks such as cerebral gas embolism, rupture of tympanic membrane, visual deficits and oxygen toxicity such as convulsions and pulmonary oedema. Any patient with COHb level > 25 per cent should be given HBO with CO_2.

Postmortem Findings

- Cherry red discolouration of the skin, mucosa, postmortem lividity (hypostasis), blood, and viscera, etc. If the blood is taken in a test tube and diluted with water and held against light or a white back ground, the pink colour will be better appreciated.
- Fine froth at nostrils/mouth.
- Pulmonary oedema.
- Anoxic necrosis of muscles
- Necrosis of heart muscles.
- Necrosis and cavitations of basal ganglia, especially globus pallidum and putamen is usually seen in delayed deaths cases.

Collection of Blood Sample for Chemical Analysis

Collect the blood from peripheral veins. Add sodium fluoride as a preservative and preferably seal the bottle with a layer of liquid paraffin on the top.

Medicolegal Importance

- It is a common mode of committing "*suicide*" in the West (US) by inhaling motor vehicle exhaust by sitting in a closed garage with a window of the car open while the fumes build up in the garage or a hose is connected at its one end to exhaust pipe of the vehicle and other end kept within the vehicle with window panels rolled up; or by putting head in gas oven, etc, which has CO (in UK).
- Accidental — cooking gas leakage in kitchen at home. At times mass deaths in conflagration wherein a large building catches fire; cause of death is by inhalation of smoke containing CO.
- Masochistic sexual asphyxia
- Homicide by CO poisoning is though rare is reported time to time. Victims are usually children. Victim will be incapacitated by drink, drug or disease or infirmity if an adult.

CARBON DIOXIDE (CO_2)[2,5,8]

Properties

Carbon dioxide is a heavy, odourless, poisonous gas, produced by complete combustion of carbon containing compounds. It is also formed during respiration, combustion, fermentation, and decomposition of organic matter, mine explosion (Choke damp – CO_2 + CO) and in refrigerating plants. In solid form it is called as dry ice. Atmospheric air usually contains 0.4 per cent CO_2. Occasionally it is detected in certain unused wells or cellars or deep leach pits kept closed for long time. People die accidentally when they enter into such wells/cellars/leach pits for cleaning purposes. These wells are popularly known as *killer wells*/*ghost wells*.

Signs and Symptoms

- Vary with the concentration of gas.
- Pure CO_2 Vagal inhibition + Glottis spasm

  ↓

  Instant death

All symptoms are chiefly due to lack of oxygen. Table 36.3 summarises sequence of symptoms with increase in blood saturation of CO_2.

Fatal Dose and Period

Maximum concentration of CO_2 in atmosphere is 5000 ppm.

Fatal Concentration

Minimum — 25 to 30 per cent
Maximum—60 to 80 per cent.

Fatal Period

Instant collapse and death.

Treatment

- Shift to fresh atmosphere
- Maintain body warmth
- Artificial respiration + O_2 therapy
- Cardiac stimulants—amphetamine sulphate (exposure to long period can lead to irreversible brain changes).

Postmortem Findings

- Lack of oxygen such as:
  - Cyanosis
  - Marked capillary and venous congestion
  - Petechial haemorrhages
  - Froth at nostrils and mouth
  - Blood is dark and fluid
  - Deep congestion of viscera
- Preserve blood for chemical examination.

Medicolegal Importance

Usually accidental, among workers involved with deep well cleaners, unloading of wet grains from cargo ships (ferments CO_2). Any ill-ventilated room containing a crowd of people.

WAR GASES

War gases are chemical that are used only in warfare.[9-11] The term designates its applicability as an agent befitted to carry destruction or damage mostly in times of war or in dispersing unruly mobs.

Qualities of Ideal War Gas

- Should be capable of being manufactured cheaply in enormous quantities as any industrial byproduct
- Must be highly toxic in low concentration

Table 36.3: Sequence of symptoms related with gradual increase of Hb: CO_2

Blood saturation of CO_2 (%)	*Symptoms*
0-2	No symptoms
4-5	Breathing —laboured
5-10	Ataxia, unconsciousness
10-20	Slow respiration, fall of BP, coma, loss of reflexes, anaesthesia-death due to pulmonary oedema and haemorrhage
20-40	Interferes with total O_2 supply— dyspnoea
40-80	Immediate unconsciousness, convulsions, death due to asphyxia from deficiency of O_2 supply to brain and tissues (cerebral hypoxia)

Table 36.4: Types of war gases along with examples

Types	*Examples*
Lachrymators (Tear gases)	Chloracetophenon (CAP), Bromobenzyl cyanide (BBC), Ethyl iodoacetate (KSK)
Lung irritants	Chlorine (Cl_2), Phosgene (CO Cl_2), Diphosgene
Nasal irritants (Sternutators)	Diphenyl chlorasine (DA), Diphenylamine chlorasine (DM), Diphenyl cyanarsine (DC)
Nerve gases	Chemical with acetylcholine like action
Paralysants	Carbon monoxide (CO), Hydrocyanic acid (HCN), Hydrogen sulphide (H_2S)
Vesicants	Mustard gas, Lewisite
Miscellaneous	Yellow/red rain, Methyl isocyanide

- Must be heavier than air
- It must be capable of enough volatilisation pervading assailed area
- Gas should not corrode the containers used for storages.

Classification

A list of various war gases commonly in use globally are presented in Table 36.4.

Lachrymators (Tear Gases)

Lachrymators (see Table 36.4) need to be fired during an attack in the artillery shells or pen guns, so as to saturate the area of attack. *Chloroacetophenone (CAP)* is a solid, in a finely divided *powder* form with *locust flower* odour. It disintegrates rapidly. *Bromobenzyl cyanide (BBC)* is *heavy oily* dark brown liquid with *fruity* odour. Its effect may persist for a maximum of 3 days. *Ethyliodoacetate (KSK)* is also heavy oily liquid with *teardrops* odour, with its effect persisting for 10 days.

Action

- These war gases are harmless to life.
- They produce in their victims:
  - — Severe lacrimation due to intense irritation of the eyes with a copious flow of tears, spasm of the eyelids and temporary blindness.
  - — Irritation to respiratory passages
  - — Long continued exposure can result in nausea, vomiting, blistering and ulceration of the skin.

Treatment

- Shift the patient to fresh air.
- Eye care—irrigate/ wash the eyes with normal saline or boric acid solution.
- Skin, nose and other site—apply weak solution of sodium bicarbonate solution to the affected parts.

Lung Irritants (Asphyxiants, Choking Gases)

Lung irritants (Table 36.5) include chlorine (Cl_2) and phosgene ($COCl_2$), which are gases, but are hot liquid under pressure. Chloropicrin, diphosgene are liquids. All of these war gases are used in shells. Phosgene is ten times and chloropicrin is four times more toxic than chlorine. Phosgene and diphosgene are called *'green cross.'*

Table 36.5: Lung irritants and their toxicity and odour

Types	*Forms*	*Odour*	*Toxicity*
Chlorine	Gas	Bleaching powder like	2-21/2 hr
Phosgene	Gas	Musty hay like	1-2 hr
Chloropicrin *	Liquid	Fly-paper like	Slow
Diphosgene *	Liquid	Like phosgene	Slow

* *Are liquid*

Action

These war gases can be fatal and kill the victim. They mainly act on the alveoli. Inhalation can bring about dyspnoea, tightness in the chest, cough, irritation of conjunctivae followed by restlessness, rapid and stertorous respiration (breathing having a heavy snoring sound), cyanosis, followed by collapse and death within 1 to 2 days. Cause of death is acute pulmonary oedema.

Treatment

- Wearing of *gas mask* to prevent the exposure
- Shift to clean atmosphere,
- Give oxygen and adrenaline.
- Eyes – irrigation/ washing with normal saline/ boric acid.
- Symptomatic measures (e.g. codeine for cough, and antibiotics for respiratory tract/lung infection, etc.)

Postmortem Findings

Usually those of asphyxia, other findings could be those of bizarre injuries due to bursting of shells containing the gas.

Vesicants (Blister Gases)

These include (Table 36.6) mainly mustard gas (dichloroethyl sulphide, yellow cross, yperite, etc.) and lewisite, which are both liquid form. Like the lachrymators, vesicants also need to be fired during an attack in the artillery shells so as to saturate the area of attack.

Action

- Mustard gas can produce severe irritation of eyes, nose, throat, and respiratory passages.
- It can also develop severe irritation of the skin especially over the oily areas, such as face, axillae, pubis, scrotum, etc resulting in redness, *vescication/blister formation* (of varying size), and ulceration especially over the moist areas. It can even pass through the worn clothes and also produce intense itching of the skin.
- There can be inflammation of stomach leading to nausea, vomiting, pain abdomen, diarrhoea, etc., (mimics arsenic poisoning)
- Rarely death can occur due to bronchopneumonia.

Table 36.6: Types of vesicants, their form, odour and toxicity

Vesicants	*Forms*	*Odour*	*Toxicity*
Mustard gas *	Volatile gas	Garlicky	Slow irritant
Lewisite	Liquid	Geraniums	Rapid

(Dichloroethyl sulphide, yellow cross, yperite, etc.)*

Treatment

- Gas mask and special cloth covering (to protect skin)
- Remove contaminated clothing worn and wash the body with soap and water
- Use solution of sodium bicarbonate for irrigation of eyes (2%) and nose (5%).
- Symptomatic measures constitute rest of the treatment in a victim.
- BAL is a good antidote for lewisite.

Sternutators (Nasal Irritants, Vomiting Gases)

These are solid compounds of arsenic, requiring firing in an artillery shell. e.g. diphenyl chlorarsine (DA)(or sickening gas), diphenylamine chlorarsine (DM), diphenylcyanarsine (CD). Diphenyl chlorarsine is six times heavier than air.

Action

Diphenyl chlorarsine can act upon the vomiting centre in the brain. Vapours can result in intense pain and irritation in the nose and sinuses leading to sneesing, malaise, and headache, salivation followed by nausea, vomiting and chest pain. Additional symptoms as those of arsenic poisoning may also be observed.

Treatment

Gas mask, symptomatic, sodium bicarbonate for washing.

Paralysants (Nerve and Blood Poisons)

They are hydrocyanic acid (HCN acid), carbon monoxide (CO), hydrogen sulphide (H_2S) (refer specific sections).

Nerve Gases

They are compounds related to phosphate esters in action and toxicity. They are colourless and odourless volatile liquids. They are absorbed from lungs, gastrointestinal tract, skin and conjunctivae. These nerve gases are chemicals with acetylcholine like action or have the action of inactivating cholinesterase enzymes leading to accumulation of acetylcholine. Some of the known war gases are *serin* and *tabun*.

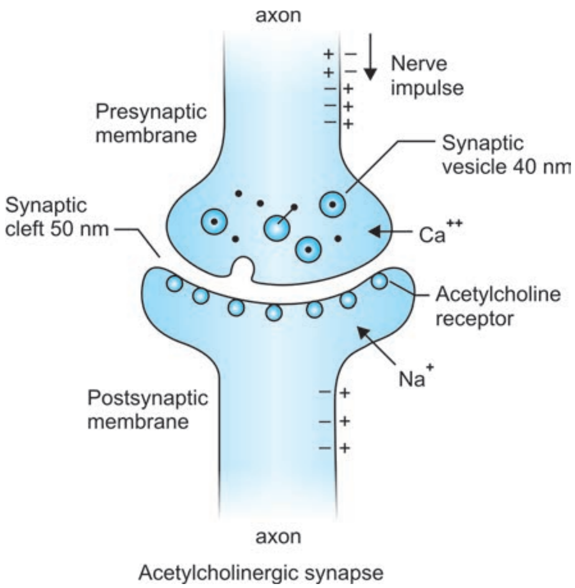

Fig. 36.1: Illustrating mechanism of action

Mechanism of Action[10]

Figure 36.1, Illustrates how nerve gases act. Nerve gases act by shutting down the transmission of nerve impulses. How this is done is of interest because it involves a basic biological mechanism that is of considerable intrinsic interest. Nerve cells transmit information electrically, by means of the state of polarisation of the cell membrane. The cell membrane is a very complex structure, with electrically-controlled channels that permit charged cations, such as Ca^{++}, Na^+ and K^+, to move across it to change electrical potentials and cause chemical reactions. The signals move along extensions of the cell called axons, and the axons of different cells are joined at synapses.

A synapse is shown diagrammatically at the right. The axons are immersed in the intercellular medium, and are filled with cytoplasm. The transmitting and receiving axons are separated by the synaptic cleft, about 50 nm wide. A nerve impulse is shown descending the upper axon. In the presynaptic membrane, acetyl coenzyme A is busy transferring the acetyl radical to the small molecule choline to make acetylcholine, represented here by small black dots. About 104 acetylcholine molecules are collected as groups in synaptic vesicles, about 40 nm in diameter. When a nerve impulse reaches the synapse, the change in membrane potential opens the Ca^{++} channel, and Ca^{++} ions enter the synapse, where they cause vesicles to adhere to the membrane and penetrate it, releasing acetylcholine into the synaptic cleft. That is, the electrical nerve impulse causes acetylcholine to be secreted in the synapse.

The acetylcholine diffuses rapidly across the 50 nm, and is received by acetylcholine receptors in the postsynaptic membrane. Within 100 μs of the arrival of the acetylcholine, the sodium channel in the membrane opens, allowing Na^+ to enter and reverse the membrane potential. This reversal then propagates forward as a nerve impulse in the receiving axon. To be ready for the next impulse, the original state must be restored in the postsynaptic membrane, and this requires removal of the acetylcholine that caused the disturbance. This is carried out by a protein enzyme of molecular weight 260,000 called acetylcholinesterase. There is a particular serine residue in the protein that binds the acetylcholine and holds the acetyl while letting the choline go. This cleavage occurs in about 40 μs. The enzyme then loses the acetyl by hydrolysis, and is ready for another job. In this way, a synapse can handle up to 1000 nerve impulses in a second.

One way to interfere with this transmission is to render the acetylcholine receptors inactive. Curare, the famous arrow poison of Brazilian natives, can do this. The disease myasthenia gravis is caused by an autoimmune attack on the receptors. Tetrodotoxin, the powerful venom of the Puffer fish and certain marine microorganisms, shuts down the sodium channel. Respiratory paralysis is the usual fatal effect of interference with nerve function.

Certain organophosphates bind strongly with the serine residue of acetylcholinesterase, rendering the enzyme inactive. Then, the acetylcholine builds up and the receptive state of the postsynaptic membrane cannot be re-established. This is the mode of action of war gases. These have small molecules that can penetrate the body easily.

However, none of these gases are announced as they are still held under military secrets.

Signs and Symptoms of Nerve Gas Poisoning

- Pupils constrict (pinpoint pupil)
- Rhinorrhoea, bronchial spasms
- Ciliary spasm—pain in eyeballs leading to photophobia
- Sudden unconsciousness and bradycardia followed by cardiac arrest and death.

Miscellaneous War Gases

- Yellow/red rain
- Methyl isocyanate.

Yellow/red rain

Laotian tribesmen often struck by two gases coloured yellow or red. It is a combination of mustard gas, phosgene, chlorine and nerve poison.

Symptoms

- Victims feel as if their body is getting blown up
- Cough and haemoptysis, followed by painful breathing
- Burning in the throat resulting in dysphagia
- Eyes may turn yellow, with blurring of vision
- Nose-tingling as if hot pepper has been inhaled
- Necrosis of the gum with loosening of the teeth
- Skin may necrotize with red to blue colouration
- Rise in body temperature.

Fatal period 2 weeks.

METHYL ISOCYANATE (MIC)[12,13]

Classification

Methyl isocyanate can be considered under several classifications, namely:

- Agro-chemicals
- War gases
- Irritants
- Miscellaneous poisons.

Properties

- Methyl isocyanate has a pungent but sweet odour.
- It is a fairly stable liquid at room temperature (except in summer)
- Has a boiling point of 31°C
- It reacts with water/moisture, alkaloids and most other common solvents. Hence, needs to be preserved under inert conditions.

Medicolegal Importance

Mostly accidental poisoning e.g.: *Bhopal gas tragedy*.

Action

MIC is a deadly substance used in pesticide and pharmaceutical industries.

- Can kill in small doses
- Can get absorbed orally, through respiratory tract or through intact skin.

Signs and Symptoms

Manifests in three forms: Acute, subacute, and chronic form.

Acute poisoning locally MIC can produce irritation to skin, eyes and mucous membranes, resulting in severe burning sensation in the throat, unbearable irritation of the eyes followed by severe chest pain with labored breathing. Death is usually due to pulmonary oedema in untreated cases.

Subacute poisoning occurs when person survives for more than 5 to 6 days after acute poisoning. The condition manifests now with more of neurological effects such as motor weakness, paralysis, convulsions, coma, and cerebral oedema leading to death.

Delayed effects develop in a victim who survive for more than a week and manifest with dyspnoea, jaundice, weakness of limbs, etc. followed by exhaustion and death.

In pregnant woman victims the delayed effects of the poison may be abortion, congenital malformation, still-birth, etc.

CYANIDE (PRUSSIC ACID, CYANOGEN)[2,5,12-19]

Properties

- Cyanide occurs as a gas or a liquid or a solid.
- In a gaseous state is referred to as hydrogen cyanide (HCN),
- The liquid form it is called as *hydrocyanic acid* or *Prussic acid* or *cyanogen* is a solution of either 2 per cent or 4 per cent of HCN in water. Pure acid is a colourless, transparent volatile liquid with a penetrating odour of *bitter almonds*. However, about 20-40 per cent of population cannot smell the gas and the ability to detect the smell is a sex-linked recessive trait.[14] On exposure to light it gets decomposed rapidly.
- Cyanide salts occur as solids and these cyanides of sodium/ potassium are white powders.
- Cyanides are considered as a *cardiac poison* also.

Action

Cyanides are protoplasmic cytotoxic poisons. They act by inhibiting *cytochrome oxidases* for utilising O_2 in a cell, leading to internal asphyxia (*cellular asphyxia*), leading to death. It can also act on other enzymes mildly. It also acts as a corrosive on mucosa.

Sources

- **Natural**: in fruits and leaves such as bitter almond, apricot, cherry, peach, plum, etc. (in their leaf, bark and seeds); apple, pear, (in their seeds); bamboo shoots, cherry laurel (in their leaves); cassava (tapioca/ manihot), lima beans, etc (in their fruit/ bean and root), as harmless *glucoside amygdalin*. However, emulsin enzymes can hydrolyze these and release hydrocyanic acid.
- HCN is the normal constituent of the human body (15 – 30 micrograms).
- Burning of plastic furniture, silk or wool, and cigarette (smoking) has traces of cyanide.

Uses

- HCN gas—used for fumigation of ships
- It is often used in laboratories in various laboratory processes and in photography, electroplating, coating silver, case hardening of steel/ iron, in tanning industries, etc.
- Calcium cyanide is utilized as fertilizer.

Absorption

Rapid absorption is possible through both skin and mucous membrane of respiratory tract and stomach. On ingestion of cyanide salts they react with acids (e.g. HCl) in the stomach and liberate HCN acid. Enzymes rhodanese present in the mitochondria of liver and kidneys metabolises cyanides converting it into cyanocobalamin (vitamin B_{12}) in the presence of hydroxycobalmin (vitamin B_{12a}). A small amount of cyanide is excreted in the breath and sweat producing bitter almond odour.

Causes of Death

Death is mainly due to either respiratory paralysis or *cytotoxicity*.

Signs and Symptoms

The clinical manifestation of poisoning presents in two forms: Acute and chronic.

Acute Poisoning

All symptoms reflect cellular hypoxia and symptoms shift rapidly depending on the extent of cyanide exposure. Inhalation brings about death instantaneously by respiratory arrest. However, consumption of large dose orally, though brings about death rapidly; is slow compared to the inhalational route. Also cases with massive dose of oral poisoning present with a few voluntary acts such as corking a bottle or throwing a stone or even walking certain distance and then dropping down dead. If, death is delayed for few minutes, the victim may complain of agonising dyspnea, followed by convulsions. These differences in onset of manifestations are basically on account of slower absorption into the circulation and also metabolism of some of it while passing through liver.

Central Nervous System

- Headache, guiddiness, anxiety, agitation, confusion, convulsions, and coma. Eyes are glassy and prominent with unresponsive pupils.
- Violent convulsions, clenched jaw, loss of muscle power and loss of consciousness, ultimately leading to death

Cardiovascular System

- Initially there will be hypertension with reflex bradycardia, sinus arrhythmia, followed by tachycardia and hypotension ventricular dysarhythmias, and cardiac collapse.

Gastrointestinal System

- When taken orally, victim may complain of bitter, acid, burning taste; constriction and numbness of throat; salivation, nausea and rarely vomiting.
- Frothing and corrosion of mouth may also be noticed.
- There will be smell of bitter almonds around mouth and breath.

Respiratory System

- Respiratory system shows initial tachypnoea followed by dyspnoea, bradypnoea, with severe respiratory depression and cyanosis.

Skin

- Skin and mucosa will be brick red in colour and characteristics. This is said to be due to increase in haemoglobin oxygen saturation in venous blood because of decreased utilisation of oxygen by tissues. Skin will be cold and clammy to touch. The skin turns *cyanotic* only in late stages.

Chronic Poisoning

Chronic poisoning comprises of exposure over a long period by inhalation of non-lethal dose of HCN acid vapors. Usual manifestations are:

- Headache, vertigo, nausea and vomiting
- *Visual defects* such as:
    - — Scotoma
    - — Progressive loss of vision (*Tobacco amblyopia*)—in the victim who is *chronic smoker*.
    - — Optic atrophy (*Leber's hereditary optic atrophy*) – due to the sensitivity of optic nerve to cyanide, is a congenital defect of *rhodanese deficiency* seen only among men.
- Tropical ataxic neuropathy—This is a condition with clinical manifestations of peripheral sensory neuropathy, optic atrophy, ataxia, deafness, glossitis, stomatitis and scrotal dermatitis. This is common among the victims who eat large quantities of tuber *tapioca* (Cassava/manihot) containing cyanogens, which they fail to remove by following proper fermentation techniques.

Fatal Dose

Varies with toxic substance used:

- Pure acid 60 mg
- Any pharmacological preparation 30 drops
- Crude oil of bitter almond 60 drops
- Potassium cyanide 200 mg
- Air pollution:
    - — 0.15 mg HCN/litre→Death in 30 to 60 minutes
    - — 0.3 mg HCN/litre→Death instantaneously.
    - — 1: 500 concentration→Death instantaneously.

Fatal Period

- In some cases—immediate death
- However, an average for HCN acid is 2 to 10 minutes; and of potassium cyanide is 30 minutes.

Treatment

Must be immediate, with specific antidotes. Never delay.

Principle

Reversal of cyanide-cytochrome combination done by:

- Giving nitrites to convert haemoglobin into methaemoglobin which combines with cyanide forming non-toxic cyanmethaemoglobin
- Giving sodium thiosulphate, which combines with cyanides and forms a nontoxic thiocyanate, which can be excreted in the urine.

Schedule of Treating Cyanide Poisoning

Stabilisation

Assisted ventilation, giving 100 per cent oxygenation, cardiac monitoring, IV access, treatment of metabolic acidosis, and vesopressors for hypotension constitute various methods of stabilising the victim of cyanide poisoning.

Decontamination

With orally ingested cases decontamination is done by performing gastric lavage preferably with 5 per cent sodium thiosulphate solution, or activated charcoal, and then cathartics are administered. However, in a skin contamination cases remove clothes of the patient and wash the skin with soap and water.

Administration of Antidote

Specific Antidote—Amyl Nitrite Therapy

Known as *Way JL Approach/Cyanide Kit Approach*. It involves three steps:

- ***Step I***: 0.2 ml ampoule/*perle* of amyl nitrite broken into a handkerchief and the victim is made to inhale for 30 seconds, every 3 min. Stop amyl nitrite if systolic BP falls below 80 mm of mercury. (Ampoule/*perle* of amyl nitrite is crushed in a handkerchief to avoid injury to hand and also facilitates faster inhalation)

- ***Step II***: 10 ml of 3 per cent solution of sodium nitrite is injected I/V slowly at a rate of 2.5 to 5 ml per minute followed by
- ***Step III***: 50 ml, of 25 per cent solution of sodium thiosulphate at the same rate, by the same needle in the vein.

Other Antidotes

Three antidotes for cyanide poisoning are widely recommended for use in the UK, namely:

- Oral administration of *'solution A' and 'solution B'* comprising of ferrous sulphate dissolved in aqueous citric acid, and aqueous sodium carbonate respectively
- Nasal inhalation of *Amyl nitrate*
- Intravenous administration of *Dicobalt edetate* (*Kelocyanor*).

However, it should be remembered that—the mixture of solutions A and B is only of value in reducing the absorption of the swallowed cyanide, whereas the majority of accidental exposures are by *inhalation* or *skin contact*. These solutions also have a very *limited shelf life*. A recently published review of the use of this antidote has questioned the efficacy of the solutions and drawn attention to their inappropriate use. It is reported that *iron poisoning* may result when the solutions have been used incorrectly. Thus this antidote is *not* recommended recently.[17]

Latest Therapy[16-19]

- Dicobalt edetate (*Cobalt-EDTA*, Trade name: *Kelocyanor in Britain and France*)—20 ml of 1.5 per cent (maximum 300 to 600 mg) is given slow I/V injection, followed by 20 ml of 50 per cent glucose. It acts by *chelating* the cyanide forming a harmless product, which is excreted in urine.
- 50 ml of 1 per cent sterile aqueous solutions of methylene blue (methyl thionine chloride VSP), which was once considered as a potential antidote acts by forming Hb-met-haemoglobin, is presently considered as ineffective and *is therefore not recommended* for cyanide toxicity.
- Hydroxycobalamine (vitamin B_{12} precursor) available as solution of 1000 mg/ml – given at a dosage of 50 mg/kg in 3.5 litres IV infusion. It acts by formation of cyanacobalmin (vitamin B_{12}), which is excreted in the urine.
- PAPP (para-aminoprepriophenone) is recommended recently as an antidote to form methaemoglobin. However, its action is slow.
- **HSE Regime:**[17,18] HSE (Health and safety executive) suggest that *speed* is essential in treating a case and also advice to obtain *immediate medical attention* of the patient. It also proposes that treating personnel should protect *themselves* and the *casualty* from further exposure during decontamination and treatment. HSE will *no longer* recommend the *use of any antidote* in the first aid treatment of cyanide poisoning and will not require employers to keep supplies. It recommends only *administration of oxygen and artificial respiration*. HSE advises that administration of *oxygen* is the most useful initial treatment for cyanide poisoning. If breathing has stopped, artificial respiration is essential. However it is better not to perform mouth-to-mouth resuscitation as it has a possible risk of secondary poisoning to the first-aider. Suitable, simplest mechanical resuscitation device is a bag and mask device connected to an oxygen supply.

Prognosis

Survival of the victim for 4 hours after the poisoning is usually a good prognosis and is usually followed by recovery.

Postmortem Appearances

Postmortem appearances are mainly those of asphyxia.

External

- Smell of bitter almonds near the body
- Face, lips (Fig. 36.2A) and body surfaces—irregular pink patches or show cyanotic tinge rarely
- Fine froth at the mouth
- Eyes—bright, glistening and prominent with dilated pupils
- Rigor mortis sets early.

Internal

- Characteristic bitter almond smell in stomach, serous cavities and brain
- Stomach mucosa may be pink in colour (Haemorrhagic gastritis) (Fig. 36.2B)
- Blood-stained froth in trachea/bronchi
- Brain is oedematous and meninges—hyperaemic
- Lungs oedematous, pleura and pericardium may show petechial haemorrhages
- Blood spectroscopy can show characteristic bands
- With potassium cyanide can show corrosion of mouth, brick red gastric mucosa.

Viscera Preserved for Chemical Examination

Biological fluid – Blood

Viscera – Stomach with contents, lungs, liver, kidneys, brain, heart, and spleen. Spleen is said to be the best specimen for cyanide analysis as it has highest concentration of poison owing to a strong presence of RBC.

Precautions — Person doing autopsy should wear gloves and mask to prevent absorption through skin, contamination or respiratory tract inhalation.

Medicolegal Aspects

- Hydrocyanic acid/cyanide is a powerful poison
- Commonly used as suicidal agent (cyanides are three times more commonly used than the acid). It fulfills *ideal suicidal agent* qualities such as:
  - — It can be taken orally
  - — It has a pleasant taste
  - — It has no repulsive odour
  - — It is cheap and easily available, etc.
  - — e.g. '*cyanide capsule*' of Nazi German's in World War II. The German soldier carried it in their shirt collars for committing suicide whenever arrested by the enemy soldiers, avoiding torture for military secrets. The similar practice may be noticed among the *Sikh militants* in India and *LTTEs* of Sri Lanka.
- Accidental incidences occur occasionally, e.g. after fumigation, eating bitter almonds, etc. Eating the seeds of apricot, peach, plum, pear and apple, may lead to fatalities occasionally. Fires involving polyurethane furniture (plastics), silk or woolen articles, can release cyanide and this may result in accidental deaths of people exposed to it.
- Gas cannot be absorbed through skin (especially wet)

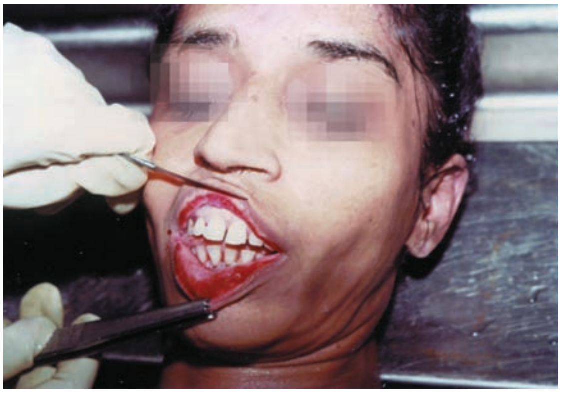

Fig. 36.2A: *Asphyxiants:* Cyanide poisoning – Note the bright red colour of the lip (*Courtesy:* Capt. Dr Santhakumar, Professor and Head, Dept of Forensic Medicine, Government Stanley Medical College, Chennai, Tamil Nadu)

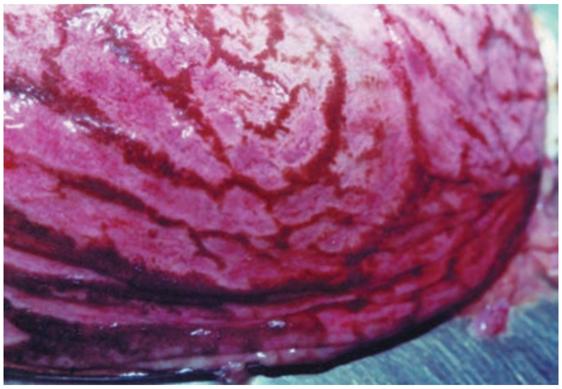

Fig. 36.2B: *Asphyxiants:* Cyanide poisoning – Note the stomach changes - bright red appearance of stomach mucosa (*Courtesy:* Capt. Dr Santhakumar, Professor and Head, Dept of Forensic Medicine, Government Stanley Medical College, Chennai, Tamil Nadu)

- *Cherry laurel—water* is often referred to as *holy sacrament.*
- Homicide—rare due to peculiar smell and taste. However, Hitler is known for killing many thousands of innocent Jews by *cyanide gassing* in the abominable concentration camps of Second World War. The *Jonestown Massacre* of 1974 is another recent time example for mass killing members of religious group called *People's Temple* in California, in which mainly mentally afflicted individuals, physically handicaps and cripples, drug addicts, exconvicts, etc were made to drink cyanide solution. Also in some of the states of USA, cyanide gas has been used in *execution* of criminals sentenced to death legally. The criminal is taken into a room called '*gas chamber*' and made to sit there, closed and the gas is released in fatal quantity for a fatal period.
- HCN is extremely volatile substance and therefore viscera or other specimens for chemical examination must be sent in airtight bottles
- Double cyanides (Potassium ferrocyanide and Potassium ferricyanide can kill if taken with acids otherwise not poisonous)
- Embalming can remove/destroy cyanides
- Small amount of cyanides may be formed in the tissue due to *putrefaction.*[19]

AMMONIA (NH_3)[2,5,20]

History

The literature on ammonia toxicity in humans largely consists of case reports. A literature review, reports of 94 previously reported cases; 20 resulted in fatality and 35 needed clinical follow-up for about one year and more. Despite lack of data, most literature is consistent regarding clinical presentation and treatment of ammonia toxicity.[20]

Properties

At room temperature, ammonia (NH_3) is a highly water-soluble, colourless, irritant gas with a unique pungent odour. Ammonia has a boiling point of –33°C and an ignition temperature of 650°C.

Uses

- The farming industry uses *anhydrous ammonia* as a component of fertilizer and animal feed.
- Before the 1970s, liquid ammonia stored under high pressure was widely used for refrigeration. Although *Freon* largely has replaced ammonia as a refrigerant, ammonia refrigeration is still used and numerous case reports exist of severe toxicity following accidental exposure.
- Ammonia also is used in the production of explosives, pharmaceuticals, pesticides, textiles, leather, flame-retardants, plastics, pulp and paper, rubber, petroleum products, and cyanide. Furthermore, ammonia is a major component of many common household cleaning and bleaching products (e.g. glass cleaners, toilet bowel cleaners, metal polishes, floor strippers, wax removers, smelling salts).

TWA, STEL AND IDLH FOR AMMONIA

Permissible levels of exposure to toxic gases are defined by time-weighted average (TWA), short-term exposure limit (STEL), and concentration at which toxic gasses are immediately dangerous to life or health (IDLH). Anhydrous ammonia has a TWA of 25 ppm, an STEL of 35 ppm, and an IDLH of 500 ppm.

Usual Fatal Dose

- Gaseous form: 0.5 per cent in air
- Liquid form: 10 to 20 ml.

Pathophysiology

- The most common mechanism by which ammonia gas causes damage is when anhydrous ammonia (liquid or gas) reacts with tissue water to form the strongly alkaline solution, ammonium hydroxide.

$$NH_3 + H_2O\ NH_4OH$$

- This reaction is *exothermic* and capable of causing significant thermal injury.
- Although injury from ammonia most commonly is caused by inhalation, it also may follow ingestion or direct contact with eyes or skin.
- Because of its high water solubility, ammonia has a tendency to be absorbed by the water-rich mucosa of the upper respiratory tract. However, unlike most highly water-soluble irritant gases that tend to affect exclusively the upper respiratory tract, ammonia can damage proximally and distally.

Signs and Symptoms

Inhalation: *Head, ears, eyes, nose, and throat (HEENT)* symptoms include running nose and increased salivation.

Ammonia inhalation can produce tachypnoea, oxygen desaturation, stridor, drooling, cough, wheezing, rhonchi, and decreased air entry asthma, severe upper respiratory tract irritation, pneumonia, pulmonary oedema, bronchitis, and obstructive lung disease. Death is usually due to bronchopneumonia.

Ingestion: *HEENT* symptoms include oedema of the lips, oropharynx, and upper airway. Ammonia ingested can produce intense pain, dysphagia followed by oesophageal stenosis. Patient may experience epigastric tenderness; mediastinitis and peritoneal signs may be present with viscus perforation, which can occur as late as 24-72 hours postingestion.

Contact – Can result in skin burns (alkali burns) and cold injury. Facial and oral burns and ulceration may be seen. Alkali burns to the skin are yellow, soapy, and soft in texture. When burns are severe, skin turns black and leathery.

Eye: Exposures results in watering of the eyes, corneal damage, conjunctivitis and palpebral oedema. Ammonia typically causes more corneal epithelium and lens damage than other alkalis. Intraocular pressure and pH of the anterior chamber rise, resulting in a syndrome similar to acute narrow-angle glaucoma. Other symptoms include iritis, corneal edema, semidilated fixed pupil, and eventual cataract formation. Blindness may be the serious consequence in severe cases.

Treatment

Management of toxic exposure to ammonia is largely supportive, and medical therapy is directed at hypoxia, bronchospasm, pulmonary oedema, hypovolaemia, and burns of the skin and eyes and includes:

- Stomach wash and emetics use is ***not*** recommended.
- Giving demulcents, and dilute solution of vinegar are useful.
- Tracheostomy, oxygen administration and assisted ventilation may help to alleviate respiratory distress.
- Antibiotics and corticosteroids are controversial therapies following ammonia inhalation and ingestion exposures. Corticosteroids are administered to decrease the incidence and severity of oesophageal strictures that occur during healing from significant alkaline injuries. Antibiotics are given because of increased risk of mediastinitis associated with full-thickness oesophageal alkaline corrosive burns. If steroids are administered, the recommended dose is 1-2 mg/kg/d of methylprednisolone for 3 wk followed by gradual tapering. If antibiotics are administered, a broad-spectrum antibiotic (second-generation cephalosporin) is appropriate. The decision to continue or stop corticosteroid and antibiotic therapy is based on endoscopic findings.
- Lesions of skin and eye needs thorough washing with copious amount of water. Diluted vinegar may be applied to skin after washing.

Postmortem Findings

Following changes are observed:

- Characteristic odour of ammonia.
- Brownish staining of affected skin.
- Grayish sloughing of affected mucosa.
- Congestion of GI walls (rarely perforations) and respiratory tract and pulmonary oedema.

Medicolegal Importance

- Ammonia is liberated during combustion of nylon, silk, wood, and melamine, etc which renders firefighters are at risk for exposure to this irritant gas.
- Ammonia is often thrown on a victim to facilitate robbery.
- Cases of industrial accidental poisoning are more common with ammonia. Suicide with ammonia is also common. However, homicide is extremely rare due to its odour.

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37 Chapter

Domestic Poisons

The poisons in daily use may be conveniently dealt with in three groups, (i) *domestic or household poisons,* (ii) *poisoning by medicine, and* (iii) *food poisoning.*

DOMESTIC/HOUSEHOLD POISONS

Some of the *common* domestic poisons in India, discussed here under two heads:

- Hydrocarbons
- Other than hydrocarbons
  - — Dishwashing powders and granules
  - — Dishwashing liquids
  - — Household detergents
  - — Disinfectants
  - — Metal polishes
  - — Cosmetics.

HYDROCARBONS

Hydrocarbons are broad group of organic compounds that contain a carbon and hydrogen only. They are of three verities:

- *Aliphatic* (straight chain),
- *Aromatic* (containing a benzene ring)
- *Halogenated* hydrocarbons (These discussed earlier).

Aliphatic Hydrocarbons

These are petroleum distillates which are common constituents of several industrial and household products and are involved in accidental poisoning, especially among the children. It is reported that every year nearly 28,000 children under the age of 5 years ingest petroleum distillates and accounts for 12 to 25 per cent death due to all poisons in this age group.[1,2] Aliphatic hydrocarbons could be of two types *low* and *high* molecular weight.

I. Low molecular weight — These include gaseous or non-liquid form and liquid forms:

Non-liquid forms/Gaseous forms — include *methane* (CH_4), *ethane* (CH_3CH_3), *propane* ($CH_3CH_2CH_3$) and *butane* ($CH_3(CH_2)_2CH_3$), all of which are flammable gases. Natural gas is primarily *methane* and *ethane*. Liquefied petroleum gas (LPG, 'bottled gas') contains propane and butane.

Liquid forms — are petroleum distillates, are break down products remaining after processing crude oil. Some of the examples are *fuels* such as ***kerosene, diesel oil, gasoline (petrol)***, and *furniture polish* like ***mineral seal oil***, and *naphtha*. Kerosene, diesel oil and petrol are inebriant type of cerebral poison and are separated by distillation at various boiling points.[3] Petrol, naphtha, etc are derived at 50-100°C and kerosene is derived at 150-300°C.

II. High molecular weight — These include:

Hydrocarbons of petroleum distillate origin: *Petroleum jelly* (Vaseline), *paraffin wax*, etc and are petroleum distillates and are derived at 300°C and above boiling points. Toxicity decreases as the boiling point increases. These are hence relatively nontoxic.

Hydrocarbons of non-petroleum distillate origin: Hydrocarbons do not necessarily have to be petroleum distillates. *Turpentine* and *carbon tetra-chloride* are hydrocarbons but are not petroleum distillate products.[1] *Turpentine* (paint thinner/paint remover) — is a hydrocarbon made of pine oil, is an *aromatic hydrocarbon* but with the properties of an aliphatic hydrocarbon, and hence dealt with under aliphatic hydrocarbons. *Rectified turpentine* has therapeutic uses as counter irritant.

Toxicity Rating

Vaseline and paraffin are nontoxic and used medicinally, whereas *petrol, naphtha* and *kerosene* are highly toxic by ingestion or inhalation. Table 37.1, below provides toxicity rating and uses of some of the aliphatic hydrocarbons.

Mode of Action

Low molecular weight hydrocarbons have a very low viscosity, and toxicity is basically due to its aspiration into the lungs. In

Table 37.1: Toxicity rating and uses of aliphatic hydrocarbons

Compounds	*Toxicity rating*	*Uses*
Diesel oil	3	Fuel
Gasoline (petrol)	3	Fuel
Kerosene	3	Fuel, solvent cleaning agent
Mineral seal oil	–	Furniture polish
Turpentine	–	Paint thinner, paint remover
Methane, ethane, butane, propane, etc	–	Butane used as lighter fuel
Paraffin wax, petroleum, jelly (vaseline), etc	1 (Relatively non-toxic)	Both – medicinal use – industrial use

the lungs these liquid nongaseous hydrocarbons can spread rapidly throughout the pulmonary tree resulting in to fulminating pulmonary oedema and bronchopneumonia. Aspiration can also occur during vomiting. The gaseous hydrocarbons can act directly as asphyxiants. Certain high molecular hydrocarbons also cause respiratory manifestations on aspiration. However, the pneumonia resulting is more localized and less inflammatory.[1-5]

Toxic petroleum distillates are *irritants* and are absorbed orally as well as nasally. It dissolves in fat, hence can act on nervous tissues. They can bring about *depression* of CNS and also *damage* the liver, kidneys and bone marrow. Thus the presenting symptoms and signs are usually related to three main organ systems: pulmonary, central nervous and gastrointestinal. The cardiovascular system, kidneys, liver, spleen, and blood also may be involved.[1]

Signs and Symptoms

Poisoning can manifest in two forms.[3-7]

Acute Poisoning

Oral ingestion of fatal doses can result in:

- Odour — a characteristic odour specific to hydrocarbon ingested is appreciated in the vicinity of the patient.
- Pulmonary system — peculiar odour is usually evident in breath, vomitus. Cyanosis can occur due to pulmonary complications such as bronchopneumonia. Aspiration can produce coughing, choking, gasping, bronchospasm, and hypoxia. Severe cases may present with haemoptysis. Haemorrhagic pulmonary oedema can result in pink, frothy sputum progressing to shock and cardiac arrest.
- Central nervous system — depression resulting in vertigo, giddiness, drowsiness, headache, tremors, convulsions, etc. Toluene sniffing may present with drunken appearance.[4] Pupils are usually constricted initially but later on dilated when coma supervenes.
- Gastrointestinal tract — ingestion of the poison results in pain, burning pain in throat, nausea, vomiting, colicky abdomen, diarrhoea, etc.
- Cardiovascular system — cardiomyopathy, arrhythmias, etc.
- Renal and hepatic system — less commonly involved.
- Haemopoietic system — blood may show aplastic anaemia and agranulocytosis.

In fatal cases:

- Drowsiness merges into coma and death due to respiratory failure.
- There may be intense excitement, hallucinations and convulsions, cyanosis, unconsciousness, profound coma and death.

Chronic Poisoning

Causes Usually occurs among those who inhale petroleum products in petroleum industries resulting in manifestations such as:

- Skin – chronic eczematoid dermatitis, with redness, itching, and inflammation. Cutaneous exposure to gasoline and other hydrocarbons can cause second degree burns.[4]
- Dizziness, weakness, weight loss, anaemia, nervousness, pain in limbs, peripheral numbness, paraesthesias, etc.

Fatal dose — Depends (10 -15 ml of kerosene). Even a few ml on aspiration can cause serious toxicity.

Fatal period — Few hours to one day.

Treatment

- Wash the contaminated skin with copious amount of water and soap
- Give liquid paraffin orally — dose 250 ml. It dissolves kerosene and reduces its absorption
- Activated charcoal in large doses is recommended, though petroleum distillers are not adsorbed.[6]
- Saline purgatives may also be useful
- Avoid gastric lavage for the fear of aspiration. However, gastric lavage with warm water containing 5 per cent sodium bicarbonate administered with extreme care to prevent entery into respiratory tract is beneficial with large amount of kerosene consumption (more than 4 ml/kg weight)
- Avoid intravenous fluid overload, as it may precipitate pulmonary oedema.
- Rest of the treatment includes symptomatic measures
- Chronic case of poisoning — isolate and prevent further absorption of poison.

Postmortem Findings

- Frothy oozing at the mouth and nostrils with characteristic odour specific to the type of hydrocarbon ingested.
- Other findings will be of asphyxia.

Table 37.2: Aromatic hydrocarbons: uses, signs, symptoms, and treatment of toxicity

Compounds	*USD*	*TR*	*Uses*	*Signs and symptoms*	*Treatment*
Benzene	15–20 ml	4	As solvent, and a degreaser	• CNS – Vertigo, lethargy, convulsions and coma. • GI Tract – Abdominal pain, vomiting and diarrhoea • RS – Pulmonary oedema and pneumonia. • CVS – cardiac arrhythmias, blood dyscrasias. • Renal and hepatic-damage • Metabolic acidosis is common in toluene poisoning. Chronic exposure particularly to benzene can result in anaemia, leukaemia and CNS damage, etc.	• Stomach wash can be done with caution after cuffed endotracheal incubation • Other treatment measures are essentially the same as for aliphatic hydrocarbons
Toluene	—	—	As solvent in cements and glues		
Xylene	—	—	• As solvent for rubber • In manufacture of terylene • In histological procedures in laboratory, etc.		

UFD - Usual fatal dose, TR - Toxicity rating

Table 37.3: Domestic poisons other than hydrocarbons

Compounds	*Types*	*Signs and symptoms*	*Treatment*
Dishwashing powders, granules	Irritant/corrosive	Irritation of mouth, throat, oesophagus and stomach seen especially in children	The stomach need not be emptied. Give milk, drinks of water, or fruit juice. Endoscopy is helpful in doubtful cases
Dishwashing liquids	Irritant/caustic	Swallow may be manifested by choking, retching and coughing is sufficient	Stomach wash, giving simple fluids to drink
Household Detergents	Soaps	Vomiting when taken orally	Stomach wash, giving simple fluids to drink is sufficient
Dettol and lysol *(Chlorinated phenols)*	Chemicals. May be admixed with isopropyl alcohol	Low toxicity, but can act like ethanol when swallowed if admixed with isopropyl alcohol	Stomach wash and any sequelae should be treated as for ethanol overdose Avoid stomach wash.
Metal polishes	May have petroleum hydrocarbon	Harmless when swallowed. However, bronchial aspiration can lead to major hazards	Just relieve any minor symptoms that are evident. Stomach wash, if large quantities ingested with palliative measures only
Cosmetics – hair dyes, hair conditioners, shampoos, bath oils, soaps	Non-toxic	In practice, be virtually disregarded toxicologically	

Table 37.4: Common domestic poisons with toxic substances in it

Preparations	*Toxic substances*
1. Babies and children	
(a) Baby powder	Boric acid
(b) Crayons (chalk)	Coloured by copper, arsenic, lead components
(c) Crayon (wax)	Paranitroaniline, azo dyes
(d) Fireworks	Arsenic, antimony, lead, thiocyanate, phosphorus,
(e) Toys (paints)	Lead, chromium, copper, etc.
2. Cosmetics	
(a) Cuticle remover	Potassium hydroxide, trisodium phosphate
(b) Depilatories	Barium sulphide
(c) Nailpolish removers	Acetone, ethylacetate
(d) Sun tan lotions	Denatured alcohol, methyl salicylate
3. Kitchen	
(a) Baking powder	Tartaric acid (mild irritant)
(b) Baking soda	Sodium bicarbonate (causes alkalosis in doses over 5 gm/kg)
(c) Dishwashing compounds (machine)	Sodium polyphosphates, sodium carbonate
(d) Domestic fuel	Kerosene
(e) Domestic gas	LPG (accumulated gas explodes with air when flame/spark is provided)
(f) Fire extinguishing fluids	Carbon tetrachloride, methyl bromide
(g) Matches	Antimony, phosphorus sesquisulphide, potassium chlorate
4. Rat poisons	
(a) Rat paste	Phosphorus, zinc/aluminum phosphide,
(b) Rodine (brown bran paste)	barium carbonate, thallium acetate
(c) Warfarin	Yellow phosphorus, it is a 4-hydroxy coumarin
5. Sanitary	
(a) Deodorants	Formaldehyde, naphthalene
(b) Drain cleaners	Sodium hydroxide
(c) Lysol	Phenol
6. Miscellaneous	
(a) Anti-rust products	Ammonium sulfide, naptha, oxalic acid
(b) Cleaning solvents (inflammable)	Petroleum hydrocarbons
(c) Cleaning solvents (noninflammable)	Carbon tetrachloride, trichloroethylene
(d) Dentifrices, mouthwashes	Hydrogen peroxide
(e) Furniture polish	Ordinary denatured spirit, resins, sodium hypochlorite (5%), oxalic acid
(g) Insecticide (spray)	Organochloro, organophosphorus and carbamate insecticides
(h) Lavatory cleaners	Mineral acids
(i) Marking ink	Aniline
(j) Moth balls	Naphthalene
(k) Paint removers	Sodium hydroxide, acetone
(l) Shoe polish	Aniline, nitrobenzene
(m) Straw hat cleaner	Oxalic acid

- Gastrointestinal tract may present with acute gastroenteritis findings. The stomach is congested and its contents produce a characteristic odour specific to type of hydrocarbon ingested.
- Lungs smell of kerosene and there will be the findings of pulmonary oedema and bronchopneumonia (i.e. pneumonic consolidation and emphysematous changes).
- Liver and kidneys show degenerative changes
- Bone marrow will be hypoplastic
- Preserve brain with other viscera for chemical examination
- Preservative used — saturated saline.

Medicolegal Importance

- Most of the cases are of accidental origin due to either industrial exposure or domestic accidents involving children.
- Since some of the hydrocarbons are easily available, they are often used to commit suicide by taking orally, or poured on the cloths and then ignited (self-immolation).
- High flammability of the fluid not infrequently renders them to be used for homicidal burning (dowry deaths). Homicide by oral route is extremely rare.
- However, most of the cases are accidental leading to poisoning or burning, e.g.—
  - — While sucking of petrol out from petrol tanks by a rubber tubing, choking due to sudden gushing of petrol into mouth and throat can sometimes take place.
  - — Inhalation of petrol fumes in petroleum industries.

Aromatic Hydrocarbons[3,4]

Most of the aromatic hydrocarbons are widely used in industry. Some of the examples are *benzene, toluene, xylene, styrene,* etc. Most of the aromatic hydrocarbons have characteristic odours and they are absorbed through inhalation, ingestion and direct skin contact. Both benzene and toluene are highly toxic, while xylene is relatively nontoxic. Table 37.2 provides the uses, signs and symptoms, treatment of toxicity by each of these.

Medicolegal Importance

- Domestic poisoning is not very common with these compounds. However, industrial exposure is common with them.
- Workers handling benzene need regular blood checks.
- Toluene is known for '*Glue sniffing*'.

DISHWASHING POWDERS AND GRANULES, LIQUIDS, DETERGENTS, DISINFECTANTS, METAL POLISHES AND COSMETICS

Brief accounts on these are given in the Table 37.3.[3,8,9]

DOMESTIC/HOUSEHOLD POISONS READY RECKONER

For the sake of convenience of reckoning some of the common poisons of household origin as well as insecticides are presented in Tables 37.4 to 37.6 for a quick reference.

Table 37.5: Medical household poisons with toxic substences in it

Preparations	*Toxic substances*
1. Antiseptics	Iodine, benzoin, phenol
2. Cough remedies	Codeine
3. Headache remedies	Asprin, phenacetin, analgin
4. Pep tablets	Benzedrine
5. Sleeping preparations	Barbiturates
6. Throat tablets	Potassium chlorate
7. Tonic syrup	Easton's syrup (strychnine)
8. Others	Antidepressants, tranquilizers, antibiotics, analgesics, etc.

Table 37.6: Garden poisons with toxic substances in it

Preparations	*Toxic substances*
1. Fungicides	Lead arsenate, copper compounds, organic mercurials, lime, sulphur
2. Insecticides, pesticides	Nicotine, tar oils, organochloro and organophosphorus compound carbamates, cyanides, etc.
3. Weed killers (herbicides)	Sodium chlorate, arsenious oxide and arsenites, dinitrocresol, paraquat

MEDICAL HOUSEHOLD POISONS

See Table 37.5.

GARDEN POISONS

See Table 37.6.

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38 Chapter

Poisoning by Therapeutic Substances

There are literally thousands of pharmaceutical substances (therapeutic drugs) that are potentially poisonous, if taken in sufficient dosage or for a long period. Toxic and fatal consequences are largely accidental or suicidal, as homicide by these compounds is rare. Though it is impossible to mention all such drugs in this book, certain medicinal compounds often involved in poisoning are enumerated: analgesics (aspirin, codeine, phenacetin, paracetamol, phenylbutazone, amidopyrine, etc.), antidepressants and sedatives, benzodiazepines, barbiturates, chloral, insulin, etc.[1-5] However, *Aspirin, Paracetamol* and *Insulin* are discussed here, as others in the list are already discussed earlier in the book.

ASPIRIN

Aspirin or acetyl salicylic acid is a non-narcotic analgesic and antipyretic.

Signs and Symptoms

Flushed face, oedema of face, skin rash, tinitus, deafness, hyperpnoea, nausea, vomiting, haematemesis, melaena, hypoprothrombinaemia, acute renal failure, pulmonary oedema, respiratory arrest.

Fatal dose 5 to 10 gm.

Fatal period Few minutes to few hours.

Treatment

Gastric lavage, leave some dilute $NaHCO_3$ in stomach. Restoration of electrolyte normality and acid-base balance. Vitamin K, blood or platelet transfusion, forced diuresis.

Medicolegal Importance

Accidental idiosyncracy and suicidal tendencies.

PARACETAMOL

Paracetamol (acetaminophen) is a non-narcotic analgesic and antipyretic.

Mode of Action

It acts by inhibition of prostaglandin synthesis. It can produce severe liver damage due to the accumulation of a highly toxic intermediate metabolite: *nacetylepbenzoquinone* (NAPQ) in overdosage. Normally in therapeutic dosage, this metabolite is detoxified by *glutathione*.

Some Preparations Sold in India

Alkasol-P, Beserol, Calpol, Cosavil, Crocin, Cyclopam, Metacin, Noragesic, Robinaxol, etc

Signs and Symptoms

Initially within first 24 hours it can produce — anorexia, nausea, vomiting, epigastric pain. This will be followed by disappearance of all discomforts giving a false sense of relief in the next 24 hours (i.e. total 48 hours). After 48 to 96 hours it can result in progressive hepatic encephalopathy noticed by vomiting, jaundice, hepatic pain, confusion, coma, and coarse flapping tremors of hands (asterixis) gastrointestinal haemorrhage, cerebral oedema, renal tubular necrosis, etc. There may be cardiac arrhythmias, haemorrhagic pancreatitis, disseminated intravascular coagulation, etc; death often takes place in this stage. In cases where this does not happen patient goes into next stage of *recovery*, which begins in about 5-7 days and the patient gradually becomes completely normal in about 2–3 months time.[1-3]

Fatal dose 10–25 gm.

Toxicity Rating 4

Fatal period Up to five days.

Treatment

- Gastric lavage.
- Oral methionine 10 gm in 12 hours over 4 doses or IV cysteamine prevents hepatic damage if given within 10 hours.
- Cysteamine 1 gm IV in 10 minutes and 400 mg in 5 per cent dextrose over, 4 and 8 hours.
- Vitamin K, charcoal haemoperfusion, hypertonic glucose IV for cerebral oedema.
- *N-acetyl cysteine (NAC)* is specific antidote of choice. Orally given at 1330 mg/kg weight in 3 days, while give IV at 300 mg/kg over 20 hours.
- General/ supportive measures such as IV electrolytes and rehydration, vitamin K for bleeding tendencies, mannitol for cerebral oedema, etc.

Postmortem Findings

- Jaundice and petechial haemorrhages in the skin are evident.
- Congestion of gastrointestinal tract, centrilobular hepatic necrosis, acute tubular necrosis of kidney and cerebral oedema are other internal findings.

Medicolegal Importance

- Used as an antipyretic and analgesics.
- Usually poisoning incidences are of accidental. However, suicidal tendencies are also noticed with paracetamol.

INSULIN

This therapeutic substance is one of the few that has been used repeatedly for homicide and is not infrequently used for suicide, usually amongst nurses and doctors who have access to large doses.

Unless suspected, it is an effective murder method as even though modern methods of postmortem assay now exist, such complex investigations are unlikely to be launched unless there is some suspicion attached to what usually looks like a natural death. Insulin is, of course, a potent hypoglycaemic agent and if severe lowering of the blood sugar persists for many hours, then brain damage and death will occur. In massive doses, especially intravenously, death can take place within few hours. If death from insulin is suspected, either from suicide, homicide or, rarely, from accidental overdose (usually in hospital,) then a search of the body must be made for recent needle marks and the surrounding skin, subcutaneous tissue and underlying muscle excised and sent unfixed for assay. Blood samples should also be taken, as modern analytical methods can now distinguish between human, bovine and porcine insulin and detect adjuvant such as zinc, which assists in tracing the origin of the extrinsic insulin. Postmortem samples should be taken as soon as possible after death and the plasma immediately separated from the cells, and kept deepfrozen until analysis. Postmortem blood glucose levels are generally unhelpful in confirming hypoglycaemia, but vitreous humour may be more useful.

Oral hypoglycaemic agents, such as the sulphonylureas and biguanides, may be taken in overdose, either suicidally or accidentally, producing hypoglycaemia, hypokalaemia and acidosis.[4,5]

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Chapter 39

Food Poisoning and Poisonous Foods

Food poisoning is a vague term. It includes illnesses resulting from ingestion of all foods containing nonbacterial or bacterial products.

The nonbacterial products include poisons delivered from plants and animals, and inorganic chemical. Foods containing such products are, by convention, known as poisonous foods. The bacterial products include bacteria and their toxins. The poisoning resulting there from is, by convention, known as bacterial food poisoning.[1-3]

The illness is characterized by: (i) simultaneous attack of many persons at the same time, (ii) history of ingestion of common food by all sufferers, and (iii) similarity of signs and symptoms in a majority of cases.

BACTERIAL FOOD POISONING

Bacterial food poisoning is of three types: (i) infectious type, (ii) toxic type, and (iii) botulism. *Ptomaine* poisoning due to advanced decomposition of food is not common. Ptomines are proteolytic degradation products formed in decomposing carcasses.

Infectious Type

This type of food poisoning results from ingestion of viable microorganisms that multiply in the GIT producing a true infection, for example, *Salmonella* and *Shigella* group of organisms, *etc*.

Toxic Type

This type of food poisoning results from poisonous substances produced by multiplying organisms that has gained access to the prepared food, for example, enterotoxin produced by the *Staphylococcus*.

Botulism Type

This type of food poisoning results from the ingestion of preformed Botulinum toxin in the preserved food. The toxin is produced by *Clostridium botulinum*.

INFECTIOUS TYPE

In this type of food poisoning, the organisms multiply in the gut and cause gastroenteritis. The common organisms responsible for the attack are the *Salmonella* group of organisms, viz, *S. typhimurium* (Aertrycke), *S. enteritidis* (Gaertner), *S. suipestifer* (cholera suis), *S. Thompson,* and *S. Newport*. Occasionally, the *Shigella* group of organisms, viz, the *S. sonnei* and the *S. flexneri* may be responsible.

The natural reservoir of *Salmonella* organisms is in certain birds, mammals and reptiles. Food may be contaminated with infected excreta of mice or rats, or infection may be transferred by flies or by human carriers employed in the handling of food. *Shigella* infection is the result of contamination of food or water supplies with the feces of the individuals who either have the disease or, less often, are asymptomatic carriers of the organism.

The types of foods which are particularly likely to be injected are twice cooked meat dishes, fish dishes, soups, custards, milk, cream, ice-cream and tinned foods which, though initially sterile, may become infected if not immediately consumed after the tin has been opened. Occasionally, but not usually, there may be visible change in the character of food. The outlook of *Salmonella* food poisoning is likely to occur whenever large amounts of food are prepared and the unconsumed food is kept for future meals. Accordingly, such food poisoning is reported far more frequently from canteens, restaurants, hospitals and other institutions than from private houses.[3,4]

Signs and Symptoms

There is great variation in the susceptibility of individuals to *Salmonella* food poisoning. Hence, while some participants may remain free from symptoms, other may be severely affected.

The condition is not merely a toxaemia but also a gastroenteritis resulting from bacterial infection. The incubation period is longer than the *staphylococcal* food poisoning. The organisms multiply in the intestine and a delay of 12 hours or more is usual before symptoms occur. The onset is sudden and sometimes a chill may be the initial symptom, followed by headache, nausea and vomiting, severe abdominal cramps, and marked prostration.

Three characteristics that help to differentiate this from poisoning with *staphylococcal* enterotoxin are:[5]

- Muscular weakness,
- Fever, and
- Very foul smelling persistent diarrhoea.

The diagnosis rests on the isolation of the causative organism from the patient and suspected articles of food.

Treatment

Stomach should be washed by gastric lavage and the bowel emptied by a cathartic if diarrhoea is not present. Most patients recover rapidly with bed rest and warmth. No food is allowed until the acute symptoms are over. The rest of the treatment is symptomatic. The antibiotic of choice is chloramphenicol, up to 2 gm daily for an adult, for not more than 7 days.

Postmortem Appearances

These are those of gastroenteritis and general toxaemia. The mucosa of the stomach and small intestine show varying degree of inflammation and even ulceration. In severe cases, the lesions may extend to the large intestine. The liver, spleen, kidneys and lungs are congested.

MUSHROOMS

Mushrooms are fungi with umbrella-shaped tops and stems, *Stropheria semeglobata, Hypholoma fasciculare*, and *Lactarius vellereus* are among the poisonous varieties of mushrooms found in India.[1,6]

Action

- Certain mushrooms act by parasympathomimetic action or may be due to hypersensitivity.

Signs and Symptoms

Nausea, vomiting, diarrhoea, bloody vomitus and stools, enlarged tender liver and jaundice, oliguria, pulmonary oedema, mental confusion, convulsions, coma.

Fatal dose ½ to 1 mushroom.

Fatal period 3 to 6 days.

Treatment

Gastric lavage, supportive treatment, atropine, exchange transfusion in children, charcoal, haemoperfusion in adults.

Medicolegal Importance

Usually instances of accidental poisoning are reported.

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Chapter

40

Drug Dependence and Drug Abuse

Hallucinogens, stimulants and *organic solvents* are some of the important drugs of dependence and abuse, which are dealt in detail under this section. Table 40.1 describes other such substances/drugs, which are already discussed in the Chapter 34: *Neurotoxics*.

HALLUCINOGENS (PSYCHEDELICS)

The term hallucinogen is an inaccurate label generally applied to certain group of drugs that produce visual illusions, sensory perceptual distortions, synesthesias, depersonalisation, and derealisation. This class of drugs is perhaps more appropriately termed illusionogenic, psychedelics, or mysticomimetic. These are drugs of both historical interest (used by Indian tribes) and immediate clinical concern (abused by many youngsters).[1]

The classic hallucinogens including LSD (lysergic acid diethylamide), morning glory seeds, the psychotomimetic amphetamine, psilocybin, DT (dimethyl tryptamine), mescaline, peyote, and nutmeg are some of the hallucinogens of medicolegal importance needing further details.

Numerous other hallucinogens, of both natural and synthetic origin, are infrequently encountered. Clinicians should discard their assumption that all hallucinations result from either drug abuse or psychiatric disorders as vast arrays of standard therapeutic agents have hallucinogenic potential in usual or moderate excessive doses and it include drugs like - *amantadine, anticholinergic drugs, carbamazepine, cephalexin, chlordiazepoxide, chloroquine, clonidine, dextramethorphan, dapsone, digoxin, diphenhydramine, disulfiram, ephedrine, griseofulvin, indomethacin, isoniazid, levodopa, lorazepam, methyldopa, methylprednisolone, minocycline, morphine, nalidixic acid, pentazocine, phenothiazines, piperazine, procainamide, procaine penicillin G, propoxyphene, propranolol, streptokinase, tricyclic antidepressants*, etc.[2]

LSD

Synonym: Lysergic Acid Diethylamide LSD-25, d-LSD

LSD is the most potent and widely abused hallucinogen. It was first prepared by Stoll and Hofmann in 1938 (at Sandoz Laboratories in Switzerland), its discovery was an outgrowth of their search for pharmacologically active derivative of ergot. *Ergot* is a biologic product of fungus *Claviceps purpurea*, a parasite of cereal grain (especially rye type) (refer Chapter 33: *Irritant Poisons*).

- Some of the common popular street names for LSD are – *acid, blotter acid, blue caps, blue dots, brown caps, crackers, Deeda, green caps, orange wedges, Paisley caps, pink dots, pink wedges, purple Owsleys, purple wedges, The Beast, The Chief, The Ghost, The Hawk, white lightning, window panes, yellow caps, yellow dots, 25*, etc.[3]
- It is synthesized from rye ergot. It is tasteless, odourless and most potent hallucinogen in minute doses. These can be supplied illicitly on sugar cubes, though it is available in the form of pills of varying colours, sizes and shapes and also in ampoules.

Table 40.1: Substances of dependence and abuse

- Alcohol
- Opium and its derivatives
- Barbiturates
- Chloral hydrate
- Cannabis
- Cocaine
- Caffeine
- Nicotine

Mescaline

- Mescaline is also derived from peyotol plants
- It is in the form of a crystalline powder
- It can be dissolved in water or taken in capsules
- It is not as potent as LSD, acts same as peyote.

Peyote

- It is obtained from a variety of cactus (peyotol) plants
- The toxic principle is present in its button shaped growth of the plant. They are just rolled into balls and kept in capsules. Rarely it may be placed in hot tea and served
- It is not as potent as LSD.

Action of Hallucinogens in General[4-5]

Basically, all hallucinogens are potentially hazardous to human psychology, resulting in disorders of the mind such as:

- Anxiety
- Panic
- Depressive and paranoid reaction
- Mood changes and confusion
- Inability to distinguish between reality and fantasy
- Impairment of normal motivation of life such as to study, work or otherwise contributions to societies.

Signs and Symptoms

- Unusual bizarre behaviour, hilarity, emotional swings and suspiciousness
- The patient may complain of nausea and vomiting (especially with peyote)
- On examination, there may be dilated pupils and tremors
- **Bad trips** — It is defined as the adverse effects experienced by a person on consuming LSD.

LSD mainly acts by interfering with filtering mechanism of the mind. The victim's sense of perception alters uniquely resulting into effects such as:

- He or she will see the colour and hear noises
- There will be total disturbance of sense of time, space and distance
- He or she will get into a dream like state with loss of awareness of body boundaries
- He or she will be experiencing fantasies and hallucinations of varied nature and might present with a flight of ambivalent emotions such as depression and elation, happiness and sadness, etc. simultaneously.

Complications of Bad Trip

Though these experiences cease after sometime, it might give a "flash back" of all the events of dreamy state for several months even up to 2 years or so requiring a long-term therapy for total cure.

- Hangover or after effect

Though after effects with any hallucinogen is rare, following may be complained of by a patient occasionally.

- Insomnia
- Headache
- Vertigo
- Psychotic reactions.

Effects of Prolonged Consumption

Taking hallucinogens for long time can lead to:

i. Permanent damage of brain cells, and
ii. Chromosomal damage in the peripheral blood smears (especially with LSD).

Fatal Dose

As the dose required for desirous effects of any of the hallucinogens is minimum, lethal dose consumption is rare and thus death is exceptionally rare.

Treatment

- Prolonged treatment is essential as 'flash back' effects lasts for long duration
- Psychotherapy is helpful
- Use of tranquilizers can be the choice of treatment helping to minimize the "flash back" effects.

Medicolegal Importance of Hallucinogens[5]

- Hallucinogens are though habit forming, addiction is rare
- They can pose hazards on psychosocial realm triggering psychotic or depressive reaction with "*flash back*" experience (especially with LSD) for long period
- Suicide attempts to commit suicide or homicide after consuming hallucinogens have been reported with drug misadventure case
- Taking hallucinogens may impair one's capacity to drive a motor vehicle or operate machinery.

STIMULANTS

Amphetamines (CNS stimulant, hallucinogen).

Signs and Symptoms

Flushed face, sweating, excitement, restlessness, insomnia, tremors, ventricular tachycardia, hypertension, delirium, hallucinations, convulsions, and deep unconsciousness. Toxic psychosis in chronic poisoning.

Fatal dose 120 to 200 mg.

Fatal period Up to five days.

Treatment

Gastric lavage, sedation with chlorpromazine, cardiorespiratory resuscitation, and general measures. Haloperidol 95-10 mg IV slowly to combat CNS effects.

Medicolegal Importance

Accidental from overdosage, addiction problems, appetite suppression, mood elevation and treatment of narcolepsy.

ORGANIC SOLVENT

A wide variety of organic solvents, which are volatile substances such as:

- *Toluene (glue sniffing)*
- *Gasoline (petrol)*
- *Xylene, benzene*
- *Methylene*
- *Ethylene chloride*
- *Fluorocarbons*
- *Carbon tetrachloride*
- *Butane*
- *Propane*, etc.

These are used for deliberate inhalation for their psychotropic and hallucinogenic effect. The usual way in which these are used is, by placing some of the solvent in a plastic bag and holding the open end over the nose and mouth. Alternatively, it is soaked in a handkerchief or rag, giving rise to the terminology '*solvent abuse*.'

Death in these abusers may be due to vagal inhibition by the gases, which may be sprayed directly into the mouth or due to the sensitisation of the myocardium, by noradrenaline, leading to ventricular fibrillation and arrest. Asphyxia and direct toxic effect of the substance on brain/heart tissue are alternative mechanisms.

Carbon tetrachloride one of the potential examples for '*solvent abuse*' is discussed below in further details:

Action

- Hepatotoxic
- Nephrotoxic.

Signs and Symptoms

When inhaled, can result in irritation of eyes and throat, headache, nausea, vomiting, mental confusion, loss of consciousness, arrhythmia, slow respirations, convulsions, etc. When ingested, can cause dizziness, headache, nausea, vomiting, colic, tremors, convulsions, coma.

Fatal dose 2 to 4 ml (adults), 1 ml (children).

Fatal period 1 to 2 days.

Treatment

In case of inhalation, remove patient from source, administer oxygen, artificial respiration, gastric lavage, saline purgative, and treatment for hepatic and renal damage. N-acetylcysteine is administered in severe cases.

Medicolegal Importance

- Accidental poisoning
- Solvent abuse
- Over dosage.

DRUG DEPENDENCE

World Health Organisation Expert Committee on Addiction Producing Drugs has coined the terminology "drug dependence" newly in place of two older terminologies which had existed till then, namely: (i) drug addiction, and (ii) drug habituation.[4-6]

The reason for this merging being the line of division between them is very difficult to demarcate practically.

However, this newer terminology though has been well accepted all over the world, the older terminology, especially "drug addiction" is still in use and is virtually used to label a person as "drug addict" who is addicted to some kind of addiction forming drug.

This has lead to the need of defining each of these terminologies (Table 40.2) separately.

Drug Dependence

Drug dependence is defined as a psychic and physical state of the person characterized by behavioural and other responses resulting in a compulsion to take a drug, on a continuous or periodic basis in order to experience its psychic effect and at times to avoid the discomfort of its absence.

Drug Addiction

Drug addiction is defined as a state of periodic or chronic intoxication harmful to the individual and to society resulting from repeated consumption of a drug such as opium and its derivatives, pethidine, cannabis, heroin, alcohol, barbiturates, cocaine, LSD, amphetamine, chloral hydrates, etc.

Drug Habituation

Drug habituation is defined as a condition resulting from repeated consumption of a drug, which produces a psychological or emotional dependency on the drug such as caffeine, nicotine, etc.

Drug Abuse

Improper use of a therapeutic or nontherapeutic drug, which may or may not be harmful, even in absence of addiction constitutes drug abuse.

Aetiology of Drug Dependence

Following factors are considered behind the aetiology of drug dependence:

- Common in adolescents and adults
- More common among persons with a tendency of:
  - Taking risks
  - Rebelliousness
  - Truancy
  - Sexual promiscuity
- The drug may be taken for the following effect:
  - Euphoria
  - Improvements in capacity to understand and creativity
  - Better relaxation
  - Improvements in capacity to overcome stress and strains of life
  - Enhanced sexual capacities
  - Experience of sexual pleasure without having actual sexual relation
  - Improve power of meditation (religious).
- Common among people with psychological disorders such as:
  - Psychoneurosis
  - Psychopathic state
  - Frank psychoses.

Unduly prolonged administration of drugs for therapeutic purposes results in medical addicts, e.g. analgesics, sedatives, etc.

Consequences of Drug Dependence

Consequences are several, but following may be considered as important:

- Daily intake of drugs requires money to buy the same from any source making
  - Males indulge in thefts, forgery, etc. for money
  - Female may take up prostitution as easiest way to get money.
- Food, personal hygiene, clothing are often neglected and an addict may appear—deshelved, unkept, unshaven, dirty, etc.

Mechanism of Drug Addiction

Mechanism of drug addiction is rather obscure. It is represented diagrammatically in Figure 40.1.

Signs and Symptoms of Drug Addiction

Signs and symptoms of drug addiction include the following.

- Irresistible desire to continue to take the drug
- Development of tolerance
- Thus a tendency to increase the dose
- Physical dependence on drug
- Desire to obtain drug by any means (even using criminal ways)
- Withdrawal symptoms when the drug is stopped.

Withdrawal Symptoms (Abstinence Syndrome)

The withdrawal symptoms are self-explanatory. They develop in 6 to 48 hours of withdrawal of drugs to which an individual has become an addict, and are characterized by:

- Restlessness
- A feeling of anxiety

Table 40.2: Differentiating characteristics of drug addiction and drug habituation*

Characteristics	*Drug addiction*	*Drug habituation*
1. Compulsion	Present	Only desire, no compulsion
2. Dose	Tendency to increase	Not so
3. Dependence	Both psychological and physical	Only mild psychological
4. Withdrawal symptoms	Characteristic	None/mild
5. Harm	To both victim and society	If any to victim only

*Though these differences are shown in tabular columns, it is very difficult to recognize when the habit formation can get transformed into addiction, making both entities one and the same, i.e. drug dependence, as stated by WHO

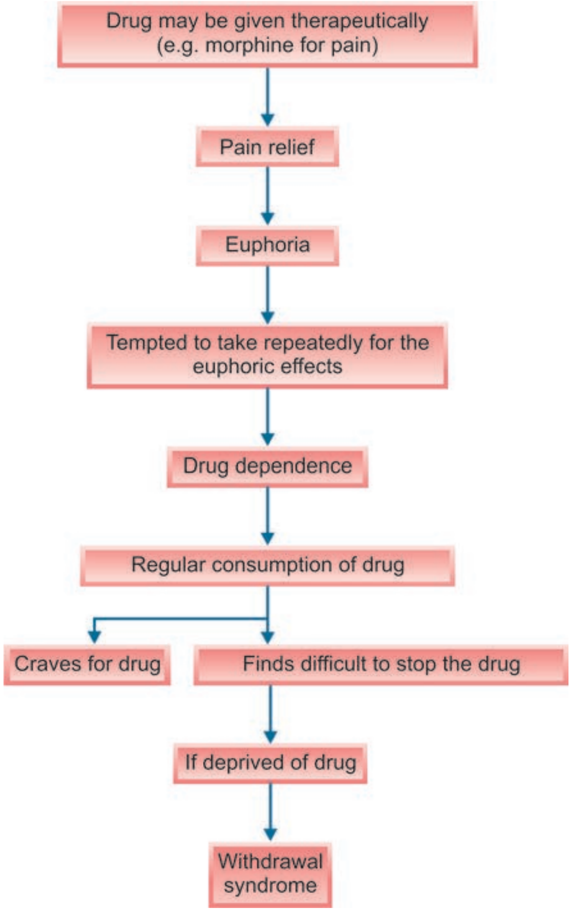
Drug may be given therapeutically (e.g. morphine for pain)
Pain relief
Euphoria
Tempted to take repeatedly for the euphoric effects
Drug dependence
Regular consumption of drug
Craves for drug
Finds difficult to stop the drug
If deprived of drug
Withdrawal syndrome

Fig. 40.1: Diagrammatic representation of mechanism of drug addiction

- Vague pain in abdomen and limbs
- Diarrhoea
- Increased libido.

These symptoms last for variable period, depending on the drug used, dose consumed, duration of addiction and suddenness of withdrawal.

Treatment of Addiction

Following measures are effective:

- Institutional treatment is recommended
- Secret watch for preventing further supply of drugs
- Gradual withdrawal of drug in stages by progressive tapering of dose
- Administration of small doses of sedatives, e.g. barbiturates
- Keeping the victim engaged with physical and mental activities
- Psychotherapy in the form of encouragement
- Improving general health by consuming good and rich food
- Symptomatic measures.

Mechanism of Drug Habituation

Like drug addiction, the measures of drug habituation are obscure. However, following facts are true:

- It is common in people with an imitative curiosity.
- Communicable from one person to other.

Signs and Symptoms of Drug Habituation

Person habituated to drug is called a drug habituate, and presents with following:

1. A desire, but not irresistible to continue to take the drugs.
2. Little or no tolerance.
3. Hence, little or no tendency to increase the dose.
4. Some degree of psychic, but no physical dependence.
5. A detrimental effect only on the person if any, but not on society
6. Absence of withdrawal symptoms.

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Appendices

APPENDIX 1: QUESTION BANK

FORENSIC MEDICINE VIVA VOCE QUESTIONS

MEDICAL JURISPRUDENCE

The Doctor and the Law

1. Mention the types of courts in India?
2. Mention the types of magistrate courts?
3. What are the powers of First Class Magistrate?
4. What are the powers of Session's court?
5. What are the differences between high court and session's court?
6. What is the power of Session's judge?
7. What is the power of coroner's court?
8. What are the different punishments laid down by law?
9. What is summons?
10. What is oath?
11. Is oath compulsory for giving evidence?
12. What is perjury?
13. What are the different types of witnesses?
14. Give example for common witness
15. Give example for expert witness
16. Who is a skilled witness?
17. Who is a hostile witness?
18. Who declares a witness to be hostile? Is it credit or discredit?
19. Enumerate the procedures of giving evidence in the court of law?
20. If you do not take oath will it be an offence?
21. What is a cognizable offence?
22. What is a non-bailable warrant (NBW)?
23. What do you mean by contempt of court?
24. How coroner's court is different from magistrate's court?
25. What do you mean by inquest?
26. Is coroner's inquest held in India at present?
27. What are the different types of inquest?
28. Enumerate the steps of police inquest?
29. Why coroner's inquest is better than police inquest?
30. Enumerate the steps of magistrate's inquest?
31. What are the indications for magistrate's inquest?
32. What are the powers of a second-class magistrate?
33. Who is a public prosecutor?
34. What is examination-in-chief?
35. What is cross-examination?
36. Who does the examination-in-chief?
37. Who does the cross-examination?
38. Among examination-in-chief and cross-examination, which is more important?
39. What do you mean by a leading question?
40. Mention the types of evidence?
41. Give examples for documental evidence?
42. Give examples for oral evidence?
43. Between documentary and oral evidence which is more important? Why?
44. What is dying declaration?
45. What is dying deposition?
46. Between dying declaration and dying deposition, which is more important?

Ethics of Medical Practice

47. What do you mean by medical ethics?
48. Enumerate the duties of a doctor in general?
49. Enumerate the duties of a doctor to the sick?
50. What is IMC/MCI?
51. What is SMC?
52. Where is the office of MCI situated?
53. Where is the office of SMC situated?
54. Enumerate the functions of State Medical Council?
55. Enumerate the functions of Indian Medical Council?
56. What is a warning notice?
57. Give one example for infamous conduct?
58. Is registration with SMC compulsory before you go for professional practice?
59. Enumerate the formalities of being registered as registered medical practitioner (RMP)?
60. Mention the privileges of being a registered medical practitioner?
61. Define infamous conduct?
62. Define professional malpraxis?
63. What is contributory-negligence?
64. What do you mean by res-ipsa-loquitur?
65. What do you mean by vicarious responsibility?
66. What do mean by res judicata?
67. What is medical indemnity insurance? What is its legal value?
68. What do you mean by consent?
69. What is informed consent?
70. What is implied consent?
71. What is loco-parentis?
72. What is blanket consent?
73. What do you mean by therapeutic privilege?
74. What is the minimum age to give consent for major surgery?
75. What is the minimum age to give consent for donating the body organs?
76. What is the minimum age for giving consent for MTP?
77. What is section 88, IPC?
78. What is the minimum age to give consent for physical examination by a physician?
79. What is section 89, IPC?

80. What is the minimum age to give consent for sexual intercourse?
81. What is statutory rape?
82. Give examples of infamous conduct?
83. What is association?
84. What is adultery?
85. What is fees splitting?
86. What is sharing?
87. What is dichotomy?
88. What is covering?
89. Give an example for direct advertising?
90. Give an example for indirect advertising?
91. What is ghost surgery?
92. What do you mean by disciplinary action by state medical council?
93. What is suspension?
94. What is penal erasure?
95. What do you mean by professional death sentence?
96. What are professional secrets?
97. What is the doctrine of privileged communication?
98. What is the doctrine of emergency?
99. What is Section 351, IPC?
100. What is Section 90, IPC?
101. What is Section 92, IPC?
102. Enumerate the indications for erasing the names of a RMP from State Medical Register?
103. What is an appeal?
104. What is warning?
105. What is warning notice?
106. What is damage?
107. What are damages?
108. What is medical etiquette?
109. What is medical mal-occurrence?
110. What is meant by intervention by third party?
111. What do you mean by contributory negligence?
112. What is medical mal-occurrence?
113. What is Act of God?
114. What is therapeutic misadventure?
115. What is error judgment?
116. What is vicarious liability?
117. Doctrine of respondent superior?
118. What is doctrine of borrowed servant?
119. What is product liability?
120. What is corporate negligence?
121. Enumerate the differences between infamous conduct and professional negligence?
122. What is euthanasia?
123. What is exhumation?
124. What is mercy killing?
125. What is Indian Factories Act?
126. What is Consumer Protection Act (COPRA)?
127. What are the purpose/principles of COPRA?
128. What are paired organs?
129. What are the rules of donating organs?
130. What is cadaver donation?
131. What are the principles of Organ Transplantation Act?
132. What is an eye bank? What is an organ bank?

FORENSIC PATHOLOGY

Thanatology

133. What is thanatology?
134. Define death?
135. What are the different types of death?
136. What are the modes of death?
137. What is manner of death?
138. What is somatic death?
139. What is molecular death?
140. What is brainstem death?
141. What are Harvard's criteria of death?
142. What is postmortem lividity?
143. What is postmortem suggilation?
144. What do you mean by fixation of postmortem lividity?
145. How will you test the fixation of postmortem lividity?
146. How does postmortem lividity differ from an antemortem bruise?
147. Mention the early changes of death in the eyes?
148. What is tachi-noire?
149. Enumerate the early changes in the retina of the eye?
150. Mention the immediate changes of death?
151. What is rigor mortis?
152. What is algor mortis?
153. What is postmortem cooling?
154. What is postmortem caloricity?
155. What is postmortem hypostasis?
156. What is instantaneous rigor?
157. What is cadaveric spasm?
158. What is putrefaction?
159. Mention the late putrefactive changes?
160. Mention the late changes of death?
161. What is mummification?
162. What is adipocere?
163. In examination of bones what are the points to be kept in mind?
164. How time since death can be estimated?
165. What is the difference between the second autopsy and exhumation?
166. What do you mean by death?
167. What is the differential diagnosis of comatose condition?
168. What is the surest sign of death?
169. What do you mean by postmortem caloricity?
170. How opthalmoscopic examination can help in determination of time since death?
171. What are the different methods of heat loss?
172. What do you mean by livor mortis?
173. How congestion can be differentiated from hypostasis?
174. What do you mean by cadaveric rigidity?
175. What is the medicolegal importance of cadaveric spasm?
176. What do you mean by putrefaction?
177. What is your idea about foamy liver?
178. What is adipocere formation?
179. What is mummification?
180. What is embalming?
181. How will you preserve the dead body for anatomical study?
182. How will you preserve the dead body to be dispatched to a far away place?
183. How biochemical analysis helps in determination of time since death?
184. What information you get from the study of bone marrow?
185. What do you mean by presumption of death?
186. What is the role of medical officer in presumption of survivorship?

Postmortem Examination

187. What is the purpose of doing medicolegal autopsy?
188. How medicolegal autopsy is different from pathological autopsy?

189. What are the different types of postmortem skin incisions used in medicolegal autopsy?
190. Mention how the scalp is incised?
191. What is an 'I' shaped incision?
192. What is a 'Y' shaped incision?
193. What is a modified 'Y' shaped incision?
194. Who can authorise you for a medicolegal autopsy in a homicidal case?
195. Who can authorise you for a medicolegal autopsy in a routine case of RTA?
196. What should be the minimum qualification of a doctor to perform a medicolegal autopsy?
197. In a case of death due to hanging which body cavity is better to be opened first?
198. In a case of death due to head injury which body cavity is better to be opened first?
199. What do you mean by obscure autopsy?
200. What is an artifact?
201. What is an en masse removal of viscera?
202. Name the autopsy instruments?
203. How will you remove the heart during medicolegal autopsy?
204. How the heart is dissected after its removal during a medicolegal autopsy?
205. How is brain removed during autopsy examination?
206. How will you demonstrate rigor mortis during autopsy?
207. How will you demonstrate fixing of postmortem lividity during autopsy?
208. What is bloodless dissection of neck?
209. How do you dissect the stomach after removal from the body?
210. What do you mean by collapsed lung?

Violent Asphyxial Death

211. What is the difference between hanging and strangulation?
212. Why hanging is chosen as judicial punishment in India?
213. What are the causes of death in hanging?
214. What is the difference between typical and atypical hanging?
215. What is the difference between partial hanging and complete hanging?
216. How can you say definitely that the case is an antemortem hanging?
217. What is petechial haemorrhage?
218. What are the different methods of strangulation?
219. What characteristic finding you will search for in case of throttling?
220. What information you can deduce from a hyoid bone examination?
221. What are the different methods of suffocation?
222. What is cafe coronary?
223. What is lynching?
224. What are the findings of homicidal hanging?
225. Give some examples of accidental hanging?
226. What is autoerotic hanging?
227. What is sexual asphyxia?
228. Will the ligature mark putrefy?
229. What is strangulation?
230. What are the different types of strangulations?
231. What is manual strangulation?
232. What is throttling?
233. What is mugging?
234. What is bhansdola?
235. What is garroting?
236. Describe the ligature mark of strangulation?
237. What is suffocation?
238. Define smothering?
239. What is burking?
240. What is choking?
241. What is traumatic asphyxia?
242. Define drowning?
243. Classify types of drowning?
244. What is typical drowning?
245. What is wet drowning?
246. What is atypical drowning?
247. What is dry drowning?
248. What is immersion syndrome?
249. What is submersion of the unconscious?
250. What is secondary drowning?
251. What is near drowning?
252. What makes the dead body float in water?
253. What is the pathophysiology of salt water drowning?
254. What is the pathophysiology of fresh water drowning?
255. What are the external findings of drowning in the face?
256. What are the external findings of drowning in the skin?
257. What is cutis anserina?
258. What are the external findings of drowning in the hands?
259. What are the external findings of drowning in the feet?
260. How froth is formed in a drowning victim?
261. Classify the different types of drowning.
262. What are the causes of death in drowning?
263. What findings can definitely say that it is a case of antemortem drowning?
264. What are the middle ear findings in ante-mortem drowning?
265. What is Getler's test?
266. What are the indications for Getler's test?
267. What are diatoms, and what is a diatom test?

Forensic Identity, Trace Evidence, Forensic DNA Profiling

268. Define identity?
269. What is corpus delicti?
270. Enumerate the factors helpful in establishing human identity?
271. How race can be determined by examination of skull?
272. Enumerate the factors helpful in establishing age of a person?
273. What is forensic dentistry?
274. What is forensic odontology?
275. What are the basic types of teeth?
276. Mention the different parts of a tooth?
277. What are the total number of teeth present in a ten-year-old child?
278. Mention the exact number of temporary and permanent teeth?
279. What do you mean by spacing of jaw? How do you measure it?
280. What is the importance of spacing in the jaw?
281. What is the Gustafson's method of examination of teeth in determining the age of a person?
282. What is the total number of skeletal bones present in an adult individual?

283. Which is the first ossification centre that appears in intrauterine life?
284. Which are the ossification centers looked for in determination of the gestational age?
285. What is the average length of a full term foetus?
286. What is the length of the umbilical cord in a full term foetus?
287. What is the circumference of the head in a newborn?
288. What do you mean by Lanugo hairs?
289. When do the nails appear in intrauterine life?
290. When does the placenta form in intrauterine life?
291. What are the various constituents of the umbilical cord?
292. What is the medicolegal importance of Wharton's Jelly?
293. In case of precipitated labour which side of the umbilical cord tears?
294. Which is the midpoint of adult human body?
295. How symphyseal face of the pubic bone can help in determination of age?
296. Name the carpal bones which ossify in the first year of age?
297. What is the medicolegal importance of age?
298. What is the age of criminal responsibility?
299. What is the age of consent for medical examination?
300. What is the age of marriage for girls in India?
301. What is the minimum age at which a girl can give consent for sexual intercourse in India?
302. Mention the age at which the pubic hairs make their first appearance in girls?
303. Mention the age at which the pubic hairs make their first appearance in boys?
304. When do the pubic and axillary hairs turn gray?
305. What is Arcus senilis?
306. What are the different methods of determining the sex?
307. What do you mean by inter-sex?
308. What do you mean by Davidson's body?
309. What is a Barr body?
310. What is sex chromatin?
311. What is the accuracy of establishing sex by skull and pelvis?
312. What is the accuracy of establishing sex by pelvis and femur?
313. What is the accuracy of establishing sex by skull and long bones?
314. What is the accuracy of establishing sex by skull alone?
315. What is the accuracy of establishing sex by pelvis alone?
316. What is the accuracy of establishing sex by long bones only?
317. What is the accuracy of establishing sex by entire skeleton?
318. Who is a juvenile offender?
319. What is a borstal?
320. What are sections 82 and 83 under IPC?
321. What are sections 127-130 under Indian Railway Act?
322. As per Indian factories Act, 1948, up to what age child cannot be employed in a factory?
323. What is the medicolegal importance of sex?
324. How sex can be determined in a highly putrefied dead body?
325. What is concealed sex?
326. Give an example for gonadal agenesis?
327. Give an example for gonadal dysgenesis?
328. What is Turner's syndrome?
329. What is Klinefelter's syndrome?
330. What do you mean by pseudohermaphroditism?
331. What is true hermaphroditism?
332. What is mathematical sexing of bones?
333. What is cephalic index?
334. How to determine cephalic index?
335. What is dolicocephaly?
336. What is mesaticephaly?
337. What is brachycephaly?
338. What is sacral index?
339. What is sternal index?
340. What is ischiopubic index?
341. What is corporobasal index?
342. What is bicondylar width?
343. What is femoral angle?
344. What is neck-shaft angle?
345. What is articulation in forensic osteology?
346. What is footprint ratio?
347. What are the recent advances in establishing sex identity?
348. What do you mean by costal cartilage calcification pattern?
349. What do you mean by mandibular canine index?
350. What do you mean by superimposition?
351. What is the best method of identifying the individual?
352. Which is the most accurate method of establishing human identity?
353. What is dactylography?
354. What is dermatoglyphics?
355. What is Galton system?
356. What is a plain fingerprint?
357. What is a rolled fingerprint?
358. Mention the different types of fingerprints?
359. What is a latent fingerprint?
360. What do you mean by poroscopy?
361. What are footprints?
362. What is the difference between walking footprint and a standing footprint?
363. In what type of soil footprints tend to be smaller?
364. Mention some of the deformities that can be appreciated in a footprint?
365. What are palatoprints?
366. What is rugoscopy?
367. What is cheiloscopy?
368. Mention the different type of lip prints?
369. How will you confirm that a given material is hair?
370. What are the different layers of hair in its cross section?
371. How will you confirm given hair is human?
372. What is precipitin test?
373. What is the principle precipitin test?
374. How to confirm the site of hair?
375. How to assess the cause of injury to the hair?
376. How will you establish the cause of stains on hair?
377. What is the significance of different stains on the hairs?
378. Name the poisons deposited in the hairs?
379. What miscellaneous information hairs can give?
380. What do you mean by fraying of hairs?
381. What is a singed hair?
382. Mention the gross changes seen in a singed hair?
383. Mention the microscopic changes seen in a singed hair?
384. Mention the differences between human and animal hairs?
385. What is a lanugo hair?

386. What is the rate of growth of hair?
387. How does scalp hair differ from pubic hair?
388. What is the shape of hair on its cross section from different sites of the body?
389. How hairs can help in establishing the racial identity of man?
390. How hairs can help in establishing the age of a person?
391. What do you mean by male distribution of hairs?
392. What do you mean by female distribution of hairs?
393. Define stature.
394. What do you mean by stature?
395. What do you mean by diurnal variation of stature?
396. Mention the factors that affect stature of a person?
397. How stature helps in human identity?
398. How is the stature of a person determined from dismembered body parts?
399. What is a Hepburn's osteometric board?
400. What is Karl Pearson's formula?
401. What is Trotter and Glaister's formula?
402. Mention the factors affecting multiplication factor (MF) of Karl Pearson's Formula?
403. How to determine multiplication factor for a long bone?
404. What is a scar?
405. Mention the medicolegal importance of scars?
406. What are tattoos?
407. How tattoos are made?
408. What is the colouring pigments used in tattooing?
409. Mention the medicolegal importance of tattoos?
410. What do you mean by tattooing under firearm wounds?
411. How tattoos can be removed?
412. What are deformities?
413. What is the medicolegal significance of deformities?
414. What are the different types of deformities?
415. Mention a congenital deformity helpful in establishing identity?
416. Mention acquired deformity helpful in establishing identity?
417. What do you mean by features?
418. Give some examples of features helpful in establishing human identity?
419. How does complexion help in establishing identity?
420. What are trace evidences?
421. What are the medicolegal significances of trace evidences?
422. What do you mean by Forensic Science Laboratory (FSL)?
423. What are the functions of FSL?
424. What is chain of custody?
425. What is the role of photography in medicolegal practice?
426. What do you mean by Locard's principle of exchange?
427. How will you determine origin or species of a biological stain?
428. Describe a human red blood cell.
429. What is the shape of a human RBC?
430. What is the size of a human RBC?
431. Where will be the antibodies in blood?
432. Is it possible to determine the sex of a bloodstain?
433. How can you say whether the blood is of arterial or venous origin?
434. What expert opinions can be derived from the shape of the bloodstain?
435. How to differentiate whether the bloodstain is of an antemortem origin or postmortem origin?
436. Name some stains which resemble bloodstain?
437. How will you distinguish menstrual blood from nasal bleeding?
438. What are the differences between arterial and venous blood?
439. Describe an animal red blood cell?
440. Mention the conditions in which human RBCs are nucleated?
441. Name the microchemical test that confirms blood?
442. What are the confirmatory tests for blood?
443. Name a screening test for blood?
444. How is the haemin crystal test done in the laboratory?
445. Describe the haemin crystal?
446. Describe the haemochromogen crystal?
447. What major information you can get from bloodstain?
448. What are the solvents used for bloodstain?
449. Name the tests for bloodstain? Which of these are confirmatory tests?
450. What are the principles of micro-chemical test for blood?
451. What is the advantage of spectroscopic examination?
452. What do you mean by group secretors?
453. What are the advantages of blood grouping in medicolegal practice?
454. What will be the appearance of seminal stain on a cloth?
455. What are the other stains that resemble seminal stains?
456. Name some chemical tests for human semen?
457. What are blood group secretor substances?
458. Is it possible to determine the blood group of a person from semen?
459. What are group secretor tests?
460. Presence of saliva and salivary stains suggests what?
461. What is the composition of saliva?
462. Name some tests for detecting human saliva?
463. Can faecal matter be tested for precipitin test?
464. Can group specific tests be done with urinary and faecal stains?
465. What is DNA profiling?
466. What is DNA fingerprinting?
467. How DNA strands can be cut?
468. What are chemical scissors?
469. Is it possible to perform DNA fingerprinting with bloodstain?
470. Is it possible to perform DNA fingerprinting with seminal stain?
471. Is it possible to perform DNA fingerprinting with RBCs?
472. Is it possible to perform DNA fingerprinting from WBCs?
473. What do you mean by PCR?
474. Mention the medicolegal importance of DNA fingerprinting?
475. What is the cost of DNA fingerprinting test in India? Where is it done?

CLINICAL FORENSIC MEDICINE

Trauma

476. Define injury?
477. Define clinical definition of injury?
478. Define hurt?
479. Define legal definition of injury?
480. What is section 44, IPC?
481. What are mechanical injuries?
482. Classify mechanical injuries?
483. Classify the injury in legal point of view?

484. What is an abrasion?
485. Classify abrasions?
486. What is a scratch?
487. What is a graze?
488. What is an imprint abrasion?
489. What is a brush burn?
490. What medicolegal information can be derived from an abrasion?
491. How time since abrasion is derived?
492. How age of an abrasion is derived?
493. What is a postmortem abrasion?
494. What are the differences between antemortem and postmortem abrasions?
495. What is an excoriation?
496. What is a pseudoabrasion?
497. When does an abrasion become grievous injury?
498. How abrasions help in assessing the direction of wounding?
499. How abrasions help in assessing the motive of wounding?
500. What is a contusion?
501. Define contusion?
502. Classify contusions?
503. What are the types of contusions?
504. What is a patterned bruise?
505. What is a deep bruise?
506. Give an example for deep bruising?
507. Mention the factors affecting bruising?
508. How will you assess age of the bruise?
509. How bruising helps in assessing time since injury?
510. Among abrasion and contusion which one is more important medicolegally?
511. What are the different colour changes observed in a bruise?
512. What is the medicolegal information derived from contusions?
513. What is an artificial bruise?
514. What are fabricated wounds?
515. What are the differences between an artificial and a true bruise?
516. How does a bruise differ from postmortem lividity?
517. What is a laceration?
518. Define laceration?
519. Enumerate the causes of laceration?
520. What are the salient features of a laceration?
521. What are tissue bridges?
522. What are tissue tags?
523. Classify types of lacerations?
524. What is a split laceration?
525. What is a stretch laceration?
526. What is an avulsion?
527. What is an incised like laceration? Give examples?
528. Do lacerations produce a permanent scar on healing?
529. Is it possible to produce suicidal lacerations?
530. Is it possible to self-inflict lacerations?
531. What is an incised wound?
532. Define incised wound?
533. Describe an incised wound?
534. Is it possible to assess direction of wounding by incised wound?
535. What is 'tailing' of an incised wound?
536. How will you assess time since injury for an incised wound?
537. What are hesitation cuts?
538. What are defense cuts?
539. What are defense wounds?
540. What is the medicolegal importance of incised wound?
541. What is a stab wound?
542. What will be the shape of stab wound by a dagger?
543. What will be the shape of stab wound by a penknife?
544. What are the Langer's cleavage lines?
545. What is volitional activity in a stab wounding?
546. Classify stab wound?
547. What is a penetrating wound?
548. What is a perforating wound?
549. Length of the stab wound corresponds to which dimension of the weapon used to stab?
550. Depth of the stab wound corresponds to which dimension of the weapon used to stab?
551. What are the different geometrical variations of an incised wound and stab wound?
552. What do you mean by Vendetta murder?
553. What do you mean by concealed punctured wound?
554. How will you measure the depth of penetrating or stab wound in a live patient?
555. While issuing an Injury certificate what precautionary measures you would like to take?
556. What is the medicolegal importance of beveling cut?
557. What is a chop wound?
558. What is a cut throat wound?
559. How will you differentiate between suicidal and homicidal cutthroat?
560. What do you mean by incised looking lacerated wound?
561. When will you say the wound is self-inflicted or fabricated?
562. What is a head injury?
563. How will you classify head injury?
564. What is a closed head injury?
565. What is an open head injury?
566. Mention the scalp injuries?
567. What is the medicolegal significance of scalp injury?
568. What is a diploic bone?
569. What is direct violence in producing skull fracture?
570. What is indirect violence in producing skull fracture?
571. Classify the types of skull fractures?
572. What is a fissured fracture skull?
573. What is a mosaic fracture skull?
574. What is a stellate fracture skull?
575. What is a depressed fracture skull?
576. What is an elevated fracture skull?
577. What is a diastic fracture skull?
578. What is a suture line fracture skull?
579. What is a gutter fracture skull?
580. What is a comminuted fracture skull?
581. What is a perforating fracture skull?
582. What is a combined fracture skull?
583. What is a signature fracture skull?
584. What is a ring fracture skull?
585. What is a transverse fracture skull?
586. What is a hinge fracture skull?
587. What is a pond fracture skull?
588. What is a black eye?
589. What is a spectacle haematoma?
590. What do you mean by 'fracture la signature'?
591. Is it possible to assess time since injury by fracture skull?

592. What are the mechanisms of brain injury?
593. What is an accelerating injury?
594. What is a decelerating injury?
595. What is a shear strain/rotational injury?
596. What is a coup injury?
597. What is a contrecoup injury?
598. What is a cerebral concussion?
599. What is stunning brain shock?
600. What is the role of meninges and CSF in preventing brain injury?
601. What is post-concussion syndrome?
602. What is retrograde amnesia?
603. What are the differences between drunkenness and head injury?
604. What is a cerebral contusion?
605. What are the common sites of cerebral contusion?
606. What is a cerebral laceration?
607. What are the common types of cerebral laceration?
608. What is gliosis?
609. What is cerebral irritation?
610. What is cerebral compression?
611. What is coning?
612. Classify the different intracranial haemorrhages?
613. What are the various causes of extradural haemorrhage (EDH)?
614. What are the causes of subdural haemorrhage (SDH)?
615. How do you confirm subarachnoid haemorrhage (SAH) during autopsy examination?
616. What are coup and contrecoup injuries of the brain?
617. What are the causes of subarachnoid haemorrhage?
618. What are the signs of cerebral oedema?
619. What are the differences between intracranial haemorrhage due to head injury and disease?
620. Can you diagnose clinically the pontine haemorrhage?
621. What is the minimum loss of blood that can cause death?
622. What do you mean by post-traumatic amnesia or automatism?
623. What are the ‘Trigger areas of body’?
624. What is a cardiac tamponade?
625. What are the causes of cardiac exsanguinations?
626. What is surgical emphysema?
627. What is concussion of spinal cord?
628. What is a railway spine?
629. What is whiplash injury?
630. What is a primary impact injury?
631. What is a secondary impact injury?
632. What are secondary injuries?
633. Can an internal injury to abdominal organ be simple, grievous or endangering to life?
634. What are the advantages of using helmet in a road traffic accident?
635. What are the disadvantages of using helmet?
636. In a case of vehicular accident how can you opine that the person was a driver?
637. What are aviation injuries?
638. What is punch drunk?
639. What is seat belt trauma?
640. What are collision and derailment trauma?
641. What are injuries due to hit by speeding train?
642. What do you mean by drowning in ones own blood?
643. What do you mean by cardiac tamponade?
644. What are the sources of fat embolism?
645. What are the sources of air embolism?
646. What is ARDS?
647. What are the differences between antemortem and postmortem blood clots?
648. What is wound biochemistry and its medicolegal significance?
649. What is murder?
650. What is justifiable murder?
651. What are the differences between sections 300, 302, 304, and 304 (A) IPC?
652. What do you mean by abetment of suicide and attempt to commit suicide?
653. What is Section 306, IPC?
654. What is Section 309, IPC?
655. What are assault, battery, hurt and insult?
656. What are the causes of death from wounds?
657. What is a crush syndrome?
658. What are the causes of fat embolisms?
659. What are the causes of air embolism?
660. What is volitional or voluntary activity?
661. What are the causes of hypothermia in newborn infants?

Firearms and Explosive Injuries

662. What do you mean by ballistics?
663. Classify firearms.
664. What does rifling mean?
665. Mention the structure of firearms in general?
666. What are the chemical constituents of igniting powder?
667. What are the chemical constituents of smokeless gunpowder?
668. What are the chemical constituents of black gunpowder?
669. What are rifling marks?
670. Where will be the rifling marks are seen?
671. What is the purpose of rifling?
672. What is the pattern of rifling?
673. How to measure caliber of a rifled firearm?
674. Classify rifled firearms or guns?
675. What are the different parts of rifled cartridges?
676. What is a shotgun?
677. What is a smoothbore firearm?
678. How to measure caliber of a smooth bore firearm?
679. What do you mean by choking of a shotgun?
680. What are the advantages of choking?
681. What are the types of choking?
682. Classify shotguns.
683. What is the difference between a bullet and pellet?
684. What is wound ballistics?
685. What is terminal ballistics?
686. Is the firearm injury simple or grievous?
687. What to do you mean chocking of a smooth bored firearm?
688. What are the different parts of shotgun cartridges?
689. How will you determine the range of firing?
690. What are the constituents of smoke less power?
691. What is the difference between a crime bullet and a test bullet?
692. Is pistol a rifled weapon?
693. What is an automatic pistol?
694. How to differentiate wound of entry and wound of exit by a rifled firearm?
695. How will you locate the position of bullet in a cadaver?
696. What is the role of comparison microscope in a case of firearm injury?

697. What are the different types of bullets in common use?
698. What do you mean by barrel and bore of a gun?
699. What do you mean by blast effect of a gun?
700. What are the findings of a wound of entry in a contact shot range of firing by rifled firearm?
701. What are the findings of a wound of entry in a close shot range firing by rifled firearm?
702. What are the findings of a wound of entry in a near shot range firing by rifled firearm?
703. What are the findings of a wound of entry in a distant shot range firing by rifled firearm?
704. What is an abrasion collar?
705. What is a grease collar?
706. What is a ricochet bullet?
707. What is a tandem bullet?
708. What is a dum-dum bullet?
709. What is a souvenirs bullet?
710. What is a crime bullet?
711. What is an exhibit bullet?
712. What is a test bullet?
713. What is Kennedy phenomenon?
714. What is Rayalseema phenomenon?
715. What is the appearance of wound of entry of a shotgun at a range of <15 cm firing?
716. What is the appearance of wound of entry by a shotgun at a range of 15 cm firing?
717. What is the appearance of wound of entry by a shotgun at a range of 90 cm firing?
718. What is the appearance of wound of entry by a shotgun at a range of 2 m firing?
719. What is the appearance of wound of entry by a shotgun at a range of 4 m firing?
720. What is billiard ball ricochet effect?
721. How will you assess the range of firing from spread area of pellets?
722. How will you explain in court of law having one wound of entry and multiple wounds of exits?
723. What is dermal nitrate test? Are there any fallacies for it?
724. What is neutron activation of analysis (NAA) and how it helps in determination of a firearm?
725. When the responsibility of an autopsy surgeon is over completely in a case of firearm injury?
726. What are the precautions you are likely to take at the time of conducting the postmortem in a firearm injury cases?
727. Can you opine the firearm wound to be homicidal by external examination?
728. What are the injuries likely to get in a bomb blast/explosion?

Effects of Cold and Heat, Electrocution, Lightning and Radiation

729. What is hypothermia?
730. What is frostbite?
731. What is chill bane?
732. What is immersion foot?
733. What is a trench foot?
734. What is hyper-thermia?
735. What are heat cramps?
736. What is heat hyper-pyrexia?
737. What is heat prostration?
738. What is heat exhaustion?
739. What is sunstroke?
740. What is thermic fever?
741. What is a scalding burn?
742. What is erythema?
743. What is vesication?
744. What is necrosis of dermis?
745. What is the medicolegal importance of scalding burns?
746. What are the various sources of flame burn?
747. What is a curling's ulcer?
748. What is a keloid?
749. What is rule of nine?
750. What are the causes of death in burns cases?
751. What is pugilistic attitude?
752. How to differentiate the antemortem and postmortem burns?
753. What is section 304 (B), Indian Penal Code?
754. What is bride burning and the important section under IPC concerned with this?
755. What are the merits of Wilson's classification of burns over Dupuytren's?
756. What are the positive findings suggestive of antemortem burns?
757. In case of burn injury what are the causes of death?
758. What is radiation injury?
759. How acid burn differs from steam burn?
760. How you can diagnose heat rupture?
761. What is a heat haematoma?
762. What do you mean by spontaneous combustion?
763. What do you mean preterm natural combustibility?
764. What are the characteristic findings of electrical burn?
765. What are the histological changes of electrical burns?
766. What is judicial electrocution?
767. What is filigree burn?
768. What is Joule burn?
769. What are the postmortem findings in starvation death?

Sexual Jurisprudence (Virginity, Pregnancy, Delivery, Abortion, Sexual Offences, Infanticide)

770. Who is a virgin?
771. What is hymen?
772. Mention the various types of hymen?
773. Who is a false virgin?
774. Can you prove that the lady is virgin?
775. What do you know about virgo intact?
776. What will be the findings in a true virgin?
777. Define defloration?
778. What are the absolute signs of defloration?
779. Define impotency?
780. What is vaginismus?
781. What are the causes of impotency in male?
782. What are the causes of impotency in female?
783. What are the causes of sterility in male?
784. Can a woman be sterile and what are the reasons?
785. What are the differences between sterility and impotency?
786. Among sterility and impotency, which one is the ground for divorce?
787. What is the medicolegal importance of sterilization?
788. What do you mean by artificial insemination (AI)?
789. What are the legal problems of artificial insemination?
790. What are the complications of artificial insemination?
791. What are the prerequisites for artificial insemination?

792. Define pregnancy?
793. What are the subjective symptoms of pregnancy?
794. What are the objective signs of pregnancy?
795. What are the absolute/conclusive/ sure signs of pregnancy?
796. What is pseudocyesis?
797. What is the normal duration of pregnancy?
798. What is the period of viability of a child?
799. What are the signs of recent delivery in a living?
800. What are the signs of recent delivery in the dead?
801. What is lochia?
802. What are the types of lochia?
803. What are the signs of remote delivery in the dead?
804. Define paternity.
805. What are paternity tests?
806. What is atavism?
807. What is super-foetation and super-fecundation?
808. Define legitimacy.
809. Who is a suppositious child?
810. What are affiliation cases?
811. What are natural sexual offences?
812. What are unnatural sexual offences?
813. What are sexual deviations/ sexual perversions?
814. Define rape?
815. What is section 375 IPC?
816. What is section 376 IPC?
817. What is the punishment for rape?
818. What is the minimum age for a female to give consent for sexual intercourse in India?
819. What are the dangers of rape?
820. What are the findings of rape in a child?
821. What are the findings of rape in a virgin girl?
822. What are the findings of rape in a deflorated woman?
823. What are the precautionary measures to be taken prior to the examination of a rape victim?
824. What are the precautionary measures to be taken at the time of examination of a rape victim?
825. What are the precautionary measures to be taken after the examination of a rape victim?
826. What are the findings in a victim suggestive of forcible sexual intercourse?
827. What are the findings suggestive of forcible sexual intercourse in an accused?
828. What is Lugol's iodine test?
829. What is smegma? What is its medicolegal significance?
830. Is it possible to rape a woman during sleep?
831. What is feigned rape?
832. Is it possible to say definitely that the girl has been raped by physical examination?
833. How will you issue your opinion in impotency certificate?
834. What is the different laboratory tests suggested in a case of rape?
835. Define sodomy.
836. Is sodomy an offence in India?
837. Who is a catamite?
838. What is pederasty?
839. What is gerontophilia?
840. Can a husband sodomise his wife?
841. What are the findings of sodomy in a victim?
842. What are the findings of sodomy in the accused?
843. What is Sin of Gomorrah?
844. What is tribadism?
845. What is lesbianism?
846. Is tribadism punishable in India?
847. What is incest? Is it punishable in India?
848. What is bestiality? Is it an offence?
849. What is psychosexual development (PSD)?
850. What are the various phases of psychosexual development?
851. What is eonism?
852. What is transvestism?
853. What is exhibitionism?
854. Who is a fetish?
855. What is masochism?
856. What is sadism?
857. What is sadomasochism?
858. What is necrophilia?
859. What is necrophagia?
860. What is lust murder?
861. Who is a voyeur?
862. Who is a nymphomaniac?
863. What is satyriasis?
864. What is frotteurism?
865. What is indecent assault?
866. What is the difference between sadism and masochism?
867. What is the difference between necrophagia and necrophilia?
868. Are eonism and transvestism same?
869. Is masturbation a crime?
870. When does masturbation become a crime?
871. How will you confirm that the given stain is of seminal origin?
872. How will you examine spermatozoa?
873. How you will confirm the advanced state of pregnancy?
874. What do you mean by live born and stillborn?
875. What is rule of Hasse's?
876. What is the medicolegal importance of the 7th month of pregnancy?
877. What are the characteristics of full term foetus?
878. What is lochia? What is the medicolegal importance of it?
879. How you can confirm the recent delivery of a woman?
880. What will be the findings in the uterus after recent delivery?
881. Define rape.
882. What do you mean by statutory rape?
883. Mention the findings in the accused of rape case?
884. Mention the findings in the victim of a rape case?
885. What do you mean by abortion? How do you classify it?
886. What is the difference between justifiable abortion and criminal abortion?
887. What do you mean by medical termination of pregnancy act (MTP) in India?
888. What are the indications of MTP?
889. What are the different methods of MTP?
890. Name some of the abortifacient drugs?
891. Give examples for ecbolics?
892. Mention how ecbolics differ in their action from emmanagogues?
893. What are the therapeutic methods for termination of pregnancy?
894. What are the complications of criminal abortion?
895. How will you diagnose on the autopsy table that the victim died of criminal abortion?

896. As a doctor what is your duty in criminal abortion case?
897. What can be the causes of death in criminal abortion?
898. What is section 312 IPC?
899. What is section 313 IPC?
900. What is section 314 IPC?
901. What is section 315 IPC?
902. What is section 317 IPC?
903. What is section 318 IPC?

Infanticide, Foeticide and Child Abuse

904. Define infanticide?
905. What are the acts of commission in infanticide?
906. What are the acts of omission in infanticide?
907. Is the offence of infanticide equivalent to murder?
908. How can you differentiate between stillborn and dead born?
909. What are the external autopsy findings of live birth?
910. What are the internal autopsy findings of live birth?
911. How can you establish time since birth by umbilical changes?
912. What are the gross changes of respiration in the lungs?
913. What are the histopathological changes in respired lungs?
914. Name some confirmatory tests for respiration in lungs.
915. What is Plaquet's test?
916. What is hydrostatic test?
917. What is a macerated foetus?
918. What is vagitus vaginalis?
919. What is vagitus uteralis?
920. What is the hydrostatic test? When is it not necessary?
921. What are the fallacies of hydrostatic test?
922. What is Breslau's second life test?
923. What is Wredin's test?
924. What is precipitate labour?
925. What do you infer by the terminology cord round the neck?
926. What is the medicolegal importance of caput succedaneum?
927. What information you can get from skin examination of newborn?
928. What is the medicolegal importance of air in GI tract of newborn?
929. What is the medicolegal importance of placenta?
930. What are the causes of death in infanticide?
931. Is abandoning of infant an offence?
932. Is concealment of birth an offence?
933. Caffey syndrome and Caffey coronary – are they same or different?
934. What do you know about cot deaths?
935. How will you diagnose cot deaths?
936. Define sudden infant death syndrome (SIDS).
937. Enumerate the various causes of SIDS.
938. What is auto-beverage syndrome?
939. What is overlying?
940. What is child sexual abuse?
941. What is reflex anal dilation test?

Forensic Psychiatry

942. What are the causes of delirium?
943. Define and classify types of delusion.
944. What is paranoid delusion?
945. Define and classify types of hallucinations?
946. What is illusion?
947. What is impulse in psychiatry?
948. What are the different types of impulse you know?
949. What is kleptomania?
950. What is pyromania?
951. What is mutilomania?
952. What is dipsomania?
953. What is dementia?
954. What is lucid interval? What is its medicolegal importance?
955. What do you mean by psychosis?
956. Who is a psychopath?
957. What is schizophrenia?
958. What is Korsakoff's psychosis?
959. What is cannabis psychosis?
960. What is schizophrenia?
961. What do you mean by feigned insanity?
962. What are the different methods of restraint of an insane?
963. What do you mean by direct restraint?
964. What is reception order on petition?
965. What are the civil responsibilities of an insane?
966. What are the criminal responsibilities of an insane?
967. What is McNaughten's rule?
968. What is section 84 IPC?
969. What is legal test?
970. What is ALI (American Law Institute) test?
971. What do you mean by partial responsibility?
972. What is the medicolegal importance of somnambulism and somnolentia?

FORENSIC TOXICOLOGY

973. What is a poison?
974. Define toxicology.
975. What do you mean by Dangerous Drugs Act?
976. What is the advantage of Pharmacy Act?
977. Which sections of IPC are related to administration of poison?
978. What do you mean by Drugs Control Act?
979. What are Schedule H and Schedule L drugs?
980. What are the common poisons in India?
981. What is an ideal homicidal poison? Give one example?
982. What is an ideal suicidal poison? Give one example?
983. Classify poisons?
984. Classify the neurotic poisons?
985. What are the routes of administration of poisons?
986. What are the routes of elimination of poisons?
987. What are the actions of poison?
988. What are the factors that modify the action of poison?
989. What do you mean by idiosyncrasy?
990. What is allergy?
991. Do allergy and hypersensitivity refer to the same condition?
992. What are the duties of a doctor in a case of poisoning?
993. What are the absolute contraindications for gastric lavage?
994. What are the relative contraindications of gastric lavage?
995. What are the complications of gastric lavage?
996. What are the various other names of gastric lavage tube?
997. Mention the different parts of gastric lavage tube?
998. Name the various gastric lavage fluids?
999. What are the indications to stop performing gastric lavage procedure?
1000. What is section 201, IPC?
1001. What is an antidote? How will you classify it?
1002. What are the constituents of universal antidote?

1003. Give some examples of physiological antidote.
1004. What are the contraindications of inducing emesis in a poisoning case?
1005. What are very common things to be preserved for chemical analysis in a living case of poisoning?
1006. What are the routine and special viscera to be preserved for chemical analysis in a case of poisoning at autopsy?
1007. What do you mean by vitriolage?
1008. In which of the corrosive poisons the chances of perforation of stomach are very common?
1009. What will be the colouration of teeth in sulphuric acid poisoning?
1010. Name the various preservatives used in preserving the viscera?
1011. Is there any contraindication of preserving the viscera, in rectified spirit, alcohol or formalin?
1012. In which of the corrosive poison the fatal period is shortest and quickest?
1013. What will be the line of treatment in a case of oxalic acid poisoning?
1014. What is carboluria? What is it due to?
1015. What will be the condition of pupil in phenol poisoning?
1016. What are the causes of death in strong corrosive poisons?
1017. Whether the carbolic acid has local or systemic action or both?
1018. What will be the changes in the stomach mucosa in a case of carbolic acid poisoning?
1019. How will you treat a case of acetyl salicylic acid (aspirin) poisoning?
1020. Classify the irritant poisons?
1021. What is the mechanism of action of phosphorus?
1022. How will you clinically diagnose a case of phosphorus poisoning?
1023. What will be the characteristic findings of liver in phosphorous poisoning?
1024. What do you mean by Phossy Jaw?
1025. What are organophosphorous compounds?
1026. What are the routes of absorption of organophosphorus compound?
1027. What is the mechanism of action of organophosphorous compound?
1028. What are the common symptoms you notice in organo phosphorous compound poisoning?
1029. How would you treat a case of organophosphorus compound poisoning case?
1030. What is atropinisation?
1031. What are oximes? What is oxime therapy for organo phosphorus compound?
1032. What will be the postmortem findings in organo phosphorus compound poisoning?
1033. What is endrin?
1034. How would you treat a case of endrin poisoning?
1035. Give examples for chlorinated hydrocarbon compounds?
1036. Name some poisonous arsenical compounds?
1037. Name the coloured compounds of arsenic?
1038. What are the main clinical symptoms of arsenic poisoning?
1039. Why arsenic is presently not a drug of choice for homicidal poisoning?
1040. What is Marsh test?
1041. What are Mee's lines?
1042. What is arsenophagy?
1043. What do you mean by mercurial erethism?
1044. What is mercurialentis?
1045. What are hatter's shakes?
1046. What is plumbism?
1047. What is the specific antidote used in the treatment of lead poisoning?
1048. What is blue vitriol?
1049. What are the postmortem findings of copper sulphate poisoning?
1050. What are organic vegetable irritant poisons? What do you mean by toxalbumin?
1051. Name some of the arrow poisons?
1052. What are Suis?
1053. What are the therapeutic and criminal uses of castor oil?
1054. Do you agree that the symptoms produced by abrin resemble viperine snakebite?
1055. What is the medicolegal use of ergot?
1056. How capsicum is used for criminal purpose?
1057. Do you know about Semecarpus anacardium? Mention its criminal use?
1058. What are the active principles of calotropis?
1059. What are the medicolegal aspects of calotropis?
1060. What is *Argemonium mexicana*?
1061. What are animal irritant poisons?
1062. What is the main site of action of cantharides?
1063. Classify poisonous snakes?
1064. How are Viperidae and Elapidae different from each other?
1065. What is the constituent of cobra snake venom?
1066. Is the fatal period of viperine snakebite longer or shorter than cobra snakebite?
1067. Can you diagnose the type of snake from its bite marks?
1068. What is the first aid treatment for snakebite cases?
1069. What is the modern line of treatment in a case of snakebite?
1070. What are the antidotes used for snakebite treatment and where they are manufactured in India?
1071. What are the signs and symptoms of a scorpion stinging?
1072. How will you treat a case of bee stinging?
1073. What are the symptoms of food poisoning?
1074. What are the microorganisms responsible for food poisoning?
1075. What is botulism?
1076. What is fish poisoning?
1077. What are ptomaines?
1078. What is insulin coma?
1079. How would you treat a case of swallowing powdered glass?
1080. How would you treat a case of swallowing pins and needles?
1081. Name some of the somniferous poisons?
1082. What is crude opium and standardized opium?
1083. What are the alkaloids of opium?
1084. What are the different symptoms produced by opium poisoning?
1085. What are the differential diagnoses of opium poisoning?
1086. What is the fatal dose of opium, morphine and pethidine?
1087. How would you treat a case of opium poisoning?
1088. What is the antidote for opium?
1089. Can there be an abstinence syndrome for opium?
1090. How will you treat abstinence syndrome?
1091. Will you do stomach wash in case of poisoning due to i/v morphine injection?

1092. What will be your typical postmortem finding in opium poisoning?
1093. What is the medicolegal importance of opium?
1094. What is the route of elimination of opium from body?
1095. What is morphinomania? How will you treat it?
1096. What will be the pupillary findings in case of an opium poisoning?
1097. Give some examples of inebriant poisoning?
1098. Name different preparations of alcoholic beverages?
1099. What is absolute alcohol?
1100. What is the difference between proof spirit and denatured spirit?
1101. What is the percentage of alcohol in rectified spirit?
1102. How will you diagnose a case of alcoholic intoxication?
1103. What are the clinical findings of alcoholic intoxication?
1104. What is the fatal dose of absolute alcohol?
1105. What is the fatal period of alcohol?
1106. How will you treat a case of acute alcohol poisoning?
1107. How will you treat a case of chronic alcohol poisoning?
1108. What is the mechanism of action of antabuse?
1109. What do you know about delirium tremens?
1110. What are the biochemical investigations done to confirm alcohol intoxication?
1111. Name the roadside test performed to diagnose the drunken driver?
1112. What are the signs and symptoms of methyl alcohol poisoning? How will you treat it?
1113. What is the site of action of methyl alcohol?
1114. What is the reaction of pupil in alcohol intoxication?
1115. What is McEwan's sign?
1116. What is alcoholic psychosis and Korsakoff's psychosis?
1117. What are the symptoms of chloral hydrate poisoning?
1118. What are knock-out drops?
1119. Mention the manifestations of kerosene poisoning? What is the line of treatment?
1120. What is the fatal dose and fatal period of kerosene poisoning?
1121. Classify barbiturates? What is their mechanism of action?
1122. What is drug automatism?
1123. What are the sign and symptoms of barbiturate poisoning?
1124. What is the cause of death in barbiturate poisoning?
1125. How do you treat a case of barbiturate poisoning?
1126. What are deliriant poisons?
1127. What are the main signs and symptoms of dhatura poisoning?
1128. What are the active principles in dhatura?
1129. What is *Cannabis sativa*? In which form it is used in India?
1130. What is *Hashish*? What are *Ganja, Majun, Bhang, Siddhi* and *Sabji*?
1131. What are the different stages of *Ganja* intoxication?
1132. What is running amok?
1133. What are cocaine bugs? What will be the colour of teeth in case of chronic cocaine poisoning?
1134. What are the symptoms of acute cocaine poisoning?
1135. What is cocainism?
1136. How will you treat a case of cocaine poisoning?
1137. What is the medicolegal aspect of cocaine?
1138. What is body packer's syndrome?
1139. What are poisonous mushrooms?
1140. What is lathyriasis?
1141. Can you tell about drug addiction and drug habituation?
1142. Name some hallucinogenic drugs.
1143. What is the mode of action of hallucinogenic drugs?
1144. How do you treat such a type of case?
1145. What are withdrawal symptoms?
1146. Name a spinal poison.
1147. What is the sight of action of strychnine?
1148. How do you differentiate between tetanus and strychnine poisoning?
1149. How are the viscera can be preserved in case of spinal poisoning?
1150. What will be the line of treatment in case of strychnine poisoning?
1151. Name some cardiac poisons
1152. What will be the symptoms of acute nicotine poisoning?
1153. What are the constituents of smoke emanating from cigar, cigarette, and pipe?
1154. What do you mean by Tobacco heart?
1155. What are *Nerium odorum* and *Cerbera thevetia*?
1156. Name some of the cardiac glycosides?
1157. What is *Aconitum napellus* and why it is called king of vegetable homicidal poison?
1158. What are the active principles of *Cerbera odollum*?
1159. What will be reaction of pupil in a case of aconite poisoning?
1160. What precautionary measure you would like to take at the time of treatment of aconite poisoning?
1161. What is the fatal dose fatal period of aconite poisoning?
1162. What is the mechanism of action of hydrocyanic acid?
1163. What are the signs and symptoms of hydrocyanic acid poisoning?
1164. What is the fatal dose and fatal period of hydrocyanic acid?
1165. What will be the line of treatment in a case of hydrocyanic acid poisoning?
1166. What will be the postmortem finding in case of hydrocyanic acid poisoning?
1167. What is the medicolegal importance of hydrocyanic acid?
1168. Name some of the asphyxiant poisons?
1169. What are the sources of carbon monoxide poisoning?
1170. What is the mechanism of action of carbon monoxide poisoning?
1171. What are signs and symptoms of carbon monoxide poisoning according to its saturation?
1172. What are the lines of treatment in a case of carbon monoxide poisoning?
1173. What will be the typical postmortem finding of the carbon monoxide poisoning?
1174. What is the normal percentage of carbon dioxide in air?
1175. What are the signs and symptoms of carbon dioxide poisoning?
1176. Name some of the war gases.
1177. What will be the line of treatment in case of lachrymator poisoning?
1178. What are the poisons that can be diagnosed by smell?
1179. What are the poisons that can be diagnosed from the reaction of pupil?
1180. What are the poisons that can be diagnosed from the changes in colour of the postmortem lividity?
1181. In a suspected poisoning case, when no poison is detected in the viscera how would you give final opinion?
1182. Name some of the poisons which are unstable in character.

1183. Name some common household poisons.
1184. What are the poisons available in the toilet?
1185. Is snakebite always accidental? Can you give an example of suicidal snakebite case?
1186. What are the poisons diagnosed by examining teeth and oral cavity?
1187. What are the poisons diagnosed by examining the eyes?

FORENSIC MEDICINE THEORY UNDERGRADUATE QUESTIONS

MEDICAL JURISPRUDENCE

The Doctor and the Law

1188. Mention the powers of chief judicial magistrate.
1189. Write short notes on:
    - i. Subpoena
    - ii. Cross-examination
    - iii. Exhumation
    - iv. Dying declaration
    - v. Valid consent
    - vi. Magistrate's inquest
    - vii. Documentary evidence.
1190. Explain the following and mention the medicolegal significance:
    - i. Informed consent
    - ii. Cross-examination
1191. Define inquest. Describe briefly different types of inquest.
1192. Describe briefly how the evidence of a witness is recorded in the Court of law.
1193. Describe the procedure of:
    - i. Recording dying declaration
    - ii. Exhumation

Ethics of Medical Practice

1194. Write an essay on 'professional misconduct'.
1195. Write briefly on: criminal malpractice, penal erasure
1196. Discuss briefly the duties of a medical officer in a court while giving evidence.
1197. Define professional misconduct. Illustrate with suitable examples. Briefly discuss the liability of medical practitioner in such cases.
1198. Give an outline of functions of Indian Medical Council.
1199. Write briefly on medical negligence.
1200. Describe various types of medical negligence with suitable examples.
1201. Define Infamous conduct in professional respect with suitable example. What is the action taken against the guilty doctor and by whom?
1202. Write short notes on:
    - i. Vicarious liability
    - ii. *Res ipsa loquitor*
1203. Write short notes on:
    - i. Privileged communications
    - ii. Professional secrets
    - iii. Functions of State Medical Council
    - iv. Infamous Conduct
    - v. Contributory negligence
    - vi. Covering

FORENSIC PATHOLOGY

Thanatology

1204. Define death. Describe postmortem changes that occur in a dead body in 12 hours after death.
1205. Mention briefly the immediate changes that occur after death.
1206. Explain the medicolegal importance of death.
1207. Describe postmortem changes occurring within 24 hours after death in summer.
1208. Discuss 'presumption of death' and 'presumption of survivorship' briefly.
1209. Discuss briefly the changes that occur as a result of the putrefaction.
1210. Discuss briefly the postmortem changes that are helpful in establishing time since death.
1211. Discuss the changes that a dead body undergoes during the first 24 hours after death. Discuss the medicolegal implications.
1212. Discuss the mechanism and factors affecting 'Postmortem cooling'. Write briefly how time since death is determined based on this.
1213. Discuss the various changes occurring in the eyes after death.
1214. Discuss the various parameters that help in establishing the 'time since death'.
1215. Enumerate and explain the changes seen in a dead body after 24 hours of death in India.
1216. Explain the mechanisms and various stages of 'rigor mortis' briefly.
1217. What is 'sudden death'? Discuss the cardiovascular and intracranial vascular causes of sudden death.
1218. What is 'suspended animation'? Write briefly on the types and causes of suspended animation.
1219. Explain how 'rigor mortis' differs from 'cadaveric spasm'.
1220. Describe the mechanisms of formation and medicolegal aspect of 'adipocere'.
1221. Explain the terminology 'casper's dictum' with relevance to putrefaction.
1222. What is Spalding's sign? Explain its medicolegal aspect.
1223. Describe four postmortem changes helpful in establishing 'time since death'.
1224. Explain 'colliquative putrefaction'. Add note on its medicolegal aspects.
1225. Describe the 'fauna of cadaver' and its application in estimating time since death.
1226. Describe the mechanism of formation and appearances of 'maceration'.
1227. What is 'thanatology'? Explain 'tripod of life' and 'Harvard's criteria' in relation to death.
1228. Explain how 'rigor mortis' differs from 'pugilistic attitude'.
1229. Explain and write medicolegal aspects of 'algor mortis'.
1230. What are maggots? Describe briefly how they can help in criminal investigation?
1231. Write short notes on:
    - i. Cadaveric lividity
    - ii. Features of a macerated foetus
    - iii. Cadaveric spasm
    - iv. Adipocere
    - v. Suspended animation.
1232. Write short notes on:
    - i. Vagal inhibition
    - ii. Molecular death
    - iii. Euthanasia.

Postmortem Examination

1233. Describe the incisions for opening the scalp and trunk during a medicolegal autopsy. Illustrate your answer with neat diagrams.
1234. Describe the procedure of dissecting the heart during a medicolegal autopsy.
1235. Explain 'negative autopsy' relevant to the causes and medicolegal aspects.
1236. Write briefly on 'objectives' and 'formalities' (rules) of medicolegal autopsy.
1237. Enumerate the objectives of medicolegal autopsy. Explain the routine skin incisions for opening the scalp and thorax and abdomen.
1238. Discuss 'negative autopsy' highlighting the various causes.
1239. What are the precautions to be taken in carrying out medicolegal autopsy? Enumerate the rules for medico-legal autopsy.
1240. Describe the procedure of dissection of the heart during autopsy.
1241. Enumerate and explain the objectives of medicolegal autopsy.
1242. Describe any four aspects of information that could be deduced by the examination of skeletal remains.
1243. Write short notes on:
    i. Consent in autopsy examination
    ii. Bloodless dissection of neck
    iii. Scalp incision and opening of skull
    iv. Removal of brain
    v. Dissection of heart
    vi. Demonstration of pulmonary embolism.

Violent Asphyxial Death

1244. Describe PM appearances in a case of drowning.
1245. Define hanging. Discuss the mechanism of death and Postmortem appearances in a case of hanging.
1246. Describe briefly the postmortem appearances in a case of hanging.
1247. Define asphyxia. Briefly describe the postmortem findings in a case of strangulation.
1248. Discuss the mechanism of death and autopsy diagnosis of drowning.
1249. A dead body of a female with a ligature around the neck is brought for autopsy. Discuss the medicolegal problems and the line of approach to solve them.
1250. Discuss the pathophysiology of drowning. What are the autopsy findings in such cases?
1251. Discuss the mechanism of death in cut-throat injuries. How will you establish that death was suicidal?
1252. Define asphyxia. Classify various types of violent asphyxial deaths and explain the external autopsy findings of any one of them.
1253. Explain the principle, procedure and inference of 'diatom test'
1254. Explain the significance of salivary dribbling mark and fracture of hyoid bone in a dead body
1255. Define and classify hanging. Discuss causes of death due to hanging.
1256. Explain 'suspended animation' and write on its medicolegal significance.
1257. Write short notes on:
    i. Traumatic asphyxia.
    ii. Café coronary
    iii. Suspended animation
    iv. Diatoms test
    v. Partial hanging
    vi. Fracture hyoid bone
    vii. Salivary marks in hanging
    viii. Sexual asphyxia
    ix. Hydrostatic test
1258. Explain and mention medicolegal importance of suspended animation.
1259. Give an account of Getler's test.

Forensic Identity, Trace Evidence, Forensic DNA Profiling

1260. Discuss the medicolegal importance of age?
1261. How will you ascertain 'a hair is human in origin'?
1262. Discuss the principles and medicolegal importance of:
    i. Precipitin test
    ii. Takayama's test
    iii. Acid-phosphse test
    iv. Polygraph.
1263. Explain the following and mention their medicolegal importance:
    i. Poroscopy
    ii. Atavism
    iii. Intersex
    iv. DNA profiling.
1264. Write short notes on:
    i. Poroscopy
    ii. Sex chromatin
    iii. Rule of Hasse
    iv. Dactylography
    v. Pearson's formula
    vi. Superimposition photography
    vii. Cephalic index
    viii. Tattoo marks
    ix. Gustafson's formula.

CLINICAL FORENSIC MEDICINE

Trauma

1265. Describe the characteristic features of injuries produced by the discharge of rifled fire arm.
1266. A dead body of a woman aged 20 years is recovered from a burning hut. How will you establish whether death has occurred due to burns?
1267. Mention the different types of intracranial haemorrhages and discuss their medicolegal significance.
1268. Outline the features of suicide by firearm. What circumstances of death by firearm arise suspicion of foul play?
1269. Write briefly on different types of skull fractures.
1270. Give an outline on causes of death from wounds.
1271. Define grievous hurt with suitable examples.
1272. Describe the appearance seen in an entry wound caused by revolver shot at a length of 3 cm and 10 m.
1273. Classify mechanical injuries. Discuss causes of death from mechanical injuries.
1274. Classify mechanical wounds. Describe briefly the various types of abrasions and their medicolegal significance.
1275. Define abrasion. Write briefly on causes and medicolegal importance of 'Imprint abrasion'
1276. Discuss 'lucid interval' with reference to its causes and medicolegal aspects.

1277. Define contusion. Discuss briefly on its causes and medicolegal importance.
1278. Define and describe briefly the various types of lacerations.
1279. Discuss how 'time since injury' and 'direction of injury' is determined from abrasions.
1280. Describe how 'time since injury' and 'weapon of crime' is determined from contusions.
1281. Define and describe briefly the various types of contusions.
1282. Describe 'wound of entry' by a rifle at contact shot and distant shot range firing.
1283. Define and describe briefly the various types of abrasions.
1284. Describe the differences between antemortem bruise and postmortem lividity
1285. Describe 'imprint abrasion'. Write on its medicolegal aspects briefly.
1286. Describe the differences between antemortem and postmortem bruise.
1287. Define laceration. Discuss briefly about its causes and medicolegal importance
1288. Describe 'incised like laceration'. Discuss its medicolegal aspects briefly.
1289. Describe 'incised wound' and 'incised like laceration'. Discuss on its medicolegal aspects briefly.
1290. What are 'closed' and 'open' head injuries? Add a note on skull fracture.
1291. Describe examination of a stab wound. Write on its medicolegal aspects.
1292. What is coup and contracoup injury to brain? Mention the role of meanings and CSF in preventing brain injury.
1293. Enumerate and explain intracranial haemorrhages with a neat-labelled diagram.
1294. Enumerate the spinal cord injuries and discuss briefly the causes of spinal injuries.
1295. Define grievous hurt giving relevant examples.
1296. Enumerate the differences between the wound of entry by a rifle and a shotgun at close shot range firing.
1297. What are chop wounds/chop lacerations? Describe briefly about the pathology and complications of whiplash injury.
1298. Explain primary impact and secondary impact pedestrian injuries in relation to 'road traffic accident'.
1299. Write briefly on causes and medicolegal aspects of scalds
1300. Describe stab wound by kitchen knife and dagger. Add a note on concealed punctured wound.
1301. Enumerate brain injuries and explain the mechanisms involved.
1302. Explain what is an 'abrasion collar'? Mention its medicolegal significance.
1303. Define grievous hurt. Discuss the causes of death from injuries.
1304. Discuss briefly the Locard's principle of exchange.
1305. Classify firearms. Describe the entry wound from shotgun at varying ranges.
1306. Explain how a contusion can be differentiated from PM staining?
1307. Explain how incised wound is differentiated from lacerated wound?
1308. Define grievous hurt and mention the section of Indian Penal Code under which it is stated.
1309. A superficially charred dead body of a female was procured from a burnt hut. Discuss the types of investigation you would adopt in arriving cause of death.
1310. Explain the 'tattooing' in firearm injuries.
1311. Discuss the points on which one can base an opinion that the discolouration present on a weapon is due to human blood.
1312. How will you ascertain that the injury is the wound of entry of bullet?
1313. How will you ascertain that the death is due to air embolism?
1314. How will you ascertain that the wound is antemortem?
1315. Short notes:
    i. Abrasion collar
    ii. Lucid interval
    iii. Contrecoup injury
    iv. Arborescent marks
    v. Suspended animation
    vi. Cardiac tamponade
    vii. Antemortem burns
    viii. Lucid interval
    ix. Patterned abrasion
    x. Grievous hurt
    xi. Patterned injury
    xii. Primary impact injury
    xiii. Air embolism
    xiv. Battered baby
    xv. Vagal inhibition
    xvi Antemortem burns
    xvii Hesitation cuts
    xviii Scalds
    xix. Extradural haemorrhage.

Sexual Jurisprudence (Virginity, Pregnancy, Delivery, Abortion, Sexual Offences, Infanticide)

1316. Define 'rape'. How will you examine a victim of a alleged rape. Indicate the possible value of your finding.
1317. What are the precautions to be taken while examining a victim of rape?
1318. Enumerate and explain the signs of virginity. Write on its medicolegal importance.
1319. Explain 'sexual asphyxia' with an example. Write on its medicolegal significance.
1320. Write briefly on 'superfoetation' and 'superfecundation'. Mention their medicolegal importance.
1321. Explain the terminology 'lochia'. Write on its medicolegal importance.
1322. Explain the terminology 'rape'. Enumerate 'corroborative findings of rape'.
1323. Enumerate and explain two findings suggestive of live birth and still-birth.
1324. Explain briefly the principle and procedure of hydrostatic test.
1325. Define rape. Explain the corroborative findings of rape in a victim.
1326. Explain the precautions to be taken by a doctor prior to and during examination of an alleged victim of rape.
1327. Define MTP act. Explain the rules of MTP.
1328. Enumerate and explain the immediate and late complications of criminal abortion.

1329. Enumerate and explain the findings in a victim of alleged sexual offence of Sodomy.
1330. Explain the corroborative findings of rape in an accused.
1331. Explain the corroborative findings of sodomy in an accused.
1332. Explain the term pederasty, pedophilia. Add note on medicolegal aspect of both.
1333. Write short notes on:
    i. Defloration
    ii. Medicolegal importance of precipitate labour
    iii. Sexual asphyxia
    iv. Cot death
    v. Signs of virginity
    vi. Complications of criminal abortion
    vii. Recent signs of sexual intercourse in a virgin
    viii. False virgin
    ix. Hydrostatic test
    x. Nullity of marriage
    xi. Macerated foetus
    xii. Battered baby
    xiii. Causes of impotency in male
    xiv. Hymen
    xv. Affiliation cases
    xvi. Act of omission in a case of infanticide
    xvii. Disputed paternity
    xviii. Transvestism
1334. What are indications for terminating pregnancy in India?
1335. Give a brief account of fallacies of hydrostatic test.
1336. Discuss the points on which one can base an opinion that:
    i. The staining on the cloth is caused by semen
    ii. A woman delivered recently
    iii. A male who is habitual passive agent of sodomy
1337. Explain the following and mention their medicolegal significance
    i. Necrophagia
    ii. Superfecundation

Forensic Psychiatry

1338. Describe the methods of placing an insane person under restraint.
1339. Describe the procedure of restraining an insane person.
1340. Explain the terminology 'feigned insanity'. Enumerate the differences between true and feigned insanity.
1341. Explain the various types of restrain of mentally ill with reference to indications and methods involved.
1342. Describe 'voluntary direct restraint' and 'reception order on petition' types of restraint of mentally ill.
1343. Describe the methods of 'reception after judicial inquisition' and 'reception of mentally ill criminal'.
1344. Enumerate and explain the civil responsibilities of a mentally ill person.
1345. Explain the civil responsibilities of a mentally unsound person with reference to management of property, marriage and divorce, competency as a witness, and testamentary capacity.
1346. What is McNaughten's rule? Explain Section 84 of Indian Penal Code.
1347. What is McNaughten's rule? Explain Section 85 of Indian Penal Code with relevance to criminal responsibility of mentally ill person.
1348. Write briefly the historical aspect and criticism of McNaughten's rule.
1349. Explain 'impulse' and 'somnambulism' and write on its medicolegal aspects.
1350. Explain the equivalent section in Indian Penal Code to McNaughten's rule.
1351. What are 'delirium' and 'impulse'? Discuss the medicolegal aspects of each one briefly.
1352. Write short notes on:
    i. Illusion
    ii. Testamentary capacity
    iii. McNaghten's rule
    iv. Delusion
    v. Hallucination
    vi. Testamentary capacity
    vii. Feigned insanity
    viii. Impulse
1353. Explain delusion with suitable examples.
1354. Explain and mention the medicolegal importance of delusion.

FORENSIC TOXICOLOGY

1355. Classify neurotic poisons. Describe the mode of action, signs, symptoms, management and postmortem findings of acute opium poisoning.
1356. Classify poisons. Discuss the clinical findings, management and autopsy findings in a case of phenobarbiturate poisoning.
1357. Define drug dependence. Mention the various forms in which *Cannabis indica* is used.
1358. Describe drunkenness. Discuss value of clinical findings in relation to laboratory results in a case of examination for drunkenness.
1359. Describe duties of medical practitioner in a case of suspected poisoning.
1360. Describe signs and symptoms and postmortem appearance of con. sulphuric acid poisoning.
1361. Describe signs, symptoms and treatment in a case of methyl alcohol poisoning.
1362. Describe signs, symptoms and treatment in a case of organophosphorous poisoning.
1363. Discuss briefly the clinical features, treatment and postmortem appearances in a case of methyl alcohol poisoning.
1364. Write mode of action, signs, symptoms, treatment and postmortem findings in acute 'cyanide' poisoning.
1365. Discuss briefly the tests to confirm dead-born and still-born children.
1366. What is meant by epidemiology of poisoning? Enumerate the various incidences of poisoning in India. Add a note on 'ideal suicidal poison' giving suitable examples.
1367. Discuss the legal duties of a doctor in a suspected case of poisoning.
1368. Write briefly on autopsy findings in stomach in fatal cases of poisoning due to concentrated sulphuric and carbolic acid poisoning.
1369. Write briefly on line of treatment of snakebite and scorpion stinging cases.
1370. Enumerate the various 'house hold poisons'. Write briefly on line of treatment of poisoning with each of them.
1371. Explain the term 'drug habituation' and 'drug addiction' giving suitable examples.

1372. Add a note on 'antabuse therapy'.
1373. Explain the term occupational toxicology and environmental toxicology.
1374. What does analytical toxicology mean? Explain four bedside common laboratory tests.
1375. Discuss briefly changes in urine in cases of poisoning by carbolic and oxalic acid.
1376. Write briefly mechanism of action and autopsy findings in cases of death due to carbon monoxide and cyanide poisoning.
1377. Enumerate the causes and line of treatment of bacterial and chemical food poisoning.
1378. Write briefly on line of management of a case of barbiturate poisoning. Add a note on barbiturate automatism.
1379. Give an account of medicolegal implication of:
    i. Delerium tremens
    ii. Vitriolage
1380. Give an outline of the management of the following poisoning cases:
    i. Methyl alcohol
    ii. Pheno-barbiturate
    iii. Potassium cyanide
    iv. Strychnine
1381. Discuss salient autopsy findings of the following:
    i. Carbon monoxide poisoning
    ii. Chronic arsenic poisoning
    iii. Nitric acid poisoning
    iv. Organophosphorus poisoning
1382. Discuss the salient clinical features of following:
    i. Chronic lead poisoning.
    ii. Dhatura poisoning
    iii. Morphine poisoning
    iv. Oxalic acid poisoning.
1383. Describe autopsy findings in sulphuric acid poisoning.
1384. Give an outline of management of snakebite.
1385. Mention the salient clinical features of organophosphorous poisoning.
1386. What does phossy jaw mean?
1387. Write short notes on:
    i. Delirium tremens
    ii. Constricted pupil
    iii. Withdrawal symptoms
    iv. Drunkenness
    v. Plumbism
    vi. Treatment of snake bite
    vii. Chelating agents
    viii. Food poisoning
    ix. Gastric lavage
    x. Cerberin
    xi. Erethism
    xii. Toxalbumin
    xiii. Drug dependence
    xiv. Schedule 'H' and 'L' drugs
    xv. Phossy jaw
    xvi. Vitriolage
    xvii. Treatment of snake bite
    xviii. Emetics
    xix. Drug automatism
    xx. Negative chemical analysis report
    xxi. Run amok
    xxii. Treatment of corrosive acid poisoning
    xxiii. Magnan's symptoms
    xxiv. Plumbism
    xxv. Universal antidote
    xxvi. Treatment of barbiturate poisoning.
1388. Describe the autopsy findings of carbolic acid poisoning.
1389. Give an account of the mode of action of potassium cyanide.
1390. Give an outline of the management of methyl alcohol poisoning.
1391. Mention the materials that are used for chemical analysis in suspected case of poisoning.
1392. What are the likely complications of gastric lavage.

FORENSIC MEDICINE THEORY POSTGRADUATE QUESTIONS

MEDICAL JURISPRUDENCE

The Doctor and the Law

1393. Discuss the legal and ethical issues of euthanasia.
1394. Discuss in detail the impact of Consumer Protection Act (COPRA) in medical practice.
1395. Discuss the medicolegal problems a doctor has to face during exhumation?
1396. Discuss the medicolegal problems associated with organ transplantations.
1397. Write your critical comments on the various methods of judicial execution.
1398. Discuss on role of a doctor in crime investigation.
1399. Discuss false accusation a medicolegal expert, defamation suits and justice.
1400. Discuss role of forensic pathologist in crime scene investigation.
1401. Discuss on handling a medicolegal cases under critical situations of law and order.
1402. Write an essay on Indian judiciary system and police services with respect to medicolegal case investigation involving the expertise of forensic medicine.
1403. Discuss on consumer forum with specific reference to cases of medical malpractices.
1404. Write short essay on:
    i. Dying declaration
    ii. Medical examiner system
    iii. Magistrates and their role in criminal justice
    iv. Death in custody
    v. Dowry death.
1405. Sections of medicolegal significance under Indian Penal Code (IPC)
1406. Sections of medicolegal relevance under Cr PC of India.
1407. Summons, warrant and bail.

Ethics of Medical Practice

1408. Discuss the role of the Medical Council of India in regulating the standard of medical education in India. Offer three suggestions (which you regard important) for improving the standard of medical education in India; defend your suggestions with logical arguments based on available data.
1409. Discuss the recent developments concerning euthanasia and your views on legalising euthanasia in India.

1410. Discuss the relevance of consent and confidentiality in medical practice
1411. Define consent. Discuss the relevance of consent in medical profession.
1412. Discuss the legal implications of the Human Organ Transplantation Act, 1994, enacted in India.
1413. Human Organ Transplantation Act, 1994. Discuss.
1414. A dangerously ill patient, after consuming a poisonous chemical was refused admission and treatment by two nursing homes. He was finally admitted to the district headquarter's hospital where he died, despite treatment, two hours after admission. Discuss the issues involved in this case.
1415. Define consent. Discuss the relevance of consent in medical practice with examples.
1416. Discuss fitness to practice medicine by a registered general practitioner.
1417. Discuss your views on patients right of access to health records.
1418. Discuss briefly the ethics of human experimentation.
1419. What facilities are required to conduct a postgraduate degree course in forensic medicine? Substantiate.
1420. Discuss a rash and negligent act giving suitable examples.
1421. Discuss how the State ensures that the citizens get proper medical care by legal and judicial processes.
1422. Write about Medical Council of India with relevance to its formation and functions assigned.
1423. Discuss in detail about HIV infected people and the public attitude and law.
1424. Discuss the medical and medicolegal aspects of deaths associated with surgery and anaesthesia.
1425. Discuss the formalities and requirements to be fulfilled by a medical graduate in establishing practice of medical profession in India.
1426. Discuss about patient autonomy with reference to diagnostic tests and treatment in medical practice.
1427. Discuss the recent developments concerning euthanasia. Add a note on your opinion on legalising euthanasia in India.
1428. Outline the general principles involved in 'law and emergency care'.
1429. Write short essay on:
    i. In-vitro fertilization and embryo transfer.
    ii. Inevitable accidents
    iii. Jehovah's witness.
    iv. Medical records and law
    v. Gratuitous services
    vi. Professional death sentence
    vii. Doctrine of res ipsa loquitor
    viii. Contributory negligence
    ix. Syndrome of evidence
    x. Products liability
    xi. Juvenile delinquency
    xii. Penal erasure.
    xiii. Vicarious responsibility with illustrative cases
    xiv. Proofs of medical negligence
    xv. Consent in relation to the examination of subjects involved in criminal cases
    xvi. Vicarious liability in civil and criminal cases.

FORENSIC PATHOLOGY

Thanatology

1430. After postmortem examination how do you diagnose a case of death due to myocardial infarction?
1431. Describe how will you be able to determine the time that has elapsed since death (postmortem interval) in a case of medicolegal postmortem examination done in your mortuary today.
1432. Describe the blood supply of brain. Write an account of sudden natural deaths due to pathological changes affecting it.
1433. Describe the different methods of estimating postmortem interval. Discuss their reliability
1434. Describe the utilities of postmortem chemistry of CSF, blood.
1435. Discus the late postmortem changes observed in a cadaver and evaluates the various changes to determine the 'time since death'?
1436. Discuss 'brain death'.
1437. Discuss the concepts of brain death and its relevance and application in the medical and medicolegal field at present.
1438. A bundle of bones have been forwarded to you for your expert opinion. How would you proceed to assist the law, bearing in mind recent advances in this field?
1439. On a suspicion of foul play a completely skeletonised body has been exhumed from the grave. How will you establish the identity, cause and time of death?
1440. Discuss the medicolegal significance of moment of death with special reference to organ transplantation surgery.
1441. Discuss the incidence, pathogenesis and morphology of myocardial infarction.
1442. Discuss the merits and demerits of the various medicolegal systems to ascertain the cause and manner of death. Indicate your preference with reasons.
1443. Discuss the method of examination of a skeletal remains.
1444. Discuss the pathology of ischaemic heart disease. Write on its medicolegal importance?
1445. Discuss the postmortem changes, which occur in cerebrospinal fluid. How far these changes are useful to determine 'time of death'?
1446. Discuss the recent advances in establishing postmortem interval (PMI) in a decomposed dead body.
1447. Discuss the recent advances on decomposition and putrefaction highlighting their differences.
1448. Discuss the value of chemical analysis of the spinal fluid in ascertaining the time since death.
1449. Discuss the various factors, which modify diagnostics and emptying of stomach. Explain their medicolegal importance.
1450. Prepare a brief write up about postmortem hypostasis.
1451. Recent advances in the estimation of age of an infarction.
1452. Describe the utilities of postmortem chemistry of CSF, blood.
1453. Write an essay on 'death'.
1454. Write briefly about bacterial putrefaction of dead bodies and the factors modifying it.
1455. Write briefly on recent advances on establishing time since death (PMI) on medicolegal autopsy findings.

1456. Write short essay on:
    i. Apparent death
    ii. Artifacts in forensic pathology
    iii. Brainstem death
    iv. Cadaveric spasm
    v. Death certificate
    vi. Donation of cornea
    vii. Diagnostic chemistry of CSF at autopsy
    viii. Embalming
    ix. Emptying time of stomach
    x. Forensic entomology
    xi. Hyaline membrane disease
    xii. Moment of death
    xiii. Postmortem interval and analysis of CSF and vitreous humor
    xiv. Presumption of survivorship
    xv. Signs of maceration
    xvi. Spaulding's sign
    xvii. Vagal inhibition
    xviii. Valvular diseases of heart.

Postmortem Examination

1457. Prepare a plan for the construction of a modern mortuary. Discuss your plan giving suitable reasons.
1458. Give suggestions to construct a mortuary to a rural medical college.
1459. Give suggestions to construct an ideal mortuary to a 1000 bedded tertiary care hospital attached to a medical college.
1460. Discuss the autopsy protocol in deaths due to 'AIDS' and 'hepatitis B'. Mention your comments if you have to offer any.
1461. Discuss how will you interpret the findings of livebirth observed in foetal autopsies, and points of consideration as to how long the child survived.
1462. Discuss the medicolegal informations, which are available from dismembered human hand.
1463. How do you proceed to examine a bunch of bones sent to you and give your opinion covering all medicolegal aspects?
1464. Discuss "correlation of postmortem findings with road side evidence".
1465. Discuss the various medicolegal aspects of autopsy on "confirmed case of AIDS."
1466. Discuss the importance of thorough postmortem examination on a completely charred body.
1467. Write short essay on:
    i. Autopsy in anaesthetic deaths
    ii. Artifacts and postmortem findings confused with child abuse
    iii. Embalming
    iv. Special autopsy techniques
    v. Bloodless dissection of neck
    vi. Removal of spinal cord
    vii. Subendocardial haemorrhages
    viii. Foetal autopsy
    ix. Negative autopsy
    x. Preserving an unidentified dead body in a morgue
    xi. Cadaver donation.

Violent Asphyxial Death

1468. What is anoxia? Mention types of anoxia and discuss the pathophysiology and medicolegal importance of asphyxia.
1469. Discuss the problems involved in establishing the cause and manner of death in bodies recovered from water.
1470. Discuss hanging with special emphasis on the medicolegal issues involved in such cases.
1471. Discuss smothering with special emphasis on the medicolegal issues involved in such cases.
1472. Discuss drowning in salt water. Add a brief note on post-immersion syndrome.
1473. Discuss the problem of determination of cause of death in cases of bodies recovered from water.
1474. Dead body of a teenaged girl is found floating in a well. How can it be proven that the death was due to drowning?
1475. With a history of dowry dispute, the dead body of a newly wed girl has been thrown in to domestic well following strangulation. How best you are going to reconstruct the offence and establish the cause of death.
1476. Write short essay on:
    i. Choking
    ii. Bloodless dissection of the neck
    iii. Postmortem findings in traumatic asphyxia.
    iv. Evidence of death due to asphyxia.
    v. Estimation of time since death of a body removed from water.

Forensic Identity, Trace Evidence, Forensic DNA Profiling

1477. A government employee claims that his age is 50 years. How do you establish his age?
1478. Describe the changes which occur in the skull sutures which establish the approximate age of a person
1479. Describe the methods for determination of stature from long bones.
1480. Describe the process of dentition. Discuss the forensic aspects of odontology.
1481. Describe the role of X-ray in forensic medicine
1482. Describe the various methods of identification by means of blood examination
1483. Discuss teeth, a test of age.
1484. Discuss briefly the DNA profiling and its role in forensic investigation.
1485. Discuss how you will determine age from teeth in the living as well as the dead.
1486. Discuss recent advances in establishing the identity of a highly decomposed cadaver.
1487. Discuss the changes in pubic symphysis in relation to age and how much accuracy can be established from these changes.
1488. Discuss the metamorphosis occurring over the symphyseal face and relevance in estimating the age.
1489. Discuss the modern techniques in establishing the identity of an individual.
1490. Discuss the problem of determination of age from skeletal bones.
1491. Discuss the utility of odontology in solving medicolegal problems.

1492. Discuss the value of radiological examination in medicolegal age determination cases
1493. Discuss the various medicolegal questions which can be answered by a forensic pathologist on examination of human skeletal remains found in a forest.
1494. Discuss the various system of data recording of dentition and its value in mass disasters, identification and crime.
1495. How far examination of teeth helpful in estimation of age of an individual—discuss.
1496. How the status of ossification of bones determined? How is it useful for determining the age of an individual?
1497. How to estimate the age of a dead body by pubic symphyseal changes?
1498. Role of clavicle in determination of age.
1499. Write an essay on sex chromatin and intersex conditions.
1500. Write short essay on:
    i. Costal cartilage calcification pattern
    ii. Ear prints
    iii. DNA fingerprinting
    iv. Foot outline ratio (FOR)
    v. Mandibular canine index (MCI)
    vi. Foetal haemoglobin
    vii. Forensic odontology
    viii. Forensic dentistry-radiology aspects
    ix. Genetic sex and gonadal sex
    x. Gustafson's method
    xi. Gustafson's formula
    xii. Age changes at symphyseal surface of pubic bone
    xiii. Skeletal appearances of neonate
    xiv. Intersex
    xv. Medicolegal examination of hair
    xvi. Hair
    xvii. Identification of blood from different species
    xviii. Radiological aspects of forensic dentistry
    xix. Sex determination in a sports person.
    xx. Sex identification from sternum
    xxi. Superimposition technique
    xxii. Stature from nose size
    xxiii. Teeth bite marks.
1501. Describe the laboratory technique and the medicolegal significance of precipitin test.
1502. Discuss the role of forensic serology in crime investigation.
1503. Discuss the medicolegal aspects of blood grouping in exclusion of paternity.
1504. Discuss the recent advance in the laboratory investigation of biological stains.
1505. Give a detailed account of group secreting substances in body tissues and their significance in crime investigations.
1506. Enumerate and explain the various immunological tests adopted in crime investigations routinely in India.
1507. State briefly the composition of human semen and describe with diagrams the morphology of normal human spermatozoa. How and why the vaginal smear is taken in case of alleged rape?
1508. Write short essay on:
    i. Neutron activation analysis
    ii. Polygraph
    iii. Locard's principle of exchange
    iv. Laboratory detection of salivary stains
    v. Precipitin test
    vi. Modern techniques for the detection and quantification of poisons/drugs.

CLINICAL FORENSIC MEDICINE

Trauma

1509. Define embolism. Outline the different forms of embolism and their medicolegal injunction.
1510. A dead body of a young woman aged 21 years is recovered from a burning hut. Discuss briefly how will you establish whether the death has occurred due to burns.
1511. Discuss wound healing. Briefly discuss the medicolegal aspects of a delay in wound healing.
1512. Discuss briefly on bomb blast explosion injuries and a detailed plan on investigation of terrorist bombing incidence.
1513. Describe and discuss the injuries sustained by the unrestrained driver and front seat passengers in frontal automobile accident.
1514. Discuss wound healing and its significance in medicolegal practice.
1515. Discuss wound healing. Briefly discuss the medicolegal aspects of a delay in wound healing.
1516. Discuss the relationship of trauma with disease and stress. Discuss in detail the medicolegal issues involved therein.
1517. Discuss in detail the thermal injuries.
1518. Discuss the pathology of cerebral compression of traumatic origin.
1519. Write an essay on the 'role of a doctor in the investigation of fatal transportation accidents'
1520. Medicolegal significance of different types of skull bone fractures.
1521. Discuss the mechanism of craniocerebral injuries. Add a note on cerebral trauma.
1522. Discuss family violence. Add a note on its prevalence in India.
1523. Discuss various aspects of posttraumatic embolism.
1524. Write an essay on adult respiratory distress syndrome (ARDS).
1525. Describe the normal bone formation and healing of a fracture in the human body.
1526. Discuss how traffic accident reconstruction is possible from autopsy appearances.
1527. Discuss the effects of blunt trauma on head.
1528. Write about the recent advances in the determination of age of wounds.
1529. Discuss briefly on craniocerebral injury and the various mechanisms involved.
1530. Discuss how you can differentiate the suicidal, accidental and homicidal falls by examining the victim physically.
1531. Describe the role of medicolegal expert in investigations fatal burns trauma.
1532. Discuss the role of a doctor in handling a case of assault due to sharp weapons.
1533. Write an account on role of forensic pathologist in mass disaster mitigation.
1534. Discuss blast injuries and highlighting various mechanisms involved in producing them.
1535. Discuss in detail terminal ballistics with reference to rifled firearms wounds.
1536. Write an account on role of microscopy in crime investigation of firearm fatalities.
1537. Discuss the role and utility of radiological examination in cases of trauma.
1538. Discuss the role of radiology in medicolegal practice.

1539. Write short essay on:
   - i. Microorganisms associated with burns
   - ii. Cleavage lines of Langer
   - iii. Acute radiation syndrome
   - iv. Ageing of bruises
   - v. Exploding ammunition
   - vi. Status thyamolymphaticus
   - vii. Hypothermic necrosis of tissue
   - viii. Endogenic and ectogenic burns sustained during electrocution
   - ix. Bacterial infection in road accidents
   - x. Vital reaction
   - xi. Use of radiography in laryngeal injuries
   - xii. Contact firearm entry wound
   - xiii. Bomb explosion wounds
   - xiv. Concealed injuries
   - xv. Explosive bullets
   - xvi. Endogenous burns
   - xvii. Ewing's postulates
   - xviii. Cardiac tamponade
   - xix. Semi-microradiography
   - xx. Acute respiratory distress syndrome
   - xxi. Primary impact injuries
   - xxii. Remote complications due to firearm injuries
   - xxiii. Air embolism
   - xxiv. Circle of Willis
   - xxv. Acute adrenal haemorrhage
   - xxvi. Cardiac tamponade
   - xxvii. Fat embolism
   - xxviii. Death due to lightning
   - xxix. Fat embolism
   - xxx. Cardiopulmonary resuscitation.

Sexual Jurisprudence (Virginity, Pregnancy, Delivery, Abortion, Sexual Offences, Infanticide)

1540. A 60-year-old male is accused of having raped a girl aged twelve. Discuss the physical and laboratory evidence which would active the medicolegal problems
1541. Critically evaluate the rape law in India. Indicate the modern trends about the subject in India and abroad.
1542. Critically evaluate the rape law is our country. Indicate the modern trends in the subject in India and abroad.
1543. "An intact hymen is not an absolute proof of virginity"—Elucidate.
1544. Mention the uses and misuses of amniocentesis. Discuss their medicolegal implications.
1545. Discuss the value of medical examination in corroborating the prosecutor in alleged cases of rape.
1546. Discuss the newer techniques in paternity blood grouping.
1547. Write about surrogate motherhood and the medicolegal problems associated with the procedure.
1548. Discuss the differences in the legal and medical concepts of live birth. How will you establish live birth in a newborn by autopsy examination.
1549. Describe foetal blood circulation. Discuss the causes of death in criminal abortion.
1550. Discuss foetal circulation and its significance.
1551. Discuss the interpretations possible from laboratory investigations to correlate with clinical findings in cases of sexual offences.
1552. Discuss on perineal and anal examination findings in an alleged sexual offence victim.
1553. Findings of an examination in a dead body of a child.
1554. Write short essays on:
   - i. Signs of maceration
   - ii. Custodial rape
1555. Write short essays on:
   - i. Male impotence
   - ii. Wife battering
   - iii. Umbilical cord
   - iv. Foetal haemoglobin
   - v. Medical Termination of Pregnancy Act, 1971
   - vi. Acts of omission in relation to infanticide
   - vii. Custodial rape
   - viii. Male impotence
   - ix. Gerontophilia
   - x. Paternity tests
   - xi. Surrogate mothers
   - xii. Cot death
   - xiii. Discuss sudden infant death syndrome
   - xiv. Law on rape
   - xv. Male menopause
   - xvi. Rh incompatibility
   - xvii. Significance of hymen as a proof of virginity
   - xviii. Medicolegal significance of AIDS
   - xix. Violence at home
   - xx. Genetic sex and gonadal sex
   - xxi. Disorders of female genitalia.

Forensic Psychiatry

1556. Write the procedure of examination and diagnosing a case of insanity and for the issuing of a certificate of insanity.
1557. Describe the procedural formalities for detention of patient in mental hospitals at present and suggest improvements.
1558. Discuss the criminal responsibility of insane.
1559. Discuss the drug-induced psychosis and its criminal responsibility
1560. Write short essay on:
   - i. Testamentary capacity
   - ii. Delusions
   - iii. McNaughten's rule
   - iv. Feigned insanity
   - v. Testamentary capacity
   - vi. Delusions
   - vii. Psychopathic personality
   - viii. Psychological autopsy.

FORENSIC TOXICOLOGY

1561. Classify insecticide poisons. Describe in detail signs, symptoms, treatment and PM findings in organophosphorous poisoning cases.
1562. Discuss food poisoning. Add a note on the laboratory investigation of food poisoning.
1563. Discuss briefly on epidemiology of poisons and poisoning.
1564. Discuss in detail on occupational and environmental toxicology.
1565. Discuss on analytical toxicology and laboratory services.
1566. Discuss the management, treatment and medicolegal aspects in mass casualty following consumption of spurious liquor in particular community.
1567. Antidotes are sometimes more dangerous than the poisons. Discuss.
1568. Discuss the psychiatric manifestations of chronic alcoholism.
1569. Prepare a brief project proposal for establishing a drug de-addiction centre at your institution.

1570. Discuss the pathological changes in liver and kidney in cases of phosphorous, lead, methyl alcohol, carbolic acid and cantharides poisoning.
1571. Discuss the clinical features, treatment, and postmortem appearances in a case of strychnine poisoning.
1572. Discuss food poisoning. Add a note on the laboratory investigation of food poisoning.
1573. Write an essay on drug addiction.
1574. Write an essay on cyanide poisoning.
1575. What is the action of cyanide on the body? Describe the signs and symptoms, the treatment and the postmortem appearances in acute cyanide poisoning.
1576. Discuss intoxication by ethyl alcohol. Comment on the legal situation in such cases.
1577. Discuss briefly on the recent improvements in analytical methods in toxicology.
1578. Define drunkenness. Discuss the medical examination and its interpretation in a case of drunkenness.
1579. Describe the mechanism of action, clinical features treatment and sequelae of methyl isocyanate poisoning.
1580. Describe the changing pattern of drug abuse in India.
1581. Give an account of principal plants of faring opportunities for injury through human use.
1582. Discuss the occupational diseases involving the lungs and their medicolegal implications.
1583. Explain the laws relating to drunkenness in your state. On what basis would you certify that the accused had "consumed alcohol and was under its influence" at the material time?
1584. Discuss the modern technique of isolation and identification of poisons from the materials preserved during postmortem examination.
1585. Discuss the different methods of elimination of unabsorbed and absorbed poisons from the body.
1586. Write an essay on modern techniques for the detection and quantification of poisons/drugs.
1587. What is the mechanism of actions of neurotoxic poisons? Discuss the management of strychnine poisoning.
1588. Discuss the signs and symptoms, treatment of a case of barbiturate poisoning. How will you confirm the diagnosis?
1589. Describe in detail the clinical examination of a person suspected of being unfit for driving through a drink or drugs. What tests can be made to attempt to establish the identity of the intoxicating substances?
1590. Discuss the medicolegal aspects of alcoholism.
1591. Write briefly the assessment of severity and clinical features of overdosage and treatment in barbiturate poisoning.
1592. Discuss the various medicolegal aspects of alcohol intoxication.
1593. Discuss the poisoning from insecticide.
1594. Discuss the present trends of poisoning in India with special reference to hazards of modern insecticides.
1595. Write an essay on:
    i. Ideal suicidal poison
    ii. Medicolegal aspects of cyanide poison
    iii. Management of a case snake bite
    iv. Thin layer chromatography (TLC)
    v. Body-packer syndrome
    vi. Methyl isocyanate
    vii. Magnan's symptom
    viii. Metal fume fever
    ix. Gas chromatography
    x. Hallucinogens
    xi. Methyl alcohol poisoning
    xii. War gases
    xiii. Antidote
    xiv. Botulism
    xv. Metal fume fever
    xvi. Carbon monoxide poisoning
    xvii. Brown sugar
    xviii. Halothane
    xix. Complications in drug abuse
    xx. Maladies of chronic alcoholism
    xxi. Ultrashort-acting barbiturates
    xxii. LD 50

GENERAL, BASICS AND RECENT ADVANCES

1596. Define 'shock' and outline the types, aetiopathogenesis, and medicolegal aspects of shock.
1597. Describe foetal blood circulation. Discuss the causes of death in criminal abortion.
1598. Describe the anatomy of 'circle of Willis'. Discuss the aetiology and pathology of intracranial haemorrhages.
1599. Describe the anatomy of female genitalia. How this knowledge is useful in evaluation of medicolegal case?
1600. Describe the blood supply of heart. Discuss the morbid anatomical features in a case of death from ischaemic heart disease.
1601. Discuss the role of radiology in medical practice highlighting the medicolegal aspects.
1602. Describe the classical description forming the criteria as the most reliable indicators of an early myocardial infarction.
1603. Describe the forensic anatomy and blood supply to the heart. Discuss the histochemical and histopathological appearances that would help the confirmation of a postmortem diagnosis in a case of myocardial infarction.
1604. Describe the forensic anatomy of neck. Describe the mechanism of death due to pressure on neck.
1605. Describe the pathophysiology of shock and autopsy findings in a case of fatal anaphylactic shock following intramuscular injection of penicillin.
1606. Describe the process of wound healing. Give an account of the factors which influence wound healing.
1607. Discuss and draw out a pattern of investigations in vehicular accident cases.
1608. Discuss briefly the role of forensic pathologist in a mass disaster.
1609. Discuss on 'use of microscopes in 'forensic medicine'.
1610. Discuss the anatomy of female genitalia and medicolegal importance of intact hymen as a sign of virginity.
1611. Discuss the complications and medicolegal importance associated with surgical and parasurgical procedures.
1612. Discuss the importance of 'spot investigation' by medical jurist including collection of trace evidence, to decide whether the case is one of the accident, suicide or murder.
1613. Discuss the mechanism of shock.
1614. Discuss the medicolegal aspects of deaths on operation table.
1615. Discuss the medicolegal significance of recent advances in molecular biology and genetic engineering.

1616. Discuss the morphological change occurring over the symphyseal face of the hipbone (innominate) and its relevance in determining age.
1617. Discuss the pathogenesis and postmortem diagnosis of 'post traumatic emboli'
1618. Discuss the pathognomonic and morphologic changes of myocardial infarction.
1619. Discuss the pathophysiology of cardiac arrest and heart failure with its relationship to the assessment of final diagnosis, if any, by the autopsy surgeon.
1620. Discuss the role of forensic medicine service to the community. How will you organize a clinical forensic medicine service in your hospital?
1621. Discuss the systemic and localized effects of hypothermia.
1622. Discuss the value of chemical analysis of spinal fluid in ascertaining the time since death.
1623. Forensic anatomy of neck with relevance to death due to pressure on neck.
1624. Give a brief outline on the gross anatomy of the neck with special reference to the medicolegal aspects of various structures.
1625. Give the method of linking the drug with clinico-pathological picture.
1626. How does the foetal circulation and anatomy forms the basis of estimation of the age at an autopsy of a newborn.
1627. Legal and preventive aspects against violence and crime in social life.
1628. Mention the common organisms causing wound infection. How do you investigate a cause of death due to wound infection?
1629. Outline forensic anatomy of the intracranial structures with special reference to intracranial haemorrhages.
1630. Outline the blood circulation of brain. Discuss cerebral hypoxia.
1631. Outline the gross anatomy of intracranial structures with special reference to intracranial haemorrhages.
1632. What are prostaglandins? Describe their pharmacological actions and their therapeutic uses.
1633. Write an essay on 'atrocities against children'.
1634. Write an essay on 'autopsy—a tool to justice'.
1635. Write an essay on 'dead do tell tales'.
1636. Write an essay on 'ethical dilemmas of modern medical man'.
1637. Write an essay on 'every criminal leaves behind some clue, it is for the investigator to find it'.
1638. Write an essay on 'forensic medicine—a savior of human rights'.
1639. Write an essay on 'laboratory—an aid to crime detection'.
1640. Write an essay on 'oncogenesis with special reference to its medicolegal aspects'.
1641. Write an essay on 'past, present and future of forensic medicine'.
1642. Write an essay on 'recent advances in establishing identity'.
1643. Write briefly a critical review with your innovative suggestions on the formalities and requirements to be fulfilled by a medical graduate in establishing and maintaining medical practice in India.
1644. Write briefly on wound healing and repair with special reference to the medicolegal aspects of the changes.
1645. Write an essay on 'abnormal cells in peripheral circulation' having medicolegal significance.
1646. Write short essay on:
    i. Acute renal tubular necrosis
    ii. AIDS
    iii. AIDS and its medicolegal importance
    iv. Alveolar duct membrane
    v. Marrow trauma
    vi. Body-packers
    vii. Circle of Willis
    viii. Cloning
    ix. Coarctation of aorta
    x. Comparison microscope
    xi. Decompression sickness
    xii. Differential diagnosis of malaria
    xiii. Embolism
    xiv. Enterocolitis
    xv. Ewing's postulate
    xvi. Exhumation and second autopsy
    xvii. Fat stains
    xviii. Foetal blood circulation
    xix. Foetal haemoglobin
    xx. Foetal skull
    xxi. Foot print ratio (FPR)
    xxii. Forensic anatomy of hyoid bone
    xxiii. Forensic cytology
    xxiv. Forensic immunology
    xxv. Forensic odontology
    xxvi. Formation of "circle of Willis"
    xxvii. Functions of thymus
    xxviii. Gastric emptying.
1647. Write short essay on:
    i. Gastric emptying
    ii. General adaptation syndrome
    iii. Genetic engineering
    iv. Gram-negative cocci
    v. Histochemistry
    vi. Human hair
    vii. Hyoid bone
    viii. Internal capsule of brain
    ix. Intersex.
1648. Write short essay on:
    i. Lines of Zahn
    ii. Liver function tests
    iii. Mandibular canine index (MCI)
    iv. Medical records and law
    v. Minipackers syndrome
    vi. Nuclear sexing
    vii. Paradoxical undressing
    viii. Pathogenesis of gas gangrene
    ix. Polyarteritis nodosa
    x. Preserving unidentified cadavers in mortuary
    xi. Primary amyloidosis
    xii. Rape trauma syndrome
    xiii. Respiratory distress syndrome
    xiv. Statutory rape
    xv. Subendocardial haemorrhage
    xvi. Test bullet
    xvii. Tumour and trauma
    xviii. Vagal inhibition
    xix. Vital reaction
    xx. Vitamin toxicity.

APPENDIX 2: SYLLABUS AND VARSITY EXAMINATION AID

(A) GOALS AND OBJECTIVES IN FORENSIC MEDICINE AND TOXICOLOGY

(As per the MCI Recommendations in Gazet of India, May 17, 1997)

GOALS

The broad goal of the teaching of undergraduate students in Forensic Medicine is to produce a physician who is well informed. He/she should also be capable of making observations and inferring conclusions by logical deductions, to set enquiries on the right track in criminal matters and connected medicolegal problems. He/she should acquire knowledge of law in relation to medical practice, medical negligence, and respect for codes of medical ethics.

OBJECTIVES

1. Knowledge

At the end of the course, the student should be able to:

- Identify the basic medicolegal aspects of hospital and general practice
- Define the medicolegal responsibilities of a general physician while rendering community service either in a rural primary health centre or an urban health centre
- Appreciate the physician's responsibilities in criminal matters and respect for the codes of medical ethics
- Diagnose, manage, and also identify the legal aspects of common acute and chronic poisonings
- Describe the medicolegal aspects and findings of postmortem examination in case of death due to common unnatural conditions and poisonings
- Detect occupational and environmental poisoning, devise measures of prevention of common poisoning, and deal with legal aspects, particularly pertaining to Workmen's Compensation Act
- Describe the general principles of analytical toxicology.

2. Skills

At the end of the course, the student should be able to:

- Make observations and logical inferences in order to initiate enquiries in criminal matters and medicolegal problems
- Diagnose and treat common emergencies in poisoning and manage chronic toxicity
- Make observations and interpret findings at postmortem examination
- Observe the principles of medical ethics in the practice of his profession.

3. Integration

The department of Forensic Medicine and Toxicology shall provide an integral approach towards allied disciplines like Pathology, Radiology, Forensic Sciences, Hospital Administration, etc. to impart training regarding medicolegal responsibilities of physicians at all levels of health care. Integration with relevant disciplines will provide scientific basis of clinical toxicology, e.g. medicine, pharmacology, etc.

(B) UG THEORY—FORENSIC MEDICINE AND TOXICOLOGY SYLLABUS AND CLASS SCHEDULE

SEMESTER III

Introduction, Medical Jurisprudence and Forensic Pathology

No. of Hours Allotted ***34***

I. *INTRODUCTION TO FORENSIC MEDICINE* 2
   1. Definition, synonyms, subdivisions, modern forensic medicine,
   2. Historical aspects.

II. *LEGAL PROCEDURE* 4
   1. Introduction, courts, magistrates, public prosecutor, witness, exhumation and inquest.
   2. Court procedures – Summons, warrant, attendance in court, conduct money, oath, recording of evidence – exam in chief, leading questions, cross-examination, re-examination, court questions.
   3. Medical evidence and dying declaration, doctor in witness box.
   4. *Tutorial/seminar*

III. *MEDICAL ETHICS* 8
   1. Introduction, MCI and SMC—formation, duration and functions, registered medical practitioner-duties and privileges.
   2. Infamous conduct (professional misconduct), punishments, appeal.
   3. Consent - its relevance in medical practice.
   4. *Tutorial/seminar.*
   5. Malpractice (medical negligence).
   6. Principles of Consumer Protection Act, medical indemnity insurance (MII), euthanasia.
   7. Concepts of Organ Transplantation Act, ethical and legal aspects of AIDS/HIV.
   8. *Tutorial/seminar.*

IV. *FORENSIC IDENTITY* 4
   1. Definition and explanation and MLI of factors establishing human identity.
   2. Corpus delicti, principles and MLI of forensic dentistry and DNA-finger printing.
   3. Determination of and ML importance of age and sex in living and dead.
   4. *Tutorial/seminar*

V. *THANATOLOGY* 6
   1. Introduction, types of death: somatic molecular, brain-stem death in relation to organ transplantation.
   2. Moment of death and sudden death
   3. PM changes: immediate
   4. PM changes: early
   5. PM changes: late and adipocere formation and mummification
   6. *Tutorial/seminar*

VI. *MEDICOLEGAL AUTOPSY* *4*

1. Definition, objectives, rules and procedures of external examination
2. Procedures of internal examination.
3. Negative/obscure autopsy, examination of skeletal remains and special techniques of ML autopsy, murder, homicide or accident
4. *Tutorials/seminar*

VII. *ASPHYXIAL DEATHS* *6*

1. Definition, pathophysiology, general signs and symptoms of asphyxia.
2. Hanging—definition, types and causes of death, PM findings and MLI.
3. Strangulation, suffocation and traumatic asphyxia: definition, types and causes of death, PM findings and MLI.
4. Drowning—definition, mechanisms, pathophysiology, types, and cause of death.
5. Drowning—PM findings, diatom test and MLI.
6. *Tutorial/seminar.*

Total 34 hr

SEMESTER IV

Clinical Forensic Medicine

No. of Hours Allotted ***34***

VIII. *TRAUMA* *18*

1. Definition (clinical and legal) and classification of injuries.
2. Blunt force trauma—abrasions, contusions and lacerations.
3. Sharp force trauma—incised, stab and chop wounds.
4. *Tutorial/seminar.*
5. Regional injuries: Head injuries—mechanism, injury to scalp, face, skull and brain.
6. Regional injuries: Intracranial haemorrhages.
7. Regional injuries: Spinal, thoracic, abdominal and pelvic injuries, and road traffic accident.
8. *Tutorial/seminar.*
9. Injury due to heat and cold and thermal injuries.
10. Electrical and lightening injuries.
11. Medicolegal aspects of trauma in general.
12. *Tutorial/seminar.*
13. Forensic ballistics: Introduction, basics of firearms and explosives.
14. Rifled firearm injuries—clinical/autopsy findings, peculiar effects of firearm injuries
15. Smooth bored firearm injuries—clinical/autopsy findings.
16. PM examination of case of firearm fatalities, medicolegal questions on firearm injuries.
17. Removal, collection and preservation of bullet, pellets for dispatching to FSL, blast injuries.
18. *Tutorial/seminar.*

IX. *SEXUAL JURISPRUDENCE* *4*

1. Explanation and MLI of: virginity, pregnancy, MTP Act, criminal abortion, delivery, paternity, impotency, sterility, and sexual offences.
2. Rape, ML significances of rape, and examination of a rape case.
3. Sodomy, ML significances, and examination of a sodomy case.
4. *Tutorial/seminar*

X. *INFANTICIDE* *4*

1. Explanation of the terminology. Examination of case for viability, Haase's rule.
2. Live born, dead born, still-born, tests to confirm, time of survival, cause of death.
3. Battered baby syndrome and sudden infant death syndrome.
4. *Tutorial/seminar*

XI. *FORENSIC PSYCHIATRY* *4*

1. Explanation of terminologies, Mental Health Act, 1987 (Principles).
2. Insanity, feigned insanity, McNaughten's rule, restraint of an insane.
3. Civil and criminal responsibilities of the insane (Mentally ill).
4. *Tutorial/seminar.*

REVISION CLASSES *4*

Total 34 hr

SEMESTER V

Forensic Toxicology

No. of Hours Allotted ***32***

XII. *GENERAL PRINCIPLES* *4*

1. Introduction to toxicology, analytical toxicology (Bedside and common lab. tests, LC, HPLC, GC, UVS); collection, preservation and dispatch of viscera for chemical analysis to FSL.
2. ML aspects of poisoning, laws on poison – relevant schedules and IPC section, duties of doctors in suspected poisoning cases.
3. Diagnosis and management of poisoning cases in general, classification of poisons.
4. *Tutorial/seminar*

XIII. *CORROSIVE AND IRRITANT POISONS* *12*

1. Corrosives (inorganic)—sulphuric, nitric and hydrochloric acid – action, diagnosis and management, PM findings and MLI.
2. Corrosives (organic)—phenol, oxalic acid, formic acid—diagnosis, management, PM findings and MLI.
3. Irritants (inorganic non-metallic)—phosphorus, halogens—diagnosis and management, PM findings and MLI.
4. *Tutorial/seminar*
5. Irritants (inorganic metallic)—arsenic: action, diagnosis and management, PM findings, and MLI.
6. Irritants (inorganic metallic)—lead: action, diagnosis and management, PM findings, and MLI.
7. Irritants (inorganic metallic)—mercury, copper, and iron: action, diagnosis and management, PM findings, and MLI.
8. *Tutorial/seminar.*
9. Irritants (organic vegetables)—abrus, castor, croton, calotropis, semicarpus, and ergot: plant morphology, diagnosis and management, PM findings, MLI.
10. Irritants (organic animal)—snakes: types, morphology, snake bite, diagnosis and management, PM findings, MLI.

11. Irritants (organic animal)—insect (bees, wasps, centipede, scorpion, spider, etc.) types, morphology, diagnosis and management of bites / stinging by these insects, PM findings and MLI and mechanical irritants – types, diagnosis and management, PM findings, and MLI.
12. *Tutorial/seminar.*

XIV. NEUROTICS *8*

1. Inebriates—ethyl alcohol, action, signs and symptoms, management of a case of poisoning, ML aspects—drunkenness, autopsy findings.
2. Inebriates—methyl alcohol, ethylene glycol, isopropanol, benzodiazepine—action, signs and symptoms, management of a case of poisoning, ML aspects, drunkenness, autopsy findings.
3. Somniferous and sedative hypnotics—opium and opium derivatives—action, signs and symptoms, management of a case of poisoning, ML aspects, autopsy findings.
4. *Tutorial/seminar*
5. Somniferous and sedative hypnotics—barbiturates, chloral hydrate: action, signs and symptoms, management of case of poisoning, ML aspects, drunkenness and autopsy findings.
6. Deliriant: dhathura, cannabis, cocaine.
7. Insecticides/pesticides/agrochemical-organophosphorus compounds, oreganochlorides, carbamates, pyrethroids, aluminum phosphide.
8. *Tutorial/seminar.*

XV. OTHER POISONS *8*

1. Cardiac poisons—oleanders, aconite, and tobacco; spinal poisons - strychnine, asphyxiants (gases)—carbon monoxide, carbon dioxide, cyanogens gas and cyanides.
2. Domestic/household poisons—kerosene, cleansing agents, soaps, detergents, disinfectants, cosmetics, rodenticide, mothballs, etc.
3. Therapeutic drug toxicity/poisoning by medicine—salicylates, paracetamol.
4. *Tutorial/seminar*
5. Therapeutic drug toxicity/poisoning by medicine—antidepressants, sedatives, antipsychotic, insulin, etc.
6. Food poisoning: bacterial, viral, mushrooms, chemical etc.
7. Drugs of dependence and drug abuse—alcohol, tobacco, hypnotics, hallucinogens, stimulants, organic solvents, etc.
8. *Tutorial/seminar.*

Total 32 hr

[Total Classes: 34 + 34 + 32 = 100] (1 hour each)

Books available on Forensic Medicine and Toxicology by the author:

1. Nageshkumar G Rao, Textbook of Forensic Medicine and Toxicology, Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, 2nd Edition, 2010.
2. Nageshkumar G Rao, Principle and Practice of Forensic Medicine, HRP Aid, Manipal, 1st Edition, 1998.

(C) UG PRACTICAL—FORENSIC MEDICINE AND TOXICOLOGY SYLLABUS AND CLASS SCHEDULE

SEMESTER III

No. of Hours Allotted ***10***

Topics

1. Documentation of identification marks and consent form for surgical operation.
2. Birth and death intimation and certification of death.
3. Sick leave certificate and fitness certificate after sick leave.
4. Fitness certificate for job recruitment.
5. Age by examination of X-rays—wrist, elbow and shoulder joints.
6. Age by examination of X-rays—hip, knee and ankle joints.
7. Age by dentition.
8. Age and sex by examination of—skull and mandible.
9. Age and sex by examination of—pelvis and femur.
10. PM instruments.

SEMESTER IV

No. of Hours Allotted ***10***

Topics

1. Weapons of ML importance and reporting on a weapon of suspected assault.
2. Soft specimen/viscera analysis and histopathological examination slides.
3. Photographs of ML importance and expert opinion by photographs.
4. Injury certification.
5. Potency certification.
6. Police intimation on ML case.
7. Police intimation on brought dead case.
8. Police intimation on dead on arrival case.
9. Analysis and interpretation of PM summary.
10. Analysis and interpretation of PM summary.

SEMESTER V

No. of Hours Allotted ***10***

Topics

1. Procedure of dispatching viscera to FSL for chemical examination.
2. Clinical appliances of ML importance.
3. Analysis on corrosives and inorganic and organic irritants.
4. Analysis on neurotoxics.
5. Analysis on cardiotoxics.
6. Analysis on asphyxiants.
7. Analysis and interpretation of PM summary (Poisoning death cases).
8. Drunkenness certification.
9. Revision practical – I.
10. Revision practical – II.

Total classes: **30 (1 hour each)**

Books available on Practical Forensic Medicine by the author:

1. Nageshkumar G Rao, Practical Forensic Medicine, 3rd Edition, Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, 2007.

(D) SPECIFICATION TABLE FOR THEORY EXAMINATION

Marks allotment for theory examination of undergraduate medical curriculum			
Content area	*Type of questions*	*Marks allotment*	*Total marks 80*
Legal procedure,	10 MCQs	0.5 marks	5 marks
Medical ethics and law,	1 Essay	6 marks	6 marks
Forensic psychiatry, and forensic identity	3 Short notes	3 marks	9 marks
Medicolegal autopsy,	10 MCQs	0.5 marks	5 marks
Thanatology, and	1 Essay	6 marks	6 marks
Sexual offences.	3 Short notes	3 marks	9 marks
Mechanical injuries,	10 MCQs	0.5 marks	5 marks
Firearms,	1 Essay	6 marks	6 marks
Regional injuries +RTA,	3 Short notes	3 marks	9 marks
Thermal injuries, and			
Asphyxial deaths.			
Forensic toxicology,	10 MCQs	0.5 marks	5 marks
Forensic science, and	1 Essay	6 marks	6 marks
FSL	3 Short notes	3 marks	9 marks

(E) SPECIFICATION TABLE FOR PRACTICAL EXAMINATION (ADOPTING OBJECTIVE STRUCTURED PRACTICAL EXAMINATION—OSPE METHOD)

Six questions: 5 marks each:	*Total: 30 marks*
1. Examine the skeletal remains and draft your *expert opinion* on sex giving reasons	5 marks
2. Examine the X-rays enclosed and draft your *opinion on age* giving reasons	5 marks
3. Study the extract of postmortem certificate/report given and write your opinion on *cause of death* and *answer 4-6 questions* posed.	5 marks
4. Study the photographs, wet / soft specimens, toxicology specimens, weapons/appliances/PM instruments, and HPE slides—answer the questions asked	5 marks
5. Drafting of drunkenness / injury / potency / death certificate	5 marks
6. Drafting birth intimation / police intimation letter and consent format	5 marks

APPENDIX 3: LAWS OF RELEVANCE TO MEDICAL PROFESSION IN INDIA

Medical practitioners should be aware of the laws/statutes of the land relevant to the medical profession. Ignorance/violation of law may leads to face the undesirable consequence like fine/compensation/imprisonment and even erasure of name from medical register. Certain laws of relevance to medical profession are described in brief as follows.

IPC (INDIAN PENAL CODE, 1860)

It defines offences and prescribes punishments along with provisions for trial. A doctor must have brief knowledge on laws on offences against the human body, as quite often doctors are involved in treating cases of medicolegal nature, e.g. a victim of road traffic accident, assault or attempted suicide, etc.

Scene of crime rarely need doctors visit in our country as unnatural death investigation is the prerogative of police system in our country. Nevertheless, when police officers feel that a forensic pathologist presence in scene investigation is necessary and invites such an expert, one must have basic idea on what to do, allocate an introduction to this topic highlighting what is the role of doctor is in this regard.

An attempt is also made here to provide brief knowledge on court and court procedures needed for the doctor. A brief knowledge of laws on offences against the human body, where in a doctor is often required to play a role, here under.

Laws on Offences Against the Human Body

The offences against the human body dealt under various sections of the *Indian Penal Code* concerning death of human beings, confinement, assault, hurt, sex offences, abortion, cruelty, etc are discussed in this part. Apart from brief consideration about the legal definitions of different types of unnatural deaths, this part also deals with different injuries or hurts, as needed for a medical man to work in conjugation with the law court people. For an easy understanding the Indian Penal Code sections coming under the purview of this are classified into four parts and they are:

1. Offences affecting life *(Sections 299 to 311 IPC)*
2. Offences related to causing hurt *(Sections 319 to 338 IPC)*
3. Offences related to assault *(Sections 349 to 358 IPC)*
4. Offences related to causing hurt and death during robbery or dacoity *(Sections 394, 396, 397 IPC)* and cruelty to married women *(Section 498-A IPC)*.

Offences Affecting Life (Sections 299 to 311)

Sec 299 IPC culpable homicide Whoever, causes death by doing an act with the intention of causing death, or with the intention of causing such bodily injury as is likely to cause death, or with the knowledge that he is likely by such act to cause death, commits the offence of culpable homicide.

If a person by injuring an ill, infirm or disordered person accelerates the process of his -death, then the first person shall be deemed to have caused his death. If a person after injuring another person arranges treatment for the person and if the person still dies, then also the first person will be deemed to have caused his death. Killing of a living foetus that is partly born out of the mother's womb, though might have not breathed, will come under the purview of this section.

Sec 300 IPC murder Except some exceptions, culpable homicide is murder under the following circumstances:

Firstly, if the act by which the death is caused is done with the intention of causing death, or

Secondly, if it is done with the intention of causing such bodily injury as the offender knows to be likely to cause the death of the person to whom the harm is caused, or

Thirdly, if it is done with the intention of causing bodily injury to any person and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death, or

Fourthly, if the person committing the act knows that it is so imminently dangerous that it must, in all probability, cause death, or such bodily injury as is likely to cause death, and commits such act without any excuse for incurring the risk of causing death or such injury as aforesaid.

Exceptions

1. *Culpable homicide* is not murder if the offender lost self-control in the face of sudden and grave provocation and causes death of the provocating person or any other person by mistake or accident.
   Note: The provocation must have not been sought for by the offender as an excuse to kill. If anything done in obedience to the law or lawful discharge of duty by a public servant that will not amount to provocation for the purpose of *exception* of this section. Anything done to exercise the right of self defence will not amount to provocation.
2. *Culpable homicide is not murder*, when the act resulting in death of a person is done in good faith of the right of private defence of person or property, without premeditation, and without any intention of doing more than what is necessary for the defence, if he has actually exceeded the power given to him.
3. *Culpable homicide is not murder* if the offender is a public servant or while aiding a public servant exceeds the power given by law for advancement of public justice, causes death of a person by his such act, done in good faith believed by him to be lawful and necessary, having no ill-will towards the person so killed.
4. *Culpable homicide is not murder* if it is committed without premeditation in a sudden fight in the heat of passion upon a sudden quarrel and without the offenders taking any undue advantage or acting in a cruel or unusual manner. In such a case it is immaterial who offered the provocation or the first assault.
5. *Culpable homicide is not murder* when the person whose death has been caused, being above the age of 18 years, suffers death or takes the risk of death with his own consent.

Sec 301 IPC Culpable homicide by causing death of a person other than whose death was intended will be considered in the line as if death of the intended person has been caused.

Sec 302 IPC Punishment of murder—whoever commits murder shall be punished with death or imprisonment for life and may also be fined.

Sec 303 IPC Punishment for murder by a life convict—A person under life imprisonment if commits a murder he will be punished by death sentence (This section has become ineffective).

Sec 304 IPC Punishment for culpable homicide not amounting to murder may be imprisonment for life or imprisonment of any description which may extend up to 10 years, with or without fine if the act was intended to cause death or such bodily injury as is likely to cause death. When the act is done without the intention to cause death, then the punishment may be imprisonment of either description for a period up to 10 years and or fine.

Sec 304-A IPC Causing death of a person by doing rash or negligent act not amounting to culpable homicide shall be punishable with imprisonment of either description for a term which may extend to two years and or fine.

Sec 304-B IPC Dowry death—When death of a woman occurs due to burn or other bodily injury or in circumstances other than normal, within 7 years of marriage, and it is seen that she was subjected to cruelty or harassment by her husband or any relative of the husband for or in connection with any demand for dowry, such death shall be called "dowry death" and the husband or his relative shall be deemed to have caused her death.

Sec 305 IPC Punishment for abetment of suicide of a child under 18 years of age or of an insane, idiot, delirious or intoxicated person may be death or imprisonment for life or imprisonment for a period not exceeding 10 years with or without fine.

Sec 306 IPC Punishment for abetment of suicide in any other case may extend up to 10 years which may be of either description (simple or rigorous), with or without fine.

Sec 307 IPC An act attempted to commit murder, if causes hurt to the person, may lead to life imprisonment of either description, or imprisonment of either description which may extend up to 10 years with or without fine. If the attempt does not cause any hurt then it is liable to be punished with imprisonment of either description up to a period of 10 years with or without fine. When a life convict makes attempt of murder and if the act hurts the person then the offence is liable to be punished with death.

Sec 308 IPC Attempt to commit an act, the commission of which would amount to culpable homicide not amounting to murder, will make the offender liable to be punished with imprisonment of either description for a term which may extend up to three years and or fine, if no hurt is actually caused by the act. If the attempt causes hurt to the person then it becomes punishable with imprisonment of either term for a period which may extend up to 7 years and or fine.

Sec 309 IPC Attempt to commit suicide makes the person so attempted, liable to be punished with simple imprisonment which may extend for a period of 1 year and or fine.

Sec 310 IPC Defines a "thug", who is a person who habitually associates with others for committing robbery or child-lifting by means of or accompanied with murder.

Sec 311 IPC A thug shall be punished for life with or without fine.

OFFENCES RELATED TO CAUSING HURT INCLUDES *(Sections 319 to 338)*

Sec 319 IPC It defines hurt. Whoever causes bodily pain, disease or infirmity is said to cause hurt.

Sec 320 IPC This section designates grievous hurt. The following is the list of grievous hurts—
- Emasculation
- Permanent privation (loss) of sight of either *eye*
- Permanent privation of hearing of either ear
- Privation of any member or any joint
- Destruction or permanent impairing of power of any member or joint
- Permanent disfiguration of head or face
- Fracture or dislocation of a bone or tooth
- Any hurt which endangers life or which causes the sufferer severe bodily pain or makes him unable to follow his ordinary pursuits for a period of 20 days.

Sec 321 IPC This section defines the term "voluntarily causing hurt". If an act is done with the intention to cause hurt or with the knowledge that, the act is likely to cause hurt to a person then, the act amounts to "voluntarily causing hurt".

Sec 322 IPC This section defines the term "Voluntarily causing grievous hurt". If an act done with intention to cause grievous hurt or done with the knowledge that, the act is likely to cause grievous hurt and if actually grievous hurt is caused then, it amounts to voluntary causation of grievous hurt.

Sec 323 IPC It describes the punishment for voluntarily causing hurt, which may be imprisonment of either description which may extend up to one year and/or fine which may extend up to one thousand rupees. *Exception*—as provided under section 334.

Sec 324 IPC As per this section, punishment for voluntarily causing hurt by dangerous weapons or means shall be imprisonment of either description for a term which may extend up to a period of three years and or fine. *Exception*—as provided under section 334 IPC.

Sec 325 IPC Punishment for voluntarily causing grievous hurt shall be imprisonment of either description for a term which may extend up to seven years and also with liability to be fined. *Exception*—as provided under section 335 IPC.

Sec 326 IPC Punishment for causing grievous hurt by dangerous weapons or means shall be imprisonment for life or imprisonment of either description for a term which may extend up to ten years with also liability to be fined. *Exception*—as provided under section 335 IPC.

Sec 327 IPC Punishment for causing hurt to extort property or to constrain to an illegal act shall be imprisonment of either description for a term which may extend to ten years with or without fine.

Sec 328 IPC Punishment for causing hurt by means of poison, etc. with intent to commit an offence shall be imprisonment of either description for a term which may extend to ten years with or without fine.

Sec 329 IPC Punishment for voluntarily causing grievous hurt to extort property, etc. or to constrain to an illegal act shall be imprisonment for life or imprisonment of either term which may extend up to 10 years, with liability to be fined also.

Sec 330 IPC Punishment for voluntarily causing hurt to extort or to compel restoration of property shall be imprisonment of either description for a term which may extend to seven years with or without fine.

Sec 331 IPC Punishment for voluntarily causing grievous hurt to extort confession or to compel restoration of property shall be imprisonment of either description for a term which may extend to ten years with or without fine.

Sec 332 IPC Punishment for voluntarily causing hurt to deter public servant from his duty, shall be imprisonment of either description for a term which may extend to three years and or fine.

Sec 333 IPC Punishment for voluntarily causing grievous hurt to deter public servant from his duty, shall be imprisonment of either description for a term which may extend to ten years with or without fine.

Sec 334 IPC Punishment for voluntarily causing hurt on grave and sudden provocation without knowledge or intent to cause hurt to any person other than the person giving the provocation, shall be imprisonment of either description for a term which may extend to one month and or fine up to five hundred rupees.

Sec 335 IPC Punishment for causing grievous hurt on grave and sudden provocation shall be imprisonment of either description for a term which may extend to four years and or fine up to two thousand rupees.

Sec 336 IPC An act endangering life or personal safety of others if done rashly or negligently shall be punished with imprisonment of either description for a term which may extend to three months and or fine which may extend to two hundred and fifty rupees.

Sec 337 IPC Causing hurt by act endangering life or personal safety of others if done rashly or negligently, shall be punished with imprisonment of either description for a term which may extend to six months and of fine which may extend to five hundred rupees.

Sec 338 IPC Punishment for causing grievous hurt by rash and negligent act endangering life or personal safety of others shall be imprisonment of either description for a term which may extend to 2 years and or fine which may extend to one thousand rupees.

OFFENCES RELATED TO ASSAULT *(Sections 349 to 358)*

Sec 349 IPC This section defines "Force". A person is said to use force to another person if he causes motion or change of motion or cessation of motion to the other person or to any substance bringing it in contact with the body of the other person or creates sense of feeling in the other person with anything which the other person is wearing or carrying or being so situated. Causation, change or cessation of motion, for the purpose of this section may be caused by: (a) own bodily power, (b) by disposing any 'substance in a manner that motion or change or cessation of motion takes place without any act on his part or on the part of any other person, (c) by inducing any animal to move, to change its motion or to cease to move.

Sec 350 IPC defines "Criminal force". Criminal force means force used intentionally without consent of the person on whom the force has been used, to commit an offence or with intention or knowledge that such use of force is likely to cause injury, fear or annoyance to the person.

Sec 351 IPC This section defines assault. Whoever makes any gesture, or preparation intending or knowing it to be likely that such gesture or preparation will cause any person present to apprehend that he who makes that gesture or preparation is about to use criminal force to that person, is said to commit an assault.

Sec 353 IPC Punishment for causing assault or using criminal force to deter public servant from discharge of his duty shall be imprisonment of either description for a term which may extend to 2 years and or fine.

Sec 354 IPC Punishment for causing assault or using criminal force to a woman with intent to outrage her modesty shall be imprisonment of either description for a term which may extend to 2 years and or fine.

Sec 355 IPC Punishment for causing assault or using criminal force with intent to dishonour a person otherwise than on grave provocation—up to 2 years imprisonment of either description and or fine.

Sec 356 IPC Punishment for causing assault or using criminal force in attempt to commit theft of property carried by a person—up to 2 years imprisonment of either description and or fine.

Sec 357 IPC Punishment for causing assault or using criminal force in attempt to wrongfully confine a person—imprisonment of either description up to 1 year and or fine up to one thousand rupees.

Sec 358 IPC Punishment for causing assault or using criminal force on grave provocation—simple imprisonment up to 1 month and or fine up to 200 rupees.

OFFENCES RELATED TO CAUSING HURT AND DEATH DURING ROBBERY OR DACOITY *(Sections 394, 396, 397)* AND CRUELTY TO MARRIED WOMEN *(Section 498-A)*

Sec 394 IPC Punishment for voluntarily causing hurt in committing robbery shall be imprisonment for life or with rigorous imprisonment for a term which may extend to 10 years with or without fine.

Sec 396 IPC Punishment for causing dacoity with murder shall be death or imprisonment for life or rigorous imprisonment for a term which may extend to 10 years with or without fine.

Sec 397 IPC Punishment for committing robbery or dacoity with attempt to cause death or grievous hurt—shall be minimum 7 years imprisonment.

Sec 459 IPC Punishment for causing grievous hurt whilst committing lurking house, trespass or house-breaking shall be imprisonment for life or imprisonment of either description for a term which may extend to ten years with or without fine.

Sec 498-A IPC Punishment for the husband or relative of husband of a woman subjecting her to cruelty, shall be imprisonment for a term which may extend to 3 years with or without fine.

Indian Evidence Act, 1872

This act deals with the evidences and describes the procedure of collection, preservation and presentation of evidence in the court and also lays down punishment for failure of collection/preservation of evidence or concealment of evidence/damaging the evidence.

CPC (Code of Civil Procedure), 1908

It regulates civil proceedings and includes Law of Fort (Medical negligence).

Workmen's Compensation Act, 1923

This act provides for payment of compensation by the employer to the employees for suffering any injury or disability or disease resulting from accident or occupational hazards during the course of delivery of duty. The amount of compensation is related to the degree of disability, extent of injury or nature of the decease.

Compensation is paid on the basis of, whether the disability is permanent or temporary and also the degree of disability whether total or partial. In case of partial disability, its percentage is counted in terms of extent of loss of function of different parts or systems of the body. If death of worker occurs resulting from accident or occupational hazard, compensation is paid to the legal heir and dependent family members of the deceased.

To be entitled for compensation, conduct of employee needs to be proved not responsible for the accident or hazard. For example, if an employee sustains injury while working with machine under the influence of alcohol, he or she may not get compensation for the accident or injury. The role of a doctor is very important to assess the degree of disability or harm suffered by the worker from the occupational sources.

The Mines Maternity Benefit Act, 1941

This act provides maternity leave and other benefits for the female workers of the mines.

Employees' State Insurance Act, 1948

This act provides a scheme of compulsory health insurance for industrial workers and managed by the Medical Benefit Council. It provides for the establishment and maintenance of hospitals and dispensaries and maternity centres. The insured employer is entitled to sickness cash benefit, maternity benefit, disablement and dependant benefit and medical treatment. The cost is met on tripartite basis i.e. by employer, employee and state. Person to whom dependant benefit is payable requires to obtain the death certificate of deceased issued by the attending Insurance Medical Officer. Insurance Medical Officer requires to fill up the Medical Certificates in prescribed forms provided as per this Act.

Factories Act No. 63 of 1948

As per provisions of this act, registered medical practitioners are appointed to inspect and supervise the factories to ascertain whether the factory is liable to create any health hazard and also to certify the health of workers. A child below the age of 14 years cannot be employed in a factory. Above the age of 14 years and below 18 years, a person can be employed in a factory provided a certificate of physical fitness is obtained from the medical officer. Such a fitness certificate is issued for a period of 1 year at a time and may be renewed annually or may be revoked depending on the effect of the job on the health of the young employee.

Constitution of India, 1950

Article 21 states that no person shall be deprived of his life or personal liberty except according to the procedure established by law (the fundamental right to life).

Article 47 states that it is the duty of the State to raise the level of nutrition and standard of living of its people and to improve public health (this is a Directive Principle).

Article 32 gives every citizen the right to ask the Supreme Court to enforce his or her fundamental rights (the right to enforce a fundamental right).

Action can be brought to remedy a situation which vidates the right to life. Where legal injury is caused to a person or a class of persons who by reason of poverty, disability or socially or economically disadvantaged position cannot approach the courts for judicial redress, any member of public acting in good faith can bring an action before the court seeking judicial redress for them. This is an useful tool to initiate proceedings in the Supreme Court or High Court in the form of PIL (Public Interest Litigation) though the applicant is not the actual victim of the harm.

The Plantation Labour Act, 1951

The provisions of this act in connection with appointment of young workers and medical supervision of plantation labours are similar to Indian Factories Act No. 63.

The Indian Medical Council Act, 1956

As per provisions of this act, Medical Council of India and also the State Medical Council for the State are constituted. Medical Council of India is concerned with maintenance of medical education, recognition of medical qualifications and degrees conferred by the recognized institutions inside the country and also the medical degrees of foreign countries on reciprocal basis, and maintains the register of registered medical practitioners and prescribes code of conduct and ethics for the medical practitioners, violation of which makes the doctor liable for appropriate action.

The State Medical Council maintains the medical register including the names of registered medical practitioners of the State and exerts disciplinary control over the medical practitioners. Issuing of warning notice, reprimentation and also penal erasure of name of registered medical practitioner found guilty of infamous conduct fall under the purview of State Medical Council. Medical practitioner punished by the State Medical Council can appeal to the Ministry of health, Government of India which refers the case to Medical Council of India and the decision of the Medical Council of India ultimately settles the case.

The Medical Termination of Pregnancy (MTP) Act, 1971

The Act provides the conditions for medical termination of pregnancy and insists on strict compliance of prescribed guidelines and lays down the punishment for violation of provisions of the Act.

Cr PC (Code of Criminal Procedure), 1973

This code lays down the provisions and procedure to deal with different crimes and regulates criminal prosecution.

Narcotic Drugs and Psychotropic Substances Act, 1985

This act repeals the Dangerous Drugs Act 1930, the Opium Act 1957 and the Opium Act 1978 and makes provisions for effective control over the drugs of abuse and prescribes enhance penalties for illicit traffic of Narcotic Drugs and Psychotropic Substances.

Consumer Protection Act (COPRA/CPA), 1986

The main purpose of the Act is to provide simple procedure and speedy remedies for suffering patient/party and enables a consumer patient to make complaint to the appropriate Redressal Forum in respect of defective service of the doctor provided that the service has been paid for.

The Mental Health Act, 1987*

This Act includes regulatory provisions for proper care and rehabilitation of mentally ill persons. Psychiatric hospital or nursing home can be established or run only on obtaining the license from State or Central Authority for mental health services.

The Transplantation of Human Organs Act, 1994*

This Act provides for regulation for removal, storage, and transplantation of human organs for therapeutic purposes and for the prevention of commercial dealings in human organs and for matters connected herewith or incident thereto.

The Prenatal Diagnostic Technique Act, 1994*

This law was enacted by the Parliament in 1994 to prevent the selective abortion of female foetus and thereby injustice to woman and also to prevent the imbalance of sex ratios. The Act prescribes punishments for the abuse of medical techniques for professional gain.

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* For detail particulars of the above Acts/Statutes (except IPC) reader is requested to refer to the concerned 'Law/Statutes'.

APPENDIX 4: STARVATION AND NEGLECT AND LAW

Definition: *'Starvation'* is defined medically in two parts and that is: the act or process of starving and the condition of being starved, while *'neglect'* is defined as - to fail to give due care, attention, or time to some one an adult/elderly/a child especially.

Malnutrition *(Synonyms: 'Cachexia', 'emaciation' and 'marasmus'):* It is better we know about this terminology as well at this juncture. This result from deficiency of bodily constituents like proteins, carbohydrates, vitamins/minerals, etc added with partial deprivation of food, qualitatively and/or quantitatively for some period. Weight of the body is diminishing gradually as a consequence of loss of carbohydrates, fats and proteins. There will be slow wasting of the body fats and muscles and emaciation of the body takes place. Rest of the findings in the body will be the same as described under starvation above. Cause of death will be usually due to various infections or malnutrition syndromes. Some of the metabolic disorders in infants and children may cause *wasting* and *'Marasmus'* which may not be diagnosed by autopsy, has to be excluded by medical history if available as it may be a criminal issue in case of intentional neglect by parents/guardians. It is better we are aware of another related terminology of legal significance

In India, *IPC Sections 317* and *318,* dealing with *'Abandoning of Infants'* and *'Concealment of Birth'*, respectively, are the steps to curb this evil. Presently, *'child abuse'* p (physical, sexual and mental) is inviting more attention than starvation and neglect.

IPC Sections 317 and 318

Section 317: Exposure and abandonment of child under twelve years, by parent or person having care of it.

Whoever being the father or mother of a child under the age of twelve years, or having the care of such child, shall expose or leave such child in any place with the intention of wholly abandoning such child, shall be punished with imprisonment of either description for a term which may extend to seven years, or with fine or with both.

Explanation: This section is not intended to prevent the trial of the offender for murder or culpable homicide, as the case may be, if the child dies in consequence of the exposure.

Section 318: Concealment of birth by secret disposal of dead body.

Whoever, by secretly burying or otherwise disposing of the dead body of a child whether such child dies before or after or during its birth, intentionally conceals or endeavours to conceal the birth of such child, shall be punished with imprisonment of either description for a term which may extend to two years, or with fine, or with both.

Forced Feeding of a Fasting Prisoner

The Law in India is currently vague. There are arguments that if the doctor does not *force feed* the prisoner, he may be sued for abetment of suicide (*Section 306 of IPC*). On the contrary if the doctor force feeds the prisoner against his wish, he may be sued for using criminal force (*Section 350 and 352* of Indian Penal Code. A lawyer can argue the case from either side).

World Medical Association *(WMA)* - has however given guidelines to doctors in its *Declaration of Tokyo* (adopted in 1975 and amended in 2005) and *Declaration of Malta* (adopted in 1991 and amended in 1992).

Declaration of Tokyo basically gives guidelines to physicians concerning *torture and other cruel, inhuman or degrading treatment or punishment* in relation to *detention and imprisonment*. Basically both these *Declarations* respect the autonomy of the patient, and consider very much the opinion that the doctor should not force feed such cases. It affirms the facts that:

Where a prisoner refuses nourishment and is considered by the physician as capable of forming an unimpaired and rational judgment concerning the consequences of such a voluntary refusal of nourishment, he or she shall not be fed artificially.

However, the decision as to the capacity of the prisoner to form such a judgment should be confirmed by at least one other independent physician.

The consequences of the refusal of nourishment shall be explained by the physician to the prisoner.

In cases of *Hunger strikers,* role of the doctor is specifically highlighted by *Declaration of Malta*. Argument is that the doctor is indeed under a serious dilemma. On one hand, *"there is a moral obligation on every human being to respect the sanctity of life. This is especially evident in the case of a doctor, who exercises his skills to save life and also acts in the best interests of his patients"* and on the other hand *"it is the duty of the doctor to respect the autonomy which the patient has over his person. A doctor requires informed consent from his patients before applying any of his skills to assist them, unless emergency circumstances have arisen in which case the doctor has to act in what is perceived to be the patient's best interests."*

Following guidelines are given to resolve this conflict: The doctor should ascertain on a daily basis whether or not the patient wishes to continue with his hunger strike. The ultimate decision on intervention or non-intervention should be left with the individual doctor without the-mtervention of third parties (such as jailor etc) whose primary interest is not the patient's welfare. However, the doctor should clearly state to the patient whether or not he is able to accept the patient's decision to refuse treatment or, in case of coma, artificial feeding, thereby risking death. If the doctor cannot accept the patient's decision to refuse such aid, the patient would then be entitled to be attended by another physician (article 4 of the preamble of this Declaration). The hunger striker must be professionally informed by the doctor of the clinical consequences of a hunger strike, and of any specific danger to his own particular case. An informed decision can only be made on the basis of clear communication. An interpreter should be used if indicated. The doctor has a responsibility to inform the family of the patient that the patient has embarked on a hunger strike, unless this is specifically prohibited by the patient. When the hunger striker has become confused and is therefore unable to make an unimpaired decision or has lapsed into a coma, the doctor shall be free to make the decision for his patient as to further treatment which he considers to be in the best interest of that patient, always taking into account the decision he has arrived at during his preceding care of the patient during his hunger strike, and reaffirming article 4 of the preamble of this Declaration. *(Source: Aggrawal A., Self assessment and review of Forensic medicine and toxicology, 1st Ed, Peepee Publishers and Distributors 2006)*

Certain Clinical and Autopsy particulars of starvation are discussed below:

Starvation may be the consequence of complete/partial deprivation of regular supply of food. It is regarded as *'acute'* when food and water are suddenly withdrawn as in mines or landslides, in entombment in pits, willful refusal to take food. Likewise when there is a gradual deficient supply of food, as in famines and /or in camp conditions constitutes *'chronic starvation'*. The minimum food requirement for an adult weighing 60 kg would be 1800 calories per day. Life in a person is at *risk* when the body weight is lost more than 40% of the original weight.

Clinical Features: In acute starvation, there is a feeling of hunger for the first 30 to 48 hours, followed by pain in the epigastrium. After 4 to 5 days of starvation, general emaciation and absorption of the subcutaneous fat begins to occur. Before death body has offensive odour. Usually the loss of 40% of body weight is fatal. Death occurs from exhaustion, circulatory failure due to brown atrophy of the heart, or inters current infection. During starvation the substance which heart uses as energy source is aceto acetate.

If starvation exceeds 7 days then the major nutrition supply of brain comes from ketone-bodies. The changes in starvation seen are Hypoglycemia, Hypothermia, Hyper triglyceremia and Keto-acidosis.

Fatal Period: Death usually occurs in 10-12 days if both water and food are totally withdrawn. If food alone is withdrawn, death may occur in 6-8 weeks or even more. The period, however, is influenced by number of factors like age, sex, condition and environment of the body. *Case Report*: A 50-year-old Jain woman s successfully completed a lO8-day religious fast. She used to have only boiled water during the fast, as t reported.

Autopsy Findings: It is always advisable to perform radiography prior to autopsy so as to exclude the physical abuse which is often associated with neglect. Photography should also be performed. In both children and adults, the major problem remains the connection between the cause and effect, especially when some disease is also present.

External: Body of a victim of *starvation death* is *emaciated* and may *emit disagreeable offensive odour*. Eyes are usually dry and open, with sunken eye balls and dilated pupils. Malar bones will be prominent as facial contours not maintained due to loss of fat. Chest is with sunken intercostal spaces and prominent ribs. Abdomen is *scaphoid* or boat shaped and also sunken. Tongue is dry and coated. Skin is dry and shrivelled. Body fat is wholly deficient.

Internal: Findings are enumerated as below:

General - Fat is absent from subcutaneous tissues and muscles.

Brain – is pale and soft with congested meningeal vessels (at times).

Heart – is small in size with cardiac muscles by and large found flaccid and empty. Hear chambers are usually empty, pale and collapsed, and show hypostatic congestion.

Gastro intestinal tract: Stomach is small, contracted, and empty. Mucous membrane of stomach and upper part of intestine is stained with bile. Large intestine has hard faecal matter.

Gall bladder - is unusually distended. It contains dark, and filled with dry and inspissated bile.

Liver - shows necrosis.

Spleen, kidneys and pancreas - are small and shrunken.

Urinary bladder - is empty.

Fatal Period

Death occurs in 10-12 days if the person is deprived of both food and water. If food only is withdrawn the life is prolonged by 6-8 weeks or even more.

Factors that Influence Fatal Period

Age - Children suffer most. Old people require less food; hence can survive the condition for sometime.

Sex - Females can withstand starvation for a longer period than males because they have more fat and consume less food.

Condition of body - Fatty healthy people survives more than the lean and weak person.

Environment of the body - Starvation is well tolerated by persons where activity is less. Exertion during starvation hastens death.

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